Scand J Work Environ Health 1980;6(2):1-86

<https://doi.org/10.5271/sjweh.2639>

Issue date: 1980

Silicosis. Observations on a case register.

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Key terms: [case register](#); [silicosis](#)

This article in PubMed: www.ncbi.nlm.nih.gov/pubmed/7384772

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Silicosis

Observations on a case register

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Section I

Introduction

Silicosis

Pneumoconiosis, the term used in this monograph to denote reaction in the lung to inhaled dust, remains one of the classical problems in occupational safety and health. Silicosis is a specific form of pneumoconiosis. It is produced by the inhalation of crystalline silica. Silicosis has a long history as a serious occupational disease of the lungs; it is characterized by nodular fibrosis and often has a progressive pathological and/or clinical course. Many years of exposure to free silica dust are usually required to produce silicosis. The diagnosis is mainly based on typical radiographic lesions in the lungs and ascertained exposure to silica dust.

The clinical, pathological, radiological, and epidemiologic features of silicosis have been reviewed by such authors as Parkes (36), Morgan & Seaton (33), Reichel (39), and Wagner (43).

In general the risk of contracting silicosis is determined by a variety of factors. For convenience, they can be grouped into the following main headings:

- (a) qualitative and quantitative characteristics of the dust in the occupational environment,
- (b) amount and characteristics, including biological activity, of dust deposited and retained in the lungs,
- (c) individual and constitutional factors that may influence the retention of dust in the lungs and the reaction of pulmonary tissue to retained dust components.

A well-known complication of silicosis is pulmonary infection by tubercle bacilli. In earlier times, complicating tuberculosis infection frequently led to a rapid progression in the course of the clinical disease and to death.

Silicosis is reported to have become less common in recent years in some countries, for instance, in the Federal Republic of Germany and the United Kingdom. This decrease has been attributed to the more efficient preventive measures and to a reduction in the number of persons in occupations involving risk of silicosis (36, 39).

The nature of the association between silicosis and its risk-determining factors is still incompletely understood. This lack of understanding can, to a large extent, be ascribed to difficulties in interpreting epidemiologic and other studies. The factors underlying these difficulties can be summarized as follows: (i) uncertainties in the available information on the environment; (ii) deficient monitoring of populations at risk with regard to exposure characteristics, eg, intensity (atmospheric concentration of noxious dust), and time-related factors (frequency of exposure, total duration and duration of brief exposures at peak intensities); (iii) varying standards for diagnosis between countries and within countries at different points in time.

Generally speaking, well-designed epidemiologic studies are essential for a proper understanding of the manner in which various factors influence the risk of contracting a disease and for knowledge of the natural course of disease. In regard to environmentally induced diseases, such studies should be population-based and based on reliable measurements of exposure to the causal factor(s) and careful assessments of the disease process.

Purpose of the study

The purpose of this monograph is to describe and analyze observations made on a national pneumoconiosis register. This register contains all the cases of silicosis no-

tified in Sweden since 1931. The observations were made with the objective of detecting any main changes in silicosis morbidity over a long period and to explore the possibility that such changes may be monitored with the aid of a central case register. It was hoped that the analysis would permit the drawing of conclusions with a bearing on the prevention of the disease.

The following questions were central to the purpose of the study:

1. Has any change occurred in the numbers of cases notified to the register or in the numbers notified for different occupational categories over the years?
2. Have there been discernible changes in features such as length of dust exposure and age at diagnosis?
3. What is the pattern of mortality in the study population?
4. Has there been any change in the relationship between silicosis and pulmonary tuberculosis during the study period?
5. Is silicosis associated with an increased mortality from lung cancer?
6. How often does silicosis take a radiographically progressive course?
7. Does cessation of exposure at diagnosis measurably influence the radiographic progression of the lesions?
8. What aspects of the disease can be studied with a central case register as the source of information?

With these questions in mind, I wrote this monograph trying to give a reasonably comprehensive presentation of the material collected in the Pneumoconiosis Register in Sweden. The questions are dealt with in the separate sections.

Two sections in the last part of the monograph deal with the environment which has generated the cases of silicosis. These two sections — "General Trends in the Environment" and "Measurements of Environmental Dust" — are based on other sources of information than the Pneumoconiosis Register. The sections are included to provide relevant background information.

The presentation of the material and the ensuing analyses have necessitated the use of a large number of tables and figures to highlight various aspects of the subject. In order to facilitate the reading of the text, some of the tables and figures have been placed in an appendix (appendix C). For the reader's convenience they have retained the numbers they would have been given had they appeared in the text. Since I worked with the intention of making each chapter of the monograph independent, the reader will also find explanatory repetitions.

The Pneumoconiosis Register

Registration

The Pneumoconiosis Register has been compiled from case notifications to the National Social Insurance Board and to private insurance firms from 1931 on. Since 1970 all notifications have been made only to the Board. At the Pneumoconiosis Registry the information on each case is extracted and systematized. The Registry was established in 1953. Since 1972 the Register has been maintained by the National Board of Occupational Safety and Health.

Compulsory notification of silicosis (including mixed dust pneumoconiosis) was introduced in Sweden in 1931, when the disease was recognized as an occupational disease in a legal sense. From 1931 on notification was to be made regardless of whether an issue of financial compensation was involved or not. Financial compensation for disablement from silicosis was introduced at the same time and has since been gradually extended. Benefits are now paid through social security funds. All these developments have led to a centralized administrative processing of all notified cases. The processing (by the National Social Insurance Board) includes special medical assessment to establish the stage of silicosis and the degree of disablement and to define the time at which the lesions became radiologically manifest.

The medical assessments have been, from the introduction of legislation in

1931, jointly made by a physician and a radiologist, both specialists in pulmonary disorders. All available information in each case was submitted to them. The physician who has reviewed all the cases notified since 1931 and entered them into the Register is Prof T Bruce, MD. In later years, beginning in the 1960s, all cases notified to the central board have been assessed by him, but only those presenting radiographically atypical features have been referred also to the radiologist.

The assessments are based on documented clinical findings, in particular in the radiographs, and on occupational history. For this purpose all the medical and occupational records, including case notes, radiographs, death certificates, etc, are collected. Each notified case is assessed in this manner, and corresponding documents are routinely collected for follow-up of the individual cases. If additional information is necessary for adequate follow-up, it is collected from the workers themselves and from employers, hospital records, medical practitioners, industrial medical officers, population registrars, etc.

The staff of the Pneumoconiosis Registry comprise one full-time assistant (Ms I Sjödin), who is responsible for all the routines, and one or more part-time assistants. The Registry staff record the extracted information from the notifications and the follow-up on individual sheets. The data are then condensed for transfer to punch cards. The following information is obtained at the time of notification and is supplemented on each follow-up occasion: (i) identity (name, date of birth, and personal identity number); (ii) data on silicosis (radiographic stage and other observations, date of detection), degree of disablement, etc; (iii) occupation (current and previous employers, types and times of employment, etc); and (iv) morbidity and mortality (tuberculosis, hospitalization, date and cause of death, etc).

The objective of all the collection and accuracy-checking of data is to attain a quality that ensures their future scientific usefulness. From the start of the Register in 1953 great efforts were made to ensure the completeness and quality of all the registered information and to retrieve all cases notified from 1931 on. To achieve this goal, considerable work went into search-

ing through files of the involved insurance companies. The Registry staff is under the scientific guidance of Prof A Ahlmark, MD, and Prof T Bruce, MD, with respect to both the registration of notified cases and the follow-up, which is constantly carried out as part of the work routine.

A presentation of the Pneumoconiosis Register was first published in 1965 by Ahlmark et al (5).

Radiography

A radiographic examination is of paramount importance for the diagnosis of silicosis. The radiological diagnosis is essentially dependent on two factors, (i) the quality of the radiographs and (ii) the skill and experience of the radiological assessor. The radiographic manifestations of silicosis, particularly in the early phase, may be difficult to recognize, and the problem of distinguishing between early silicotic lesions and exaggerated normal markings is well known to chest radiologists. The objectives of the radiographic examinations are:

- (a) to establish the presence of pulmonary fibrosis,
- (b) to determine whether such fibrosis may be due to silicosis,
- (c) to search for evidence of other previous or concurrent pulmonary disease,
- (d) to assess the relationship of pulmonary fibrosis to such other disease,
- (e) to classify the stage of silicosis.

The assessors of the cases notified to the National Social Insurance Board also attempt to achieve these goals.

The classification of individual radiographs is aimed at providing a simple and reproducible staging of silicosis. Stage I, II, and III comprise the basic categorization, both in the individual assessments and in the follow-up.

The criteria underlying these stages conform, by and large, with the scheme recommended at Johannesburg in 1930. Stage I thus indicates silicotic opacities up to the size of a pinhead, and stage II larger lesions, up to 4 mm. Still larger lesions

and coalescence to form massive shadows are described as stage III (17). If, in the present study, the lesions were in transition from one of these stages to another, they were consistently assigned to the lower stage.

Since the Johannesburg conference in 1930 other systems for classifying radiographic lesions have been constructed and internationally recognized, mainly under the auspices of the International Labor Office (ILO). Foremost among these are the Sydney classification (1950), the (revised) Geneva classification (1968), and the ILO U/C 1971 and ILO U/C 1979 classifications.

The principal reason for retaining the Johannesburg scheme in this study was that it had been accepted by the radiologists and was routinely applied in the same way throughout the period covered by the study. In the register, no attempt was made to reclassify older cases as new schemes of classification were proposed.

Liddell & Morgan (18) strongly emphasized the importance of defining the objectives of a classification scheme fully and precisely at the outset. Apart from the practical advantage of constancy over a study period of more than 40 a, retention of the Johannesburg scheme provided the present study with a simple and relevant classification according to the radiographic severity of silicosis.

An important point in this connection is that the diagnoses in the Register are retrospective. In other words, all previous and current radiographs are collected and studied in series. The date of the first radiograph showing evidence of silicosis is then recorded as the time at which the disease became manifest. Consequently, the lesions at that time may not have yet reached the size corresponding to stage I. Most retrospective diagnoses are regarded

as early stage I. Obviously retrospective diagnoses can only be made for patients who have earlier been examined radiographically.

It should be mentioned that in the assessment of the results of follow-up examinations the same procedure is applied. All available radiographs are examined in every case.

Since 1938 there has been a legal requirement (statute) for periodic medical examinations of persons exposed to silicosis hazards in their work environment. Therefore in many cases notified to the National Social Insurance Board and to the Register there are radiographs from earlier examinations.

Classification of exposure

Exposure to silica-bearing dust is qualitatively categorized by the allocation of cases to occupational categories. These categories and the relevant codes are listed in appendix A. A requirement for allocation to any particular occupational category is that at least 90 % of the total time of exposure to silica dust had been in that occupation. For each main type of industry, a subcategory is reserved for persons who had previously held more than one occupation within that particular industry. Otherwise, the person was included in the category designated as "others, including mixed exposure." The classification and the coding system were devised by the Pneumoconiosis Registry. Many industries and occupations are represented in the study population. In the present study the length of employment in a dust-exposed environment was used as a correlate for quantitative exposure. Duration of exposure was equated with years of employment in the dusty occupation.

Section II

Survey of the study population

In this section the total study population is presented. The variables used in the descriptions are age, duration of prediagnostic dust exposure, and, in some instances, latency time (from commencement of exposure to diagnosed silicosis).

In one subsection some occupational categories are described in more detail, and in another the cases from these occupational categories are described according to the radiographic stage of the disease at diagnosis.

In many of the tables that follow, the study population is grouped according to period during which silicosis became manifest, ie, 1931—1950, 1951—1960, and 1961—1975. The dividing year 1950 was chosen as roughly coinciding with the breakthrough of effective antituberculous drugs in Sweden. This therapeutic innovation against a disease so closely associated with silicosis is used as a guideline also in later presentations of mortality. The 1960 division was somewhat arbitrarily set.

All occupational categories

All the cases of silicosis or mixed dust silicosis reported to the Pneumoconiosis Register from 1931—1975 are presented in table 1, regardless of occupation. The year of notification to the National Social Insurance Board is cross-tabulated with the year in which silicosis, according to the Board's decision, became radiologically manifest. For reasons of space, 5-a groupings are used.

The figures in table 1 show a general trend towards an increased reporting of silicosis, with peaks in the immediate postwar years and in the periods 1951—1955 and 1966—1970.

The year of notification was frequently later than the year of disease manifestation. The diagnosis in these cases had been made retrospectively, with the aid of serial radiographs.

In table 2 the notified cases of silicosis are shown according to the main industrial

Table 1. Distribution of registered cases of silicosis (including mixed-dust fibrosis) according to year of notification and year of manifestation. — All occupational categories.^a

Cases notified	Silicosis manifest										Total
	≤ 1930	1931—1935	1936—1940	1941—1945	1946—1950	1951—1955	1956—1960	1961—1965	1966—1970	1971—1975	
1931—1935	4	121									125
1936—1940	7	81	149								237
1941—1945	1	42	85	247							375
1946—1950	2	22	133	197	326						680
1951—1955		9	54	95	206	363					727
1956—1960		5	10	19	43	129	235				441
1961—1965			7	9	16	57	131	310			530
1966—1970		1	4	3	12	43	69	192	343		667
1971—1975			2	1	11	16	28	76	188	328	650
Total	14	281	444	571	614	608	463	578	531	328	4,432

^a The table includes only cases in which the year of notification could be confidently confirmed. The number of excluded cases is 172.

categories and the time of diagnosis. By far the largest of these categories were "mining, quarrying, and tunneling" and "iron and steel industries." The category "others" was composed of persons with dust exposure in two or more of these main occupational categories or those whose silicosis was contracted in other occupations. The number of women with silicosis was considered to be too small to warrant special statistical analysis of the sex factor (table 2). In all the tables and figures, therefore, the women are included but are not presented separately.

Distribution of the study population according to age at disease manifestation is shown in table 3. There is a shift towards higher age at the manifestation of silicosis; it is especially pronounced for the period 1961—1975 (mean age 1931—

1950: 49.9 a; 1951—1960: 51.5 a; 1961—1975: 55.6 a).

The duration of exposure to dust preceding the radiological appearance of silicosis is reviewed in table 4. The mean exposure time was 23.1 a in cases with disease manifest in 1931—1950. For the cases from 1951—1960 the corresponding mean value was 23.3, and for those from 1961—1975 it was 26.4 a.

The age at commencement of exposure to silica dust is shown in table 5. In all three periods approximately three-fourths of the affected workers began such exposure before they were 31 a of age. Conversely, commencement of dust exposure after age 50 was rare.

The age when exposure to dust began did not vary appreciably in the different periods of the analysis.

Table 2. Distribution of silicosis cases according to occupational categories and year of disease manifestation. — All occupational categories.

Occupational category	Silicosis manifest									Total	Number of women
	1931—1935	1936—1940	1941—1945	1946—1950	1951—1955	1956—1960	1961—1965	1966—1970	1971—1975		
Mining, quarrying, tunneling	77	243	255	189	255	191	262	279	204	1,955	4
Iron and steel industries	62	167	191	315	254	216	239	196	109	1,749	1
Ceramic industry	121	26	58	57	59	26	39	26	17	429	41
Others	21	26	85	62	58	46	69	63	27	457	6
Total	281	462	589	623	626	479	609	564	357	4,590	52

Table 3. Distribution of silicosis according to age at the manifestation of disease. — All occupational categories. (N = number of cases)

Silicosis manifest	Age (a)										Total (N)
	21—30		31—40		41—50		51—60		≥ 61		
	N	%	N	%	N	%	N	%	N	%	
1931—1950	45	2.3	305	15.6	657	33.6	632	32.3	316	16.2	1,955
1951—1960	12	1.1	125	11.3	347	31.4	427	38.6	194	17.6	1,105
1961—1975	5	0.3	67	4.4	293	19.2	703	45.9	462	30.2	1,530
Total											4,590

Selected occupational categories

The cases of pneumoconiosis in the Pneumoconiosis Register derived from many occupations. The technical conditions of exposure to injurious dust thus varied widely in the total study population, and the characteristics of the dust and patterns of exposure varied accordingly.

A comprehensive account of the technical background of the occupations from which the study population is derived was given in 1960 by Ahlmark et al (3) and by Ahlmark in 1967 (2). The reader is referred to these accounts for detailed technical information.

For an illustration of the developments in some industries, four categories were selected for tabulations concerning the time when dust exposure occurred, the age when the exposure began, and the age at which silicosis became manifest. These categories were as follows: (i) the mining industry (codes 01—18), (ii) the steel industry (codes 32—40, 76), (iii) the iron in-

dustry (codes 41—46), and (iv) the ceramic industry (codes 49—55, 81, 82, 85, 87). The code numbers refer to the standard list of classifications and the occupational codes used for the Pneumoconiosis Register (appendix A).

Each category was selected as being reasonably homogeneous concerning type of exposure to dust and access to the medical surveillance of dust-exposed workers. The latter factor can be assumed to improve health monitoring in risk groups and, consequently, to improve case finding. However, comprehensive case-finding programs came relatively late in the iron industry and resulted in an increased number of notifications in the period 1961—1975.

Mining industry

Pneumoconiosis in Swedish mines arises mainly from the extraction of various ores, predominantly iron but also sulfide ores and others. Drillers, loaders, and haulage

Table 4. Distribution of silicosis cases according to duration of dust exposure preceding the manifestation of silicosis — All occupational categories. (N = number of cases)

Silicosis manifest	Prediagnostic exposure (a)												Total (N)		
	≤ 10		11—20		21—30		31—40		41—50		51—60			≥ 61	
	N	%	N	%	N	%	N	%	N	%	N	%		N	%
1931—1950	269	13.8	587	30.2	587	30.2	349	18.0	132	6.8	18	0.9	2	0.1	1,944
1951—1960	194	17.6	301	27.3	263	23.9	240	21.8	90	8.2	13	1.2	—		1,101
1961—1975	137	9.0	362	23.8	472	31.0	340	22.4	182	12.0	24	1.6	3	0.2	1,520
Total															4,565 ^a

^a In 25 additional cases the duration of exposure could not be ascertained.

Table 5. Distribution of silicosis cases according to age at commencement of dust exposure — All occupational categories. (N = number of cases)

Silicosis manifest	Age at commencement of exposure (a)										Total (N)
	≤ 20		21—30		31—40		41—50		≥ 51		
	N	%	N	%	N	%	N	%	N	%	
1931—1950	699	35.8	834	42.7	304	15.6	97	5.0	20	1.0	1,954
1951—1960	414	37.5	420	38.0	186	16.8	70	6.3	14	1.3	1,104
1961—1975	587	38.5	560	36.7	250	16.4	102	6.7	27	1.8	1,526
Total											4,584 a

^a In six additional cases the age at commencement of exposure could not be ascertained.

workers constitute a large proportion of the mining workers. Other occupations, below or above ground, are also included in this occupational category.

Table 6 (appendix C) reviews age at the first radiographic showing of silicosis. A shift to a higher mean age at the time of diagnosis occurred in the period 1961—1975.

The prediagnostic duration of exposure to dust is presented in table 7 (appendix C) and summarized according to age in table 8 (appendix C).

A slight shift towards longer prediagnostic exposure to dust occurred in the period 1961—1975. Within the age groups ≤ 50 a and ≥ 51 a (age at diagnosis) the mean duration of dust exposure showed no change. There was, however, a distinct shift towards higher age. This shift, entailing a greater proportion of cases with relatively long exposure, is reflected as an increase in the mean duration of exposure for the total occupational category in the period 1961—1975 (mean = 25.8 a). The absolute number of diagnosed cases showed a sharp decrease.

The age at commencement of dust exposure remained fairly constant throughout the study period (mean commencing age 1931—1950: 25.4 a; 1951—1960: 26.1 a; and 1961—1975: 25.2 a).

The latency times according to age group and time are shown in table 9 (appendix C). In the younger of the two age groups there was a tendency towards an increased latency time in the period 1961—1975.

Steel industry

Some of the processes in steel making that are known to involve a risk of pneumoconiosis are the construction and repair of furnaces, work with ladles (receptacles for molten steel), the preparation of sand (for molds and cores), the molding of steel, the knocking out of molds and cores, and the cleaning of steel castings. Table 10 (appendix C) shows the mean age at the manifestation of silicosis in workers with these tasks. A shift to a higher age at diagnosis is clear in 1961—1975. Concurrently there is a distinct decrease in the number of cases diagnosed at that time.

No clear trends emerged as regards duration of dust exposure prior to detection of silicosis (table 11, appendix C). The median exposure times were 19.2 a for 1931—1950, 17.4 a for 1951—1960, and 20.0 a for 1961—1975.

Table 12 (appendix C) shows the prediagnostic duration of dust exposure of cases according to age group and time. In 1961—1975 there was a shift towards a higher age at the manifestation of silicosis. In the older cases there was also a shortening of the mean duration of exposure.

The mean latency time showed some tendency to increase in the younger of the two age groups in table 13 (appendix C). A reverse trend emerged for the older group.

Iron industry

The various processes in the iron industry that involve risk of pneumoconiosis correspond by and large to those in the steel industry. The most important are the construction and repair of furnaces, sand preparation, core making and the molding of iron castings, the knocking out of molds and cores, and the cleaning of iron castings.

Table 14 (appendix C) shows the mean age at the manifestation of silicosis. The only distinct change is a rise of this mean age in the period 1961—1975.

The analysis of duration of dust exposure preceding the detection of silicosis showed no distinct changes with time (table 15, appendix C). The respective medians in 1931—1950, 1951—1960, and 1961—1975 were 30.3, 26.0, and 28.9 a.

Table 16 (appendix C) shows the prediagnostic duration of dust exposure of cases according to age group and time. An increase in the proportion of workers older than 50 a at the time of diagnosis was associated with a shortened mean duration of dust exposure in this older group. There was also a rise in the absolute number of diagnosed cases from iron foundries.

The mean age at the commencement of exposure to silica dust did not increase with time (mean commencing age 1931—1950: 23.6 a; 1951—1960: 24.1 a; and 1961—1975: 23.8 a).

The latency time showed no change in

workers aged ≤ 50 a. In the older group there seemed to be some shortening of latency time (table 17, appendix C).

Ceramic industry

Most of the pneumoconiosis contracted in the ceramic industry derived from potteries (sliphouse, forming shop, furnace, glazing, etc), although the makers of silica brick and of glasshouse pots were also represented.

The mean age at the establishment of the diagnosis rose successively in the study period (table 18, appendix C). There was a great decrease in the number of diagnosed cases.

The prediagnostic duration of exposure to dust is described in tables 19 and 20 (both in appendix C). The shortening of exposure time was clearest in the age group ≤ 50 a. The reduction in the number of diagnosed cases was considerable in this age group.

A slight increase in mean age at the commencement of exposure to dust appeared in the analysis, 2.4 a. The increase, not unexpectedly, correlated with an increase in age at diagnosis. No trends were discernible with respect to latency time (table 21, appendix C). A reduction in the number of diagnosed cases and a shift to a higher age were also seen.

Stages of silicosis

The cases of silicosis and mixed dust fibrosis that were notified between 1931 and 1975 were classified according to the stage of the lesions on the first positive radiographs. The relationship of this initial stage to the age of the workers and to the duration of exposure to dust was analyzed

in the following four groups: (i) the mining industry, (ii) the steel industry, (iii) the iron industry, and (iv) the ceramic industry.

In the following tables the stage of dust disease when first radiologically manifest is correlated to the time of diagnosis, the prediagnostic duration of exposure to dust, and the age of the affected workers. The duration of dust exposure is shown as the weighted arithmetic mean based on tabulations of exposure time with 5-a intervals. The mean age was calculated in 10-a intervals.

Mining industry

Table 22 shows that, among mine workers, the stage of silicosis on the first positive radiograph shifted during the period of the study, the proportion of stage I cases being greatest in the latest years. There was also a substantial reduction in the frequency of stage III.

Table 23 (appendix C) shows the age and length of prediagnostic dust exposure of cases diagnosed in stages I to III.

Steel industry

In the steel industry, as in mining, there was a steady decrease, relative and absolute, of silicosis in stages II or III on the first positive radiographs (table 24). Table 25 (appendix C) shows the age and length of prediagnostic dust exposure of cases diagnosed in stages I to III.

Iron industry

Lesions that have reached stage III at the time of their detection have always been rare in iron foundry workers (table 26). Table 27 (appendix C) shows the age and

Table 22. Stage of silicosis on first positive radiograph — Mining. (Number of cases)

Silicosis manifest	Silicosis stage						Total (N)
	I		II		III		
	N	%	N	%	N	%	
1931—1950	421	71.4	106	18.0	63	10.7	590
1951—1960	241	87.3	27	9.8	8	2.9	276
1961—1975	257	93.1	17	6.2	2	0.7	276

length of prediagnostic dust exposure of cases diagnosed in stages I to III.

Ceramic industry

A shift from more severe silicosis to stage I on the first positive radiographs occurred among workers in the ceramic industry (table 28). The variables age and prediagnostic dust exposure time are shown in table 29 (appendix C).

Summary of results

The tables are predominantly self-explanatory. Since they have been included for

the purpose of description, no attempt is made to give a detailed account of their contents. The general trend of the registered data can be summarized as follows:

- (a) a steady inflow of notifications to the Pneumoconiosis Register as the study period progressed,
- (b) a rising mean age, mean length of prediagnostic dust exposure and mean latency time,
- (c) a successive reduction in the proportion of workers with stage II or III on the first radiographs showing silicosis.

Table 24. Stage of silicosis on first positive radiograph — Steel industry. (N = number of cases)

Silicosis manifest	Silicosis stage						Total (N)
	I		II		III		
	N	%	N	%	N	%	
1931—1950	292	71.0	72	17.5	47	11.4	411
1951—1960	155	86.6	23	12.8	1	0.6	179
1961—1975	153	97.5	4	2.5	—	—	157

Table 26. Stage of silicosis on first positive radiograph — Iron industry. (N = number of cases)

Silicosis manifest	Silicosis stage						Total (N)
	I		II		III		
	N	%	N	%	N	%	
1931—1950	244	88.7	23	8.4	8	2.9	275
1951—1960	249	93.6	16	6.0	1	0.4	266
1961—1975	309	91.4	28	8.3	1	0.3	338

Table 28. Stage of silicosis on first positive radiograph — Ceramic industry. (N = number of cases)

Silicosis manifest	Silicosis stage						Total (N)
	I		II		III		
	N	%	N	%	N	%	
1931—1950	157	59.9	72	27.5	33	12.6	262
1951—1960	68	79.1	17	19.8	1	1.2	86
1961—1975	76	92.7	5	6.7	1	1.2	82

Discussion

The trends with regard to age, prediagnostic duration of dust exposure, latency time, and number of diagnosed cases of silicosis are all consistent with diminishing silicosis risk. Improved environmental conditions, with a resultant reduction in disease impact, can contribute to such trends. Before this explanation is considered, some other factors must be recognized, since they may determine the detection of silicosis and thus also influence the characteristics of individual cases.

First, silicosis — like many other forms of pneumoconiosis — is generally detected in a clinically or subjectively silent stage. The incentives to seek medical attention for any insidious disease vary considerably. The exposed person's awareness of the occupational risks and his general attitude towards medical consultations, including expectations of their benefits, are influential factors.

Second, the likelihood of detection is dependent on the thoroughness and the comprehensiveness of the case-finding procedures used for dust-exposed persons.

Third, examination does not guarantee diagnosis. The recognition of silicosis is dependent upon the standards of diagnosis and the training and attitudes of the consultant physician and radiologist. Technical radiographic standards are also important in this respect.

From the biological viewpoint, the result of exposure to silica dust is not simply silicosis or no silicosis. According to the type of exposure and the individual characteristics of exposed persons, the dust evokes varying degrees of pulmonary fibrosis. The persons, in a dust-exposed population, who are considered to have silicosis depends in practice primarily on the sensitivity and specificity of the applied diagnostic techniques.

The three aforementioned factors may operate selectively so that parts of the parent population, ie, the exposed population from which the cases are drawn, may evade the detection of pneumoconiosis. Even in industries that provide medical care for employees, one cannot safely assume that all persons at risk are under constant medical surveillance throughout their lifetime. Health monitoring is likely

to be more efficient however when such medical services are available.

Nevertheless it may reasonably be assumed that improvements in diagnosis, a better understanding of risks, an increased subject awareness of risks, and increasingly energetic case-finding programs have all contributed to the persistently high numbers of notifications.

A relevant point in this connection is that in 1938 periodic medical examinations (including radiography) of persons exposed to work environments implying risk of pneumoconiosis became compulsory by statute in Sweden. In practice, this legal requirement gradually began to be implemented during the first half of the 1940s.

Factors which cannot be elicited from a case register are the size, composition, and mobility of the total risk population from which the notified cases of pneumoconiosis are derived. Changes in this population may be reflected in an apparently altered incidence of the disease, even if the disease-producing environment is unchanged and the standards of case finding and case notification are constant.

Increase in the size and mobility (turn-over rate) of a population at risk may result in a reduced incidence of dust disease. In addition, if the dusty occupation is exchanged for safer work after a period of exposure sufficient to produce pneumoconiosis, later manifest lesions may escape detection.

For these reasons, the trends in age, prediagnostic exposure, and latency time should be interpreted with caution. Latency time, for instance, can be influenced by many factors. From the biological aspect, it is plausible that a lower grade of exposure can result in a prolongation of this latency time, ie, slower development of pneumoconiosis. Latency time and, of course, prediagnostic duration of exposure, are also dependent on the efficiency of diagnosis and medical surveillance. Many of the cases diagnosed in the 1930s and 40s might have been detected earlier if the subsequent diagnostic improvements had been available and if the case-finding programs had been as comprehensive as they were later to become. The shortening of intervals between periodic examinations tends to shorten the latency period. On the other hand, the mean latency time

may be expected to increase if surveillance programs are extended to low-risk occupations, since the proportion of cases with slowly developing lesions presumably then increases. Since it was not possible to control such factors in the present study, the data on latency time and prediagnostic exposure should be cautiously interpreted.

It is also recommendable to view the decreasing number of diagnosed cases of silicosis with some suspicion. Many cases are retrospectively diagnosed and classified (table 1). If the time pattern observed before 1975 for the receipt of notifications and the establishment of disease manifestation time is projected to future years, one may reasonably expect that the figures for 1961—1975 will be added to in the coming decade. The reduction in number of manifest cases may then become less impressive and may indeed be eradicated.

For the same reason, a reservation should be made regarding the shift in mean age and duration of dust exposure. Retrospective detection of silicosis tends to reduce the age at diagnosis when compared with the age in cases in which diagnosis and notification are based on the same film. The prediagnostic dust exposure likewise tends to be shorter in the former group.

The differences in age and prediagnostic

exposure time between cases manifest in 1931—1950 and cases from 1961—1975 may therefore be reduced to some extent in the future if there is a substantial inflow of cases diagnosed in retrospect.

Despite these sources of error the tendency towards a high mean age at the time of diagnosis of silicosis should serve as a reminder that silicosis can be a late phenomenon. If the true incidence of silicosis is to be determined for a dust-exposed population, particular attention must be paid to older employees and exemployees. Many cases of silicosis may be found among such groups long after the exposure to dust has ceased.

Concluding remarks

The observed trends in age, prediagnostic duration of dust exposure, latency time, and disease stage at diagnosis are, in themselves, consistent with the concept of an improving environment producing less disease. There are, however, other factors which could have produced the registered trends even if the environment had remained unchanged. Only truly population-based analyses can conclusively demonstrate the influence of these factors and disentangle them from the results of reduced exposure to the hazardous dust.

Section III

Radiographic progression of silicosis

In 1976 Reichel (39) reviewed the available information on the natural course of silicotic disease. His review emphasized the unpredictability of the pathological process. Although differences in the nature of silicotic disease according to occupation are well known, the factors that determine whether the lesions will progress or remain stationary are incompletely understood. It is widely recognized, however, that exposure to dust with a high quartz content is conducive to rapidly progressive disease.

In the present section, two aspects of radiographic progression have been examined. First, cases of silicosis with and without radiographically verified progression are compared with regard to age at the time of the first radiographic evidence of silicosis, the duration of exposure to silica dust at that time, and the latency time (from commencement of exposure to manifestation of silicosis). Second, cases from the Pneumoconiosis Register with a 20-a follow-up after diagnosis are analyzed with regard to radiographically verified

progression during the period of observation.

Material

The study populations in both these analyses were extracted from the Pneumococcosis Register. The ordinary follow-up routines for cases with diagnosed silicosis include a periodic radiographic examination at 3- to 5-a intervals. The results of these examinations are assessed and recorded in the Register. Silicosis progression, in the context of the present study, was considered the advancement of radiographic pulmonary lesions from stage I to stage II or III.

The comparisons between progressive and nonprogressive silicosis were restricted to cases from the mining and steel industries (codes 01—18 and 32—40, 76), ie, from industries with periodic examinations of dust-exposed persons that could be expected to reveal silicosis in an early stage of development. Only cases in which silicosis had not reached more than stage I at diagnosis were included in the analysis. Persons with known tuberculosis during the observation period were excluded because of the tendency of tuberculosis to promote the progression of silicosis. At least one radiographic examination after the first appearance of silicosis was required for admission to the study. The observation period extended from 1931 through 1975.

The analysis of long-term follow-up comprised silicosis diagnosed between 1931 and 1950 in workers from the following occupational groups: (i) the mining industry (codes 01—18), (ii) quarrying, tunneling, etc (codes 19—31, 84), (iii) the steel industry (codes 32—40, 76), (iv) the iron industry (codes 41—46), and (v) the ceramic industry (codes 49—55, 81, 82, 85, 87). Admission to this part of the study also required a diagnosis of silicosis in stage I and an absence of known pulmonary tuberculosis.

Results

The cases with and without progression in the comparative analysis are shown jointly

in tables 30—33 (appendix C). Tables 30 and 31 concern cases from the mining industry, and tables 32 and 33 cases from the steel industry. In both the mining and steel industries, the cases of progressive silicosis tended to have a lower mean age at diagnosis, a shorter prediagnostic dust exposure, and a shorter latency time than the cases without silicosis progression. The trends were similar in both occupational categories even though there was a considerable spread of the values around the means.

An apparent decline in the frequency of progressive silicosis occurred during the observation period. The decline was particularly obvious for the steel industry, for which the ratio of progressive to nonprogressive silicosis cases fell from approximately 2:1 in 1931—1950 to 0.4:1 in 1951—1975.

The second analysis, the 20-a follow-up, concerned silicosis diagnosed between 1931 and 1950 in stage I and without known tuberculosis in the observation period. Tables 34—38 and fig 1—5 present the findings (tables 35—38 & fig 2—5 in appendix C). The observations in each case began with the first radiograph showing silicosis. The tables give figures for progression at 5-a intervals from admission to the study. The number deaths in each interval is also shown.

Fig 1—5 illustrate the total number of survivors at the end of each 5-a interval and also the number of survivors with known progression of silicosis. The cumulative percentage of radiographically registered progression to stage II or III over the 20 a, including progression known to have occurred in workers who died in the respective 5-a intervals, is also shown. The mortality and nonattendance at follow-up (tables 34—38 & fig 1—5) resulted in substantial diminution in the numbers of observed cases, particularly towards the end of the 20-a period.

Among the mining workers there was substantial cumulated progression to more advanced disease, 151 of 363 or 41.6 % (fig 1 & table 34). Among the 252 deaths at the end of the observation period were 105 cases with radiographically verified progression. Six of the persons listed as nonattenders had shown progression in previous examinations.

For workers who had contracted silicosis in quarrying, tunneling, and stone working, the cumulated percentage of silicosis progression at the end of the observation period was higher than that for the mining workers, 46 of 65 or 71.8 % (table 35 & fig 2, appendix C). A similar cumulative frequency was found for workers from the steel industry, 162 of 241 or 67.2 % (table 36 & fig 3, appendix C). A much lower rate of progression, 57 of 219 or 26.0 %, was found for the iron industry workers (table 37 & fig 4, appendix C). In the ceramic industry progression to stage II or III was frequent, 92 of 131 or 70.2 % (table 38 & fig 5, appendix C).

A notable observation was that progression to the most advanced stage of silicosis

was relatively rare among workers who had contracted silicosis in the iron industry (13 of 57 or 23 %). The corresponding percentage for cases from the mining and steel industries were 42 and 46, respectively.

In addition to these two analyses, cases of silicosis diagnosed after 1950 (stage I at diagnosis and no known tuberculosis in the observation period) were followed for at least 20 a. The cumulated known progression at the end of the 20-a observation period was as follows:

Mining	30 of 65 or 43 %
Steel industry	15 of 30 or 50 %
Iron industry	9 of 48 or 19 %
Ceramic industry	8 of 13 or 62 %

Summary of results

The analysis of silicosis progression in cases from five occupational categories diagnosed in 1931–1950 showed differences in the frequency of progression after 20 a of observation. The lowest cumulated rate was observed in the iron industry workers (26 %). High rates (around 70 %) were found for workers from the steel and ceramic industries. A similar long-term analysis of silicosis diagnosed after 1950 resulted in progression rates of similar magnitude. These estimates are, however, less precise because of the small number of cases.

Fig. 1. Progression of stage I silicosis. Height of columns shows number of survivors after 5-a intervals. [□] number without progression, [▨] number with radiographic progression (from I to II or III), ● cumulative percentage of known radiographic progression including the progression in subjects who died in the 5-a interval and the progression in nonattenders]

Discussion

The tendencies as regards differences in age, exposure time, and latency time between progressive and nonprogressive sili-

Table 34. Progression of stage I silicosis in 20 a of follow-up — Mining. (Mean age at start of observation = 48.7 a, SD = 9.7)

Observation time (a)	Silicosis stage			Cumulated deaths	Nonattendance at follow-up ^a
	I	II	III		
0	363				
5	314	31	3	15	—
10	250	47	17	47	2
15	180	52	35	89	5 (1)
20	36	25	15	252	28 (5)

^a Numbers within parentheses give number of cases with known progression among nonattenders.

cosis were similar for mining and steel workers. The sources of the differences could have been:

- (a) the characteristics of the dust exposure (type, intensity, etc);
- (b) the biological susceptibility of the exposed persons.

Rapid progression of silicosis is indicative of a serious pathological course of the disease. It may arise from high-grade exposure to silica dust. It may also occur in persons especially susceptible to the inhaled dust. Available Register data on dust exposure did not permit analysis of the environmental conditions for the progressive and nonprogressive groups. The data are therefore presented for descriptive purposes only.

Differences in the progression of silicosis in comparisons between occupational categories are documented in the literature (33). Thus the findings in this study were not unexpected. In a long-term analysis, however, depletion of the study population by deaths from intercurrent causes can create difficulties. Death and silicotic progression are competing events, and this competition should be kept in mind when the data are interpreted. The cumulative rate of known progression should preferably be regarded as a minimum esti-

mate, particularly towards the end of the observation period.

For cases diagnosed in 1931—1950 and followed for 20 a, the cumulated rate of the progression of silicosis from stage I to stage II or III was about 40 % in the mining industry and about 70 % in quarrying and tunneling, in the steel industry, and in the ceramic industry. Among cases contracted in the iron industry, with exposure to dust of mixed type, the cumulated rate was roughly 25 %. The transition from mild to more advanced disease seemed to follow a linear trend, the frequency of progression increasing with time after diagnosis. Available data from the follow-up of cases diagnosed after 1950 did not indicate significant changes in progression rates.

Concluding remarks

Radiographic progression of silicosis may occur long after diagnosis.

There are substantial differences between occupational groups with regard to the tendency to undergo radiographic progression of silicosis.

The tendency towards radiographic progression was considerable also in cases diagnosed after 1950.

Section IV

Tuberculosis

The literature on the well-known association between tuberculosis and pneumoconiosis was reviewed and commented on by Bailey et al in 1974 (9), Worth & Stahlmann in 1976 (47), and Parkes in 1974 (36). Additional references to the extensive literature on this subject can be obtained from these reviews. In 1967 Bruce (13) reported the situation with regard to silicosis and complicating tuberculosis in Sweden.

Experimental evidence and clinical experience have thus shown that pulmonary

lesions caused by silica dust may predispose to tuberculosis of the lungs. The precise mechanism of this process is not known, although several plausible hypotheses have been advanced. The association between silicosis and tuberculosis is not unidirectional however. There is evidence from animal experiments that the course of induced tuberculosis is unfavorable if exposure to fibrogenic dust is concomitant. This conclusion tallies with general clinical experience. Tuberculosis and silicosis in combination tend to produce ex-

tensive pulmonary fibrosis. In the literature, too, there seems to be a consensus that the coexistence of silicosis and pulmonary tuberculosis may have a synergistic effect on the fibrous tissue reaction. In explaining this effect, some writers have emphasized the destruction of cells and tissues and the proliferation of fibrous tissue evoked by quartz particles. Others have drawn attention to the obstruction of lymph channels caused by the dust and the ensuing hindrance to the lymphatic clearance of infectious material from the lungs. Other suggested mechanisms are altered properties of tissue macrophages in response to tuberculous infection and silica dust.

In this section some aspects on the association between silicosis and tuberculosis are demonstrated. They concern the distribution of known tuberculosis in the study population diagnosed from 1931 through 1975, the time relationships between silicosis and complicating tuberculosis, and a comparison of silicosis cases with and without complicating tuberculosis with regard to age, duration of dust exposure, and latency time (from commencement of exposure to diagnosis of silicosis).

Material

In the first analysis the whole study population was used. It was divided according to the time periods 1931—1950, 1951—1960, and 1961—1975. Furthermore, it was divided according to the following occupational categories: (i) mining, quarrying, and tunneling (codes 01—31), (ii) the steel industry (codes 32—40, 76), (iii) the iron industry (codes 41—46), (iv) the ceramic industry (codes 49—55, 81, 82, 85, 87), and (v) others.

In the second analysis the tuberculosis risk in the study population was compared with the incidence rates of tuberculosis for the country as a whole. For this analysis all cases of silicosis diagnosed from January 1951 through December 1969 were extracted from the aforementioned occupational categories.

In the third analysis cases from the mining industry (codes 01—18) were selected.

The usual follow-up routine of silicosis in Sweden includes pulmonary radiographs at intervals of 3 to 5 a. The radiographs are centrally assessed, and the results are recorded in the Pneumoconiosis Register. Additional examinations are frequently ordered, particularly if there is any suspicion of complicating pulmonary disease.

The almost invariable criterion for pulmonary tuberculosis in the present study was demonstration of tubercle bacilli in sputum or gastric washings at any time during the observation period. In the first half of the 1930s such demonstration was achieved with the direct microscopic technique. From the middle of the 1930s a positive bacteriologic culture test or positive guinea-pig test was required for verification. In the few exceptional cases in which tuberculosis was diagnosed without bacteriologic evidence, typical cavitary lesions were present in pulmonary radiographs. The diagnoses of tuberculosis were made by the same specialist responsible for the assessments of silicosis.

Methods

In the second type of analysis the data compiled by the Swedish Tuberculosis Index were used to calculate general popu-

Table 39. Silicosis plus tuberculosis, distribution according to stage of silicosis on first positive radiograph — All occupations.

Silicosis manifest	Stage I			Stage II			Stage III			Total		
	Total	TB	%	Total	TB	%	Total	TB	%	Total	TB	%
1931—1950	1,351	154	11.4	384	80	20.8	219	36	16.4	1,954	270	13.8
1951—1960	950	81	8.5	137	12	8.8	19	2	10.5	1,106	95	8.6
1961—1975	1,351	41	3.0	143	14	9.7	30	2	6.8	1,524	57	3.7
Total										4,584	422	9.2

lation incidence rates as regards tuberculosis. The Index comprises all notifications of tuberculosis. Notification of tuberculosis is compulsory by statute. The functions of the Index are described and its routine statistics are published in annual reports (41).

The material from the Tuberculosis Index used in this study comprised notifications from 1951 on, specified according to sex and age (5-a intervals). On the basis of the annual sex-specific and age-specific rates in the general population, the expected rates in the study population could be calculated for comparisons with the observed rates.

A life-table technique was used in the analysis of the data. The occurrence of tuberculosis was regarded as the health end-point. A detailed account of the method is given in appendix B.

The study population in this analysis consisted of all cases in which silicosis was established as manifest from January 1951 through December 1969. The occupation, age, date of silicosis manifestation, and findings at follow-up, including intervals between detection of tuberculosis and death were the data used in the analysis. The closure date for follow-up was 31 December 1970.

Results

The results of the first step of the analysis are presented in tables 39—44. Table 39 presents the cases of silicosis complicated by tuberculosis in the whole study population. Tables 40—44 (appendix C) show data on the five mentioned occupational categories. The tables show the total number of silicosis cases according to the stage of this disease on the first positive radiographs and the absolute and percentage frequencies of tuberculosis in the respective categories.

The six tables show a progressive and steady decrease in the incidence of tuberculosis. This decrease was both absolute and proportional to the number of silicosis cases. However, even in the period 1961—1975, cases with complicating tuberculosis were still being notified.

Stages II and III of silicosis showed a greater tendency than stage I to be com-

plicated by tuberculosis. This finding was conspicuous in the early years. As the number of cases of advanced silicosis diminished, the tendency towards complicating tuberculosis became less prominent.

The results of the second step of the analysis, the comparison of incidence rates for tuberculosis in the study population and the general population, are presented in tables 45—50 and fig 6—10.

Table 45 presents the total study population. The column "Number entering" shows the total number of persons observed in the respective intervals, the year of entry being that when silicosis was diagnosed.

All entries were calculated from July 1st of the first year of observation. The observed number of tuberculosis cases each year is also shown. These numbers are expressed in table 46 as observed tuberculosis risk. They can be compared with figures for expected risk, derived from data published by the Swedish Tuberculosis Index. Observed risks are given as smoothed values (3-point moving average).

Fig 6 illustrates the observed and expected percentages of the study popula-

Table 45. Analysis of tuberculosis risk. Numbers of observed persons and observed cases with tuberculosis — All occupational categories.^a

Observation time (a)	Number entering	Observed number of tuberculosis cases
0—1	2,228	23
1—2	1,861	14
2—3	1,847	5
3—4	1,715	5
4—5	1,602	8
5—6	1,476	8
6—7	1,365	6
7—8	1,218	8
8—9	1,075	7
9—10	951	4
10—11	824	5
11—12	705	5
12—13	610	4
13—14	530	2
14—15	438	2
15—16	366	0
16—17	294	2
17—18	221	2
18—19	154	2
19—20	100	0

^a Date of entry = year of established silicosis diagnosis.

tion that remained free from manifest tuberculosis during the period of observation. The 10-a intercept of the curve for observed freedom from tuberculosis lies at 93.9 %. The interval estimate [± 1.96 standard error (SE)] is 92.7—95.1 %. The intercept for expected freedom from tuberculosis lies at 99.1 %.

The incidence of tuberculosis was thus higher in the study population than in the general population. The data in table 45 and in fig 6 also show that the manifesta-

tion of tuberculosis often came late in the course of silicosis.

Corresponding data on tuberculosis, but confined to workers in the mining industry, are presented in tables 47 and 48 (both in appendix C) and fig 7 (appendix C). The trends among these workers resembled those of the total study population. Tuberculosis could appear after many years of observation. In fig 7 the 10-a intercept of the curve for observed freedom from tuberculosis is at 94.4 %. The interval

Table 46. Analysis of tuberculosis risk. Calculated observed and expected risks. Observed risk values smoothed (3-point moving average) — All occupational categories.

Observation time (a)	Observed tuberculosis risk (%)	Expected tuberculosis risk (%)	Ratio observed/expected
0—1	—	0.098	—
1—2	0.736	0.096	7.68
2—3	0.463	0.093	4.97
3—4	0.385	0.091	4.23
4—5	0.484	0.089	5.45
5—6	0.589	0.087	6.80
6—7	0.648	0.086	7.57
7—8	0.667	0.085	7.86
8—9	0.649	0.085	7.64
9—10	0.634	0.085	7.48
10—11	0.657	0.084	7.85
11—12	0.761	0.082	9.24
12—13	0.681	0.081	8.40
13—14	0.594	0.080	7.44
14—15	0.303	0.079	3.82
15—16	0.423	0.080	5.32
16—17	0.612	0.079	7.65
17—18	1.391	0.080	17.38
18—19	1.133	0.083	13.65
19—20	—	0.086	—

CASES ADMITTED 1951-1974

N=2229

Fig 6. Observed and expected freedom from tuberculosis in the total analyzed group of silicosis cases.

estimate (± 1.96 SE) is 92.0—96.8 %, and the 10-a intercept for expected risk 99.1 %.

The data from the iron industry workers are given in tables 49 and 50 (both in appendix C) and fig 8 (appendix C). They fit well with the general trend (10-a intercept of observed probability or risk curve at 94.7 %, interval estimate 92.5—97.0 %, intercept of expected risk at 99.1 %).

Freedom from tuberculosis among the steel industry workers with silicosis is diagrammatically presented in fig 9 (appendix C). The data from the stone quarry workers are illustrated in fig 10 (appendix C). For the steel workers (N = 165) the trend was in line with that of the previous groups, with the 10-a intercept of the observed curve at 96.6 % (93.8—99.9 %). Among the quarry workers (N = 189) there was some deviation from the trend of the total study population, with the intercept of the observed curve at 83 %, the interval estimate at 75.8—90.2 %, and the 10-a intercept of expected risk at 99.1 %.

In the third step of the analysis, a subcategory was extracted from the broad occupational category mining, quarrying, tunneling, etc. This subcategory comprised cases from the mining industry, which to a large and increasing extent has provided medical services for its employees from about the middle of the 1930s on.

Table 51 shows the age when exposure to silica dust began, the age when silicosis was detected, and the preceding duration of exposure to the dust among mine workers with and without tuberculosis. Only cases with silicosis which had not progressed beyond stage I on the first positive radiographs were included. The analysis was also limited to workers who were radiographically reexamined at

least once after the manifestation of silicosis. The figures are weighted arithmetic means (groups with 10-a intervals for age at diagnosis and at commencement of dust exposure and with 5-a intervals for the duration of exposure).

Table 51 shows that the cases with tuberculosis did not appreciably differ from those without in regard to age at detection of silicosis, duration of exposure to dust, and age at first exposure. During the study period the upward shift in mean age and mean prediagnostic exposure time was similar in both groups.

The latency time for silicosis in cases with complicating tuberculosis (TB +) did not differ from the latency time in cases without known tuberculosis (TB —). For TB + cases in 1931—1950 (N = 43) the mean latency time was 24.4 a, and in 1951—1975 (N = 25) it was 26.8 a. The corresponding mean values for TB — cases were 23.6 and 26.0 a.

Summary of results

The overall proportion of cases developing pulmonary tuberculosis in the study population diminished from 14 to 4 % between 1931—1950 and 1961—1975.

Manifest pulmonary tuberculosis showed a steadily decreasing incidence among persons with silicosis contracted in all the studied occupations. The trend was irrespective of the stage of silicosis at diagnosis.

A concurrent reduction in the number of cases initially presenting stage II or III made estimates of the occurrence of tuberculosis in such cases somewhat unreliable from 1951 on. During the period 1931—

Table 51. Some characteristics of stage I silicosis with and without tuberculosis. Means of age, duration of dust exposure, and age at first exposure — Mining.^a

Silicosis manifest	Number of cases		Age at diagnosis (a)				Dust exposure (a)				Age at first exposure (a)			
	TB+	TB—	TB+	SD	TB—	SD	TB+	SD	TB—	SD	TB+	SD	TB—	SD
1931—1950	43	378	49.5	9.8	48.3	10.0	22.2	11.8	21.4	10.2	26.0	6.8	25.4	6.5
1951—1960	16	225	49.9	13.2	49.4	9.5	19.6	10.6	20.7	10.6	26.6	6.1	25.9	6.6
1961—1975	9	248	55.5	8.7	54.0	7.2	28.0	10.6	26.1	9.9	24.4	7.2	25.3	6.7

^a TB+ = with tuberculosis, TB— = without tuberculosis.

1950 tuberculosis was more common when silicosis was detected in stage II or III than in stage I.

The crude annual risk ratio for tuberculosis incidence (observed/expected) in the population, notified in 1951–1969, was roughly 6–7.

The risk of manifest tuberculosis seemed to vary between occupational categories. A high rate of tuberculosis was observed among quarry workers. A lower rate was found among cases from the steel industry. The analysis of the time relationship between silicosis and tuberculosis showed that tuberculosis may appear late in the course of silicosis.

A special analysis of the mining industry showed that workers with silicosis and tuberculosis did not differ from those with silicosis alone with regard to duration of dust exposure preceding the detection of silicosis, age at this time, and latency time.

Discussion

In studies of the associations between silicosis and tuberculosis the difficulties in achieving diagnostic accuracy should be recognized. It is often difficult to distinguish between silicosis alone and silicosis complicated with tuberculosis. To achieve this goal, bacteriologic and radiographic examinations are of decisive importance. Even with the great technical advances of the past decades, however, the radiographic differential diagnosis may still present insurmountable problems. The lesions of silicosis or tuberculosis, or of the two combined, may look very much alike, and supervening tuberculosis may be indistinguishable from silicosis progression. The summary effects of radiographic densities, which may individually be small, can also create interpretation difficulties which cannot be solved even with optimum radiographic techniques (28). Bacteriologic examination is indispensable for high diagnostic accuracy.

Furthermore, when the incidence of manifest tuberculosis in a study population such as the present one, or in categories of it, is considered and when the risks of contracting tuberculosis are compared with those for the population as a whole,

the following factors should be taken into account:

1. The incidence rate is related to the quality and extent of case finding and of bacteriologic examinations.
2. Statistical estimates of annual risks of contracting tuberculosis in a country's total population cover many subgroups with varying degrees of risk.
3. Some cases of tuberculosis presumably remain undetected, or are not notified, inadvertently or because the lesions have healed without treatment.

The problems associated with studies of the epidemiology of tuberculosis were reviewed in 1978 by Styblo (40). Experience from a national tuberculosis register in Denmark was published in 1978 by Horwitz (22).

When two separate sources of information are used in a comparison of the observed and expected incidence of a disease, differing characteristics of these sources may suggest discrepancies in incidence where none, in fact, exist. For instance, substantial underreporting to a register such as the Swedish Tuberculosis Index will lead to underestimates of the true risks in the general population. Stricter criteria for the diagnosis of tuberculosis in the study population than those for the general population will lower the ratio of observed to expected cases and thereby give rise to an underestimate of the risk in the study population.

Duplicate notifications to a tuberculosis register can, unless there is strict control, result in excess registrations and, thus, overestimated risks in the general population. Although the Swedish Tuberculosis Index routinely checks for such duplication, the same person may have more than one entry; for instance when a name is removed from the register because tuberculosis has healed, but is reentered because of a recurrence of tuberculosis. The number of notifications of active tuberculosis in 1977 was 1,105. No less than 254 (23 %) of these were reregistrations following a recrudescence of the disease. This practice slightly raises the figures for the incidence of tuberculosis in the general population. In the present study, tuberculosis occurring in a case of silicosis

constituted a health end-point in the analysis; in other words, it was registered only once. This difference also tended to bias the ratio of observed to expected tuberculosis downwardly.

For the purposes of this analysis it was assumed that the completeness of notifications to the Tuberculosis Index was adequate enough to permit reasonably unbiased estimates of population risks. A highly relevant point in this connection is that the diagnostic criteria for tuberculosis were stricter for the study population than for the general population. In the cases notified to the Swedish Tuberculosis Index, the diagnosis has frequently been made without positive bacteriologic evidence. The diagnosis thus may be based solely on the radiograph changes in the lungs, which constitute sufficient grounds for notification to the Index. In 1977, for instance, bacteriologic studies were negative for 21.9 % of the men and 20.9 % of the women notified to the Index as cases of postprimary pulmonary tuberculosis. In the study population, by contrast, the diagnosis of tuberculosis was primarily based on bacteriologic findings. In the few bacteriologically negative cases, typical cavitory lesions were the radiographic requirement for diagnosis. Such differences in diagnostic criteria tend to inflate the figures for risk in the general population and, conversely, to minimize them in the study population.

All tuberculosis in the silicotic subjects was of the postprimary pulmonary type. The expected incidence, however, was based on all cases of tuberculosis notified to the Tuberculosis Index. In 1977 postprimary pulmonary tuberculosis was the diagnosis in 71 % of the notified cases. If this figure is representative for the total study period, the reported ratios of observed to expected incidence of tuberculosis were corresponding underestimates of the true ratios for postprimary pulmonary disease.

When all these factors are taken into account, it can confidently be argued that the reported differences between the observed and expected incidence of manifest pulmonary tuberculosis reflect true

and substantial differences, not artifacts. It is, indeed, recommendable to regard the ratios of observed to expected incidence as minimum estimates.

Tuberculosis thus continues to be a frequent and important complication of silicosis. One of its notable features is late appearance in a considerable number of cases. Therefore the reported frequency of tuberculosis among cases of silicosis manifest in 1961—1975 is likely to be an underestimate. Many of these workers have been observed only for a few years. Among them the proportion contracting tuberculosis will probably increase in the course of time. A practical implication of this late appearance of tuberculosis is that, in order to permit early detection of complicating tuberculosis, persons with silicosis must undergo long-term health monitoring. Monitoring programs should preferably be lifelong. The heightened tuberculosis risk in silicosis applies even when the risk of tuberculosis in the general population is fairly low. The contagious nature of tuberculosis is an added reason for health monitoring in cases of silicosis. Every new case of tuberculosis is a potential source of infection in a general population with low immunity.

Concluding remarks

Tuberculosis remains a relatively frequent complication of silicosis, even after 1951, despite the declining rate of tuberculosis in the general population.

For workers with silicosis diagnosed from 1951 on and followed through 1970, the overall risk of contracting tuberculosis was seven times greater than for the general population. In view of the nature of available tuberculosis statistics this figure is best regarded as a minimum estimate. The true risk is probably greater—10- to 20-fold.

The tuberculosis risk for silicosis cases varies between occupational categories.

Complicating tuberculosis may occur late in the course of silicosis.

Section V

Mortality

The serious and indeed potentially fatal nature of silicosis has been amply documented in the literature. [For references the reader can consult Morgan & Seaton (33)]. In view of the substantial technical changes in the environment over the past decades, an analysis of mortality patterns in the study population seemed to be warranted.

The importance of the following factors to the mortality pattern was studied: (i) occupational category, (ii) stage of silicosis at notification, and (iii) suspected or verified tuberculosis during the observation period.

Material

The analysis was limited to part of the total study population, ie, to the cases of silicosis notified from 1931 through 1969. The cases were followed to the end of 1969. The occupational distribution of

these 3,610 cases is presented in table 52.

The cases were grouped according to whether or not radiographic changes leading to a suspicion of pulmonary tuberculosis appeared during the observation period (table 53). It should be observed that this radiographic criterion is different from the more strict criterion of a positive bacteriologic finding used in the preceding section on tuberculosis.

In the majority of cases with such suspect radiographic findings, the tuberculosis could not be verified with positive findings in bacteriologic examinations. Therefore the category TB + in all tables and figures in this section contains both cases with verified pulmonary tuberculosis and cases in which tuberculosis was only radiographically suspected. The purpose of using this rather broad definition of tuberculosis was to obtain a means for analyzing categories of silicosis in which the existence of complicating tuberculosis could be excluded with a high degree of confidence.

The stage of silicosis was defined as the stage of disease at notification (I or II—III).

There were 24 postmortem notifications, all notified within 12 months of death. Of these 24 deaths, 22 were due to tuberculosis and 2 to lung cancer.

Methods

In the analyses of mortality two methods were used. The first method entailed the observation of the number of deaths in the study or index population and a comparison of this number for each of follow-up with the expected number of deaths. The expected number of deaths was calculated with the aid of official Swedish mortality statistics covering the period 1901—1969. The second method was actuarial, of the type usually referred to as a life-table method. The probability of survival or death in each year of follow-up

Table 52. Silicosis population in the mortality analysis — All occupational categories.

Occupational category	Number of cases
Mining, quarrying, tunneling, etc	1,420
Steel and iron industries	1,455
Ceramic industry	384
Other industries or dust exposure in more than one of the above groups	351
Total	3,610

Table 53. Distribution of silicosis cases according to occurrence of suspected or verified pulmonary tuberculosis.

Cases reported	No tuberculosis	Tuberculosis
1931—1948	720	343
1949—1969	1,860	687
Total		3,610

was the basic variable. Observed survival or — conversely — mortality was compared with the expected values, calculated from the official statistics. A detailed account of the methods can be found in appendix B.

In the analyses the study population was divided into two time periods according to the time of notification. The chronological division 1931—1948 and 1949—1969 was chosen to coincide approximately with the major breakthrough of specific antituberculous chemotherapy in Sweden.

In all analyses of mortality, the date of admission was that of the notification of pneumoconiosis, according to legal requirements and not, as in the foregoing sections, the date of the first radiograph showing pulmonary lesions.

All cases in the study population were followed through December 1969. There were no losses to follow-up.

Results

The cumulative mortality in the total study group is illustrated in fig 11—14 without regard to occupation. In these diagrams the study population is divided according to time of admission (1931—1948 and 1949—1969) and presence or absence of suspected or verified tuberculosis. The cumulative entries into the study group are shown.

Fig 11—14 show that, with the mortality in the general population as the reference standard, the cumulative mortality rate of the study population was higher than the expected rate. The difference was greater for the cases admitted to the analysis in 1931—1948 than for the 1949—1969 admissions. The cases in which silicosis was complicated by suspected or verified tuberculosis show particularly large differences between observed and expected mortality rates, though the difference in the period 1949—1969 was less than in the earlier period.

The mortality rate of some occupational categories is presented in a condensed form in tables 54—56 (appendix C). The subgroups were categorized according to time of admission to the analysis, stage of silicosis on admission, and whether or

not complicating pulmonary tuberculosis appeared or was suspected. The tables show the observed and expected death rates for 10-a periods and also the ratios between these figures. The differences between the observed and expected mortality rates were greater for cases which had reached stage II or III at the time of admission to the study than for those with only stage I.

The data in table 54 (appendix C) indicate suspected or verified tuberculosis and radiographic stage of silicosis as risk factors of mortality for mining, quarrying, and tunneling workers. The coexistence of an advanced stage of silicosis and suspected or verified tuberculosis enhances the impact on mortality.

Among the steel and iron industry workers, the difference between the observed and expected mortality rates tended to diminish with time (table 55, appendix C). Once again, concomitant suspected or verified tuberculosis and advanced silicosis was associated with increased mortality.

The observed and expected death rates of mining, quarrying, and tunneling workers admitted in 1931—1948 with silicosis stage II or III and with suspected or verified tuberculosis at some time in the observation period are illustrated in fig 15 (appendix C).

The mortality of the steel and iron workers admitted in 1931—1948 with silicosis stage I and no tuberculosis is graphically presented as a contrast in fig 16 (appendix C).

Since the various categories of workers in the ceramic industry were relatively small, all stages of silicosis are collectively presented. The impact of silicosis in combination with suspected or verified tuberculosis is evident also from these figures. The observed mortality rate continued to exceed the expected rate in the 1949—1969 period (table 56, appendix C).

In fig 17—20 the chronological patterns of the observed mortality rates for various subgroups of the study population are presented in a diagrammatic form. The mortality rate in a theoretical sample of the general Swedish population, identical with the study population with regard to size, age, and time of admission, is set as

Fig 11. All occupational categories — Cumulative mortality in the part of the total study population admitted to the study in 1931—1948: All stages of silicosis, no tuberculosis (TBC). [Number (N) = 120]

Fig 12. All occupational categories — Cumulative mortality in the part of the study population admitted to the study in 1931—1948: All stages of silicosis, suspected or verified tuberculosis (TBC). [Number (N) = 343]

the index 100. The cumulated mortality experienced by the study population in each year is included in the rate index standardized mortality ratio (SMR), $\frac{\text{observed}}{\text{expected}} \times 100$. Appendix B should also be consulted.

Fig 17 shows the SMR pattern in the various subgroups of mining, quarrying, and tunneling workers admitted to the study in 1931—1948. The lines start with the mortality among those admitted before 1945. The corresponding patterns in the steel and iron industries are seen in fig 18 (appendix C). In both industries the cumulated SMR starts from high levels for silicosis in stages II or III with suspected

or verified tuberculosis. For silicosis in stage I without tuberculosis the starting level is just above 100.

Fig 19 and 20 (both in appendix C) present comparisons of mortality between silicotic workers in the steel and iron industries and in mining, quarrying, and tunneling. The data in fig 19 concern silicosis in stage I or stages II and III uncomplicated by tuberculosis. The cases in fig 20 were known or suspected to have had tuberculosis during the observation period. The SMR of the mining, quarrying, and tunneling workers was higher than that of the iron and steel workers, irrespective of tuberculosis.

In the second method of analysis the

CASES ADMITTED 1949-1969

NO TBC N=1860

Fig 13. All occupational categories — Cumulative mortality in the part of the study population admitted to the study in 1949—1969: All stages of silicosis, no tuberculosis (TBC). [Number (N) = 1,860]

CASES ADMITTED 1949-1969

TBC N=687

Fig 14. All occupational categories — Cumulative mortality in the part of the study population admitted to the study in 1949—1969: All stages of silicosis, suspected or verified tuberculosis (TBC). [Number (N) = 687]

MINING QUARRYING AND TUNNELING

CASES ADMITTED 1931-1948

Fig 17. Mining, quarrying, and tunneling — Cumulative overall mortality rate (SMR) and time after admission for the part of the study population admitted in 1931—1948: Smoothed curves (3-point moving average) for stages of silicosis (SIL) with or without suspected or verified tuberculosis (TBC).

differences between observed and expected mortality were studied with the application of a life-table technique. The cu-

mulative survival rates for the index population and a theoretical sample of the general population were followed from

Fig 21. Steel and iron industries — Survival experience according to time after admission for cases admitted in 1949—1969: Stage I of silicosis (SIL), no tuberculosis (TBC).

Fig 22. Mining, quarrying, and tunneling — Survival experience according to time after admission for cases admitted in 1931—1948: Stage II or III of silicosis (SIL), suspected or verified tuberculosis (TBC).

Table 57. Median reduction (a) of life expectancy. Estimated confidence intervals within parentheses — Mining, quarrying, and tunneling.

Cases reported	Stage of silicosis	Tuberculosis ^a		
		+	—	+ and —
1931—1948	I	12.3 (6.5—14.5)	2.0 (—0.5— 5.0)	
	II—III	17.0 (15.5—21.5)	14.0 (12.0—15.5)	
1949—1969	I		3.5 (1.2— 5.2) ^b	6.1 (5.0—7.0)
	II—III		6.5 (3.5— 8.5)	7.8 (7.0—9.5)

^a + = with suspected or verified tuberculosis, — = without suspected or verified tuberculosis.

^b 75 % survival intercept.

zero time, ie, time of notification, to the time when half of the subjects under study had died. The time difference between the 50 % intercepts of the observed and expected curves was used as a measure of the shortening of life. The term used for this measure is "shortening of median survival time" (appendix B).

Fig 21 illustrates these calculations for 697 steel and iron workers without tuberculosis and with stage I silicosis when admitted to the study in 1949—1969. By way of contrast fig 22 shows data concerning 93 mining, quarrying, and tunneling workers admitted in 1931—1948 with silicosis in stage II or III and with suspected or verified tuberculosis at some time in the observation period. The time difference is clearly greater in fig 21, ie, there was a greater reduction of the median survival time.

Some of the reductions in median survival time are illustrated in tables 57 and 58 (table 58, appendix C). When the numbers in the related subcategories were small, they were aggregated. The values denote time in years and are given with an approximate confidence interval ($\pm 2 \times \text{SE}$). When the analysis of reductions in median survival time was restricted to cases with bacteriologically verified tuberculosis, the following results were obtained. In cases notified in 1931—1948 from all occupations ($N = 175$) the reduction in median survival time was 18.0 a (16.5—18.5) and in cases notified in 1949—1969 ($N = 208$) 9.0 a (5.0—11.0).

Summary of results

The mortality among persons with silicosis was higher than the corresponding expected mortality when the general population was used as the reference standard. For cases drawn from mining, quarrying and tunneling, the iron and steel industries, and the ceramic industry, this excess mortality was observed in cases notified in 1931—1948, and in cases notified in 1949—1969. The mortality was particularly high when silicosis was notified in stage II or III.

Intercurrent suspected or verified pulmonary tuberculosis was associated with heightened mortality irrespective of the silicosis stage. For silicosis contracted in

the iron and steel industries, in stage I at diagnosis and without suspected or known tuberculosis, a lower mortality risk ratio (observed/expected deaths) was observed for cases admitted in 1949—1969 than for those admitted in 1931—1948. For silicosis contracted in mining, quarrying and tunneling, the ratio was unchanged between 1931—1948 and 1949—1969 for cases in stage I without suspected or verified tuberculosis.

The median survival time after the manifestation of silicosis varied according to occupational category, time of admission to the study, disease stage at admission to the study, and presence or absence of suspected or verified tuberculosis. The reduction in median survival time ranged from 1 to 18 a.

For the 1949—1969 cases in stage II—III of silicosis the reduction in median survival time was less than for cases admitted in 1931—1948; this finding suggests a reduction in total mortality risk.

Discussion

In studies of differential mortality, the influence of occupation has long been of major interest. But the use of occupation as a differential factor is beset with difficulties (25). Occupation, income, and education, though interrelated, may each have a different relationship to mortality. The total life situation, including use of tobacco, alcohol, etc, and nutrition, are among the other aspects that must be taken into account when mortality risks are considered for a population.

Analyses of relationships between mortality and occupation have nevertheless demonstrated the dependence of mortality risk on socioeconomic level. A progressive narrowing of differences has been reported, however, in this respect in past decades (25).

No studies have been published on the relationship between socioeconomic level and mortality risk in Sweden.

A person's occupation may reflect not only social origin, but also geographic situation and educational level, all of which may result in differences in income, in general attitudes, and in health parameters (15, 25). Furthermore, health may operate in the choice of occupation. Some

jobs thus attract healthy people who normally would have a lower mortality risk than the general population. This healthy worker effect (31) cannot be assumed to be evenly distributed in a study population that has been observed over a long period of time. In all probability it varies according to age, time, and geographic circumstances, as well as according to occupation.

Key questions are:

1. How far does mortality in an occupational group reflect the work environment and other factors intrinsic to that particular occupation?
2. How much of the mortality of an occupational group can be attributed to non-occupational factors?

Social class standardization has been discussed as a method of bringing occupational factors into the perspective of factors relating to social class (16).

Mortality in a total population used as a reference standard is a statistical concept which receives contributions from diversely employed sectors of the population, as well as from those not gainfully employed. The hazards to health and life in this general population are correspondingly varied. All these factors preclude any precise estimate of the expected mortality relevant to the study population.

The analyses in this section of the study were basically performed with the life-table technique. They showed that the index populations, consisting of persons with silicosis contracted in various occupations, had considerably higher death rates than the reference standard. Not unexpectedly, the differences were greater when silicosis was advanced at the time it was reported and when the disease was suspected or known to be complicated by pulmonary tuberculosis.

There was excess mortality among mining, quarrying, and tunneling workers admitted to the study in 1949—1969, as well as among those admitted in 1931—1948. In cases with silicosis notified in stage I and without tuberculosis, the mortality was apparently unchanged between these two periods. In such comparisons, however, the shorter observation period for the 1949—1969 admissions should be

kept in mind. It is also conceivable that the stage I cases from the two periods differed in some characteristics of the disease. If the standards of diagnosis and the efficiency of case finding had been as high in 1931—1948 as in 1949—1969, some of the stage II and stage III cases from the earlier period might have been detected in stage I.

Among iron and steel workers, the stage I silicosis cases reported in 1949—1969 and with no tuberculosis showed no significant deviation from the expected mortality. In contrast, cases from the same occupational groups and with the same characteristics, but admitted in 1931—1948, showed a statistically significant ($p < 0.05$) excess mortality. Once more, reservation should be made for the relatively short observation time for the 1949—1969 cases. Another point to remember is that pulmonary dust disease in the iron industry is often mixed-dust pneumoconiosis with a relatively favorable prognosis (30).

Calculations of median survival time from the detection of silicosis correspondingly demonstrated a shorter life expectancy for the silicosis cases than for the general population. With regard to the order of magnitude of this reduction, a point worthy of mentioning is that this study design was used by other authors to examine the impact of cigarette smoking on mortality. Hammond et al (21) thus used it to compare the mortality of 100,000 male cigarette smokers and 100,000 male nonsmokers. All the men were 40 a old when admitted to the study. According to the published data, the median life expectancy of the smokers was 7.4 a less than that of the nonsmokers. This shortening of median life expectancy was well within the range of the reductions found in the present study. The study population and the reference standard in the present study of silicosis includes both smokers and nonsmokers.

A significant part of the mortality among the cases of the present study was undoubtedly attributable to silicosis and to concomitant tuberculosis. The contributions made by purely occupational factors and those by nonoccupational factors to the discrepancies between the observed and expected mortality cannot be wholly disentangled however. The characteristics

of the dust-exposed populations from which the silicosis cases were drawn are unknown. The healthy worker effect has already been mentioned. It is not feasible to make any estimates concerning this possible effect among the risk populations or concerning factors relative to life-style and socioeconomic level, all of which may have directly or indirectly influenced the mortality rates.

In a study of workers in Finnish iron foundries, Koskela et al (27) found the overall SMR to be less than 100. This finding suggests that the healthy worker effect in that particular study population in one of Sweden's neighboring countries had a greater impact on mortality than the factors which tend to increase SMR.

If the findings by Koskela et al are accepted as valid also for the risk populations of the present study, the data indicate that in this study the mortality attributable — directly or indirectly — to silicosis was considerable. Other indications are the much higher mortality rates among the workers with silicosis admitted in stage II or III than among those with stage I silicosis. The difference can be regarded as analogous to a dose-response relationship. The relationship between SMR and length of follow-up, with a high SMR closely following admission to the study, also suggests silicosis as an important cause of the differences between observed and expected mortality.

The data from the analyses suggest that the situation concerning excess mortality among cases of silicosis from the iron and steel industries improved in the cases admitted in 1949—1969 as compared with those admitted earlier. This result is consistent with the concept that improvements in hygienic standards and in occupational environments reduce death rates for exposed populations. The evidence cannot be accepted as conclusive, however, since other factors may have been of equal importance. No such change could be demonstrated in the comparisons of stage I silicosis detected in mining, quarrying, and tunneling workers in two time periods.

A noteworthy observation seemed to be the difference in the reduction of median survival time in cases of stage II or III silicosis admitted in 1931—1948 as com-

pared with those admitted in 1949—1969. Tables 57 and 58 show that, in the combined categories of silicosis stage II or III with or without suspected or verified tuberculosis and admitted in 1949—1969, the reduction was less than in the nontuberculous stage II or III cases from 1931—1948. This result implies that life expectancy in silicosis stage II or III had improved with the passage of time.

When seeking explanations for this change, one must first consider the diagnostic standards and the criteria for classifying silicosis. Changes in the routine of classification and in diagnostic standards may result in radiographic lesions that were earlier classified as stage I being later classified as stage II or III. This phenomenon could well alter the composition of the study population, diluting it with milder forms of the disease. As consistent care was taken to apply the same classification criteria throughout the study, it is reasonable to assume such changes to be of minor importance. It is nevertheless quite conceivable that the practices of classification were slightly modified and the diagnostic standards improved during a study period extending over 40 a. Other explanations which must be considered are:

1. A change in the natural disease course of stage II or III silicosis with the passage of time that resulted in a more favorable clinical course for cases detected between 1949 and 1969. Such a change could arise from selection phenomena or could constitute a true shift towards less aggressive forms of silicotic disease, a shift arising from improvements in the work environment.
2. Improved standards of medical surveillance and the care of silicosis and its complications, the result being a lengthening of life expectancy and thus of median survival time.
3. A time-related lessening of the impact of nonoccupational socioeconomic factors on mortality. This result applies to socioeconomic factors which can produce excess mortality relative to a general population standard and which are not counterbalanced by the healthy worker effect.

An important factor to consider in intergroup comparisons of median survival time is the age structure of the groups compared. Differences in age structure will affect expected survival times. A group composed of older persons naturally has a shorter life expectancy than a group of younger persons. This is a source of difficulty in comparing reductions in median survival time.

At admission to the study the mean age of the mining, quarrying, and tunneling workers was approximately 5 a higher in the period 1949—1969 than in 1931—1948. The corresponding difference for iron and steel workers was 4.5 a. The value for the median expected survival time in the cases from the mining, quarrying, and tunneling occupations in the period 1931—1948 was approximately 20.1 a. In the period 1949—1969 it was 18.4 a. The difference of 1—2 a clearly could not account for the total difference in the reduction of median survival time (tables 57 & 58).

The age factor is evidently not the only factor to be considered when an attempt is made to explain the improving life expectancy of silicosis cases in stage II or III. Solely from the data in the Pneumococcosis Register, it was not possible to decide which other factors, singly or in combination, were the most important.

Concluding remarks

The overall mortality rate of silicotic workers is higher than the expected mortality if the general population is used as the reference standard. This conclusion applies both to cases notified in 1931—1948 and to those notified in 1949—1969.

The presence of verified or radiographically suspected tuberculosis is a factor which increases mortality risk.

A radiographically advanced stage of silicosis — stage II or III — is a factor which increases mortality risk.

Comparisons between occupational categories — mining, quarrying, and tunneling versus iron and steel industries — suggest differences with respect to mortality risk.

The data from the iron and steel industries suggest an improvement in mortality risk for cases notified in stage I and with no known or suspected tuberculosis when cases from 1949—1969 are compared with those from 1931—1948.

For both occupational categories compared, such an improvement over time was also found for cases notified in stage II or III.

As this study is not truly population-based, there are many possible explanations for such an improvement. The credit cannot be unreservedly given to improved work conditions.

Section VI

Lung cancer

The question of a possible association between silicosis and lung cancer has received considerable attention in the literature. [For extensive reviews of this subject the reader can consult Reichel (39) and Könn et al (26)]. Most of the contributors to the scientific discussion of this question have not accepted the concept of a causal association between silicosis and lung cancer. The present study included an investigation on such association.

Material

The following occupational categories were included in the investigation: (i) mining, quarrying, and tunnelling (codes 01—31), (ii) the steel industry (codes 32—40, 76), (iii) the iron industry (codes 41—46), (iv) the ceramic industry (codes 47—55, 81, 82, 85, 87), and (v) other occupations.

In the tables, this study population was divided into two categories, 1931—1948 and

1949—1969, according to year of notification.

Methods

The observed numbers of deaths from lung cancer in these categories during the period 1951 to 1969 were compared with the expected mortality from the disease during the same period. Mortality from cancer in other organs was compared in the same way. The analysis was performed as a proportional mortality study.

The ratio lung cancer deaths/total deaths for the period 1951—1969 in each age group of the general population could be read from the official Swedish mortality statistics. The expectancy of lung cancer mortality could be obtained from an application of these ratios to the observed deaths in the study population. This situation applied also to cancer in other organs. (An explanation is presented in appendix B.)

In all of the studied occupational categories, the observed number of deaths from all causes was higher than the expected number. In the calculation of the latter, the national mortality statistics were used as the reference standard. The excess numbers of deaths were assumed, for the purpose of the analysis, to be distributed among the various causes of death in the same proportions as the age-specific deaths

in the general population (appendix B).

A relevant point is that the observation period concerning cancer mortality covered only 1951 through 1969. The reason was that available information on cause-specific mortality in the general population before 1951 did not provide an adequate basis for the calculation of the expected number of lung cancer cases or other cancers. Therefore, workers who entered the study in 1931—1948 and died before 1951 were not included in the tabulated calculations. For the workers who entered later, the corresponding period covered, at most, 2 a (1949 & 1950). For this reason the conclusions and the discussion apply primarily to cases admitted in 1949—1969.

Results

The data on observed and expected mortality from lung cancer according to occupational category and time period of notification are presented in table 59. Table 60 presents corresponding data on cancers of other organs.

The ratio > 1 for observed/expected mortality indicates an increased risk of death from cancer of the lung (table 59). The risk was particularly marked for mining, quarrying, and tunneling workers (ratio = 3.8) with silicosis notified between 1949 and 1969. The excess was prominent also for steel and iron industry workers

Table 59. Observed and expected mortality from lung cancer in silicosis. Rates based on expected number of cases ≤ 1.0 within parentheses — All occupational categories.

Occupational category	Silicosis manifest	Deaths from lung cancer		
		Observed	Expected	Ratio observed/expected
Mining, quarrying, tunneling	1931—1948	10	1.7	5.9 **
	1949—1969	20	5.3	3.8 **
Steel and iron industries	1931—1948	2	2.5	0.8
	1949—1969	10	4.6	2.2 *
Ceramic industry	1931—1948	1	0.8	1.3
	1949—1969	2	0.7	2.9
Other industries	1931—1948	3	0.9	3.3
	1949—1969	1	1.0	1.0

* $p < 0.05$, ** $p < 0.01$.

Table 60. Observed and expected mortality from cancer other than lung cancer — All occupational categories.

Occupational category	Silicosis manifest	Deaths from other cancer		
		Observed	Expected	Ratio observed/expected
Mining, quarrying, tunneling	1931—1948	16	18.0	0.9
	1949—1969	36	65.1	0.6 **
Steel and iron industries	1931—1948	16	25.9	0.6
	1949—1969	32	51.5	0.6 **
Ceramic industry	1931—1948	6	7.3	0.8
	1949—1969	8	11.7	0.7
Other industries	1931—1948	4	6.3	0.6
	1949—1969	8	15.1	0.5

** $p < 0.01$.

(ratio = 2.2). The upward deflection in these groups was not explained by a sampling variation ($p < 0.01$ and $p < 0.05$, respectively). Although the observed/expected ratio showed an upward tendency also in the two remaining occupational categories, the numbers of cases were too small for the testing of statistical significance.

As regards deaths from cancer in organs other than the lung, the trend seemed to be reversed, with a downward deflection of the ratio observed/expected deaths (table 60). Among the workers with silicosis notified in 1949—1969 the tendency was particularly pronounced for mining and metal foundry workers, the statistical significance being on the level of 1 %.

Summary of results

An association between silicosis and lung cancer deaths was found for mining, quarrying, and tunneling workers and steel and iron industry workers. The association appeared as increased mortality from lung cancer when the general population was used as the reference standard. In the study population admitted in 1949—1969 the excess mortality risk from lung cancer corresponded to a risk ratio of 3.8 for the mining, quarrying, and tunneling occupations and 2.2 for the iron and steel industries.

Discussion

The indications from the data that mortality from cancer of one organ was greater than expected in workers with silicosis, while mortality from cancer of other organs was less than expected (tables 59 & 60), raise some puzzling considerations.

The study population, it should be remembered, was exposed to mortality risk from various causes. These can be regarded as competing risks; death from one cause obviously reduces mortality from another.

In the absence of other evidence that the workers with silicosis were more cancer-resistant than the rest of the population, alternative explanations must be sought. A reasonable assumption is that silicosis and tuberculosis, as common causes of death among these workers, reduced the likelihood of death from cancer. If so, the impact of lung cancer should have been reduced in the same way. If the true ratio of observed to expected deaths from other cancer is assumed to be 1.0 and silicosis and tuberculosis are judged to have caused the observed reduction of that ratio, it is reasonable to infer that the observed excess risk of death from lung cancer is an underestimate. Theoretically, removal of silicosis and tuberculosis as causes of death should bring the mortality risk from cancer of other organs to 1.0. It should also raise the figures for

lung cancer since these two important causes of death also compete with lung cancer. For the mining, quarrying, and tunneling workers with silicosis notified in 1949—1969, the resultant ratio for lung cancer mortality, from the data in tables 59 and 60, would be $R = 3.8/0.6 = 6.3$. This risk ratio for death from lung cancer would apply if silicosis and tuberculosis did not per se cause an excess mortality risk.

The data indicate an association between silicosis and lung cancer in two of the studied groups. In the other groups the numbers were too small to permit statistical analysis.

The data supporting a relationship between silicosis and lung cancer do not imply that the relationship is causal. The cases of silicosis were drawn from occupational groups in which lung cancer risks are unknown. The risk factor(s) need not lie in the reaction of lung tissue to silica dust per se. The risk may instead lie in occupational or nonoccupational factors acting independently from the silicosis risk. Among mining workers, for instance, exposure to ionizing radiation from radon and radon daughters should be taken into account. Axelsson & Sundell (8) have demonstrated an increased mortality from

lung cancer in populations of miners in Sweden. For metal foundry workers no equivalent factor is known. A relevant observation was reported in Finland by Koskela et al (27), who found increased lung cancer mortality for iron foundry workers, particularly molders.

Among nonoccupational factors, smoking habits are important. The relationship between lung cancer mortality and varying smoking habits in occupational groups has been discussed by Fox (15). The importance of the smoking factor for the findings of the present study is not known.

The hypothesis that silicosis per se increases the risk of lung cancer should not be rejected however. The chronic irritation caused by quartz dust retained in the lung tissue and its consequences may not lack importance in this particular respect. Additional studies seem to be called for.

Concluding remarks

Cases of silicosis, notified in 1949—1969 from mining, quarrying, and tunneling and from the iron and steel industries, have a higher risk of dying from lung cancer than the general population.

Section VII

Competing mortality risks

In the preceding sections of this monograph it was shown that the progression of silicosis and the manifestation of tuberculosis may be late events. The competing impact of such events on deaths from various causes should therefore be considered. Without such deaths, it may be argued, the rates of silicosis progression and the cumulative incidence of tuberculosis would have been higher. Intercurrent death from various causes constitutes a risk competing with other possible outcomes in cases of silicosis. The purpose

of this section of the study was to highlight this competition of risks in considerations of cause-specific mortality rates.

The study was performed in the following three steps: (i) an investigation of causes of death of cases notified in 1931—1969, (ii) an investigation of the impact of silicosis and tuberculosis on the mortality of cases notified in 1949—1969, and (iii) an investigation of the consequences of eliminating silicosis and tuberculosis as causes of death in cases notified in 1949—1969.

Material

All the information was extracted from the Pneumoconiosis Register. For all three steps the study population consisted of cases from mining, quarrying, and tunneling (codes 1—31) and the iron and steel industries (codes 32—46, 76).

In the first step the main causes of death were classified as silicosis, tuberculosis, lung cancer, and other causes. In the second and third steps of the investigation the class "other cancer" was separated from the class "other causes" to constitute a fifth cause-of-death class.

An important point in this connection is that, at the Pneumoconiosis Registry, decisions on causes of death have a mixed medicolegal basis. Since the 1931 legislation, death of silicotic persons from silicosis and/or tuberculosis has constituted grounds for financial compensation to surviving dependents.

Because of the phrasing of the statutes, considerable bias is possible in the judgments concerning the causes of death that are entered in the Register. Death is attributed to silicosis or tuberculosis whenever it is deemed that their contribution to the fatal outcome has been sufficient to entitle compensation.

Each death is routinely entered in the Register under a single main cause. The decision on cause is made by the consultant physician after he has considered all available information in each case. Tuberculosis as cause of death is ascertained with bacteriologic examinations and/or a postmortem examination. In cases of silicosis with diagnosed cancer, deaths are recorded with cancer as the main cause. The cancer diagnosis is ascertained with pathological examinations. The acceptance of cancer diagnoses is conditional on such an examination. These routines have remained unchanged from the start of the Register.

Methods

In the second and third steps of the analysis the following entities were calculated for silicosis, tuberculosis, lung cancer, cancer of other organs, and other causes of death:

- q_x — death risk during the interval $(x, x + 1)$ — all causes
 $q_{x,r}$ — net probability of dying in the interval $(x, x + 1)$ when the specific cause of death (r) is eliminated, ie, of death from any cause other than r .

Table 61. Causes of death in relation to stage of silicosis at time of report — Mining, quarrying, and tunneling. (N = number of cases)

Silicosis reported	Silicosis stage	Cause of death (%)				Total (N)
		Silicosis	Tuberculosis	Lung cancer	Other causes	
1931—1948	I	10.7	18.5	17.7	53	130
	II—III	55.0	35.4	8.0	14	200
1949—1969	I	14.4	9.2	21.0	55	195
	II—III	43.9	16.7	8.9	30	180

Table 62. Causes of death in relation to stage of silicosis at time of report — Steel and iron industries. (N = number of cases)

Silicosis reported	Silicosis stage	Cause of death (%)				Total (N)
		Silicosis	Tuberculosis	Lung cancer	Other causes	
1931—1948	I	13.6	12.5	6.8	67	176
	II—III	55.8	17.0	6.8	22	147
1949—1969	I	6.5	4.5	17.0	72	200
	II—III	40.2	14.1	8.7	35	92

These entities and their implications are explained in appendix B.

The relative importance of a specific cause of death can be visualized as a comparison between the net probability of dying when this cause has been eliminated and the death risk (q_x) from all causes. The equation

$$\frac{100 (q_x - q_{x,r})}{q_x} \%$$

can be used to express the theoretical percentage reduction in annual mortality risk by elimination of a particular cause of death.

Results

The results of the first step are presented in tables 61 and 62. The tables show the distribution of deaths according to cause, time period, and stage of silicosis at notification. The tables include all deaths in the study population which have occurred before the end of 1970. As can be seen, silicosis and tuberculosis were more common causes of death when silicosis had reached stage II or III at the time of notification than in the cases notified in stage I.

The results of the second analytical step

MINING QUARRYING AND TUNNELING
CASES ADMITTED 1949-69 SIL=2

Fig 23. Mining, quarrying, and tunneling — Reduction (%) of annual net death risk when various causes of death are eliminated separately: silicosis (SIL) reported in stage I in 1949—1969. (TBC = tuberculosis)

MINING QUARRYING AND TUNNELING
CASES ADMITTED 1949-69 SIL=2

Fig 24. Mining, quarrying, and tunneling — Reduction (%) of annual net death risk when various causes of death are eliminated separately: Silicosis (SIL) reported in stage II or III in 1949—1969. (TBC = tuberculosis)

are shown in fig 23—26. The diagrams in the figures represent the decrements in the annual net risk of death which were obtained from the theoretical elimination of the various causes of death. Each cause of death is represented by one curve. The reductions from the total annual mortality (100 %) are given on a percentage scale. Zero on the time axis is the year when silicosis was notified. Fig 23 and 24 concern mining, quarrying, and tunneling workers. Fig 25 and 26 concern workers from the iron and steel industries. For a clearer presentation, the curves were smoothed with the moving 3-point average technique.

In both occupational categories silicosis

and tuberculosis were the dominant causes of death over many years of observation when silicosis had been notified in advanced (fig 24 & 26). In cases with stage I silicosis these causes were less important, and consequently their elimination as a net cause of death produced less spectacular reductions (fig 23 & 25).

The differences between stages II—III and stage I in these analyses were striking. In the former groups the curves for silicosis and tuberculosis together exceeded the curves for other causes.

The third step of the analysis was an attempt to assess the validity of the medicolegal statements on cause of death in the Register. The silicosis cases notified

STEEL AND IRON INDUSTRY

CASES ADMITTED 1949-69 SIL=2

Fig 25. Steel and iron industries — Reduction (%) of annual net death risk when various causes of death are eliminated separately: Silicosis (SIL) reported in stage I in 1949—1969. (TBC = tuberculosis)

STEEL AND IRON INDUSTRY

CASES ADMITTED 1949-69 SIL=2

Fig 26. Steel and iron industries — Reduction (%) of annual net death risk when various causes of death are eliminated separately: Silicosis (SIL) reported in stage II or III in 1949—1969. (TBC = tuberculosis)

among mining, quarrying, and tunneling workers and among steel and iron industry workers in the period 1949—1969 were followed starting with the year of notification, ie, the time of admission to the study. The observed survival rate was noted as the percentage remaining under observation 10 a after admission. The percentage of the expected survival rate was calculated for the sample effectively under observation, and the age-specific mortality rates for the general population were applied. The hypothetical 10-a survival was also calculated if silicosis and tuberculosis had been eliminated as causes of death. This elimination procedure was carried out with the same technique as in the second step of the analysis. The results are shown in table 63. The right marginal column of this table shows the percentage of the study population that would have remained under observation after 10 a if silicosis and tuberculosis had been eliminated as causes of death.

Table 63 shows that the elimination of silicosis and tuberculosis as causes of death among persons with silicosis detected in stage I would have resulted in diminished mortality. The position of this hypothetical figure on the 10-a intercept was roughly midway between the expected and the observed value. Among the cases of silicosis notified in stage II or III, the observed mortality was predictably higher than in the stage I cases. In these cases of more advanced silicosis, the percentage of survivors after the elimination of silicosis and tuberculosis as causes of death was practically identical with the expected survival rate.

Summary of results

The analyses in this section indicate that among workers with silicosis notified in 1949—1969, silicosis and tuberculosis were particularly important as causes of death when the silicosis had reached stage II or III at the time of the notification. The analyses comprised mining, quarrying, and tunneling workers and steel and iron industry workers.

Silicosis and tuberculosis were important causes of death in cases admitted to the study in the period 1949—1969. Among cases admitted in stage II or III, the proportion of deaths attributable to these two causes was 60 % for cases from mining, quarrying, and tunneling and 54 % for cases from the iron and steel industries. For cases admitted in stage I, the corresponding percentages were 24 and 11.

The proportion of deaths caused by lung cancer were less for cases admitted in stage II or III of silicosis than for cases admitted in stage I.

A theoretical elimination of silicosis and tuberculosis as causes of death among workers with silicosis notified in stage I markedly reduced the difference between observed and expected survival. Even after such correction, however, the expected survival remained higher than the observed. This result applied to both of the analyzed occupational categories. For the cases of silicosis notified in stage II or III, the same theoretical elimination procedure resulted in almost equal expected and observed survival rates.

Table 63. Survival expressed as percentage of study population still alive 10 a after admission (1949—1969) — Mining, quarrying, and tunneling.

Occupational category	Number	Silicosis stage	Observed survival (%)	Expected survival (%)	Corrected survival ^a (%)
Mining, quarrying, and tunneling	655	I	71	85	77
	398	II—III	51	79	77
Steel and iron industries	845	I	79	85	81
	214	II—III	60	77	79

^a Refers to values obtained after the elimination of silicosis and tuberculosis as causes of death.

Discussion

Data from cause-specific mortality statistics must be cautiously interpreted. The first question in dealing with such statistics is whether observed differences between populations are true or are products of differences in diagnostic practice or some other biasing factor. The practical difficulties in classifying the main cause of death should not be overlooked. In this study the classification in all cases was made by the consultant physician at the National Social Insurance Board (T Bruce) and was based on all available records. The judgements on causes of death could conceivably have been subject to bias in that silicosis and tuberculosis may have been used more often than they would have been in situations not involving issues of financial compensation. Even without such issue, however, totally unbiased judgements of causes of death are probably hard to achieve. In view of the consultant physician's knowledge of and access to the records in each case, silicosis or tuberculosis was likely to have been accorded some extra weight over other possible causes of death. For example, knowledge that a deceased worker had silicosis in stage II or III on the first positive radiographs could conceivably increase the likelihood that death would be attributed to this disease.

Reliable and reproducible criteria for using diagnoses such as silicosis in classifying causes of death do not exist. This circumstance applies to many diseases. The responsible physician's personal preferences play an important part, and these in turn depend upon training, experience, and attitudes.

The analyses with the theoretical elimination of silicosis and tuberculosis as causes of death are useful in discussions of the potential importance of physician bias. In the cases of silicosis reported as stage I, the exclusion of silicosis and tuberculosis as causes of death failed to eliminate all the difference between observed and expected survival. Therefore there was excess mortality in the study population as compared with the general population. It could not be accounted for by silicosis and tuberculosis as causes of death.

If one assumes that the expected survival rate — or, conversely, mortality rate — in the general population is a valid basis for comparisons with the population in this study, one may confidently argue that the medicolegal use of silicosis and tuberculosis as causes of death was reasonably accurate and did not lead to biased overestimation of their impact on mortality. On the contrary, any bias was more likely to be an underestimation. In theory, if a healthier population is used as the basis for comparison, this underestimation becomes even greater.

In a consideration of the residual difference between observed and expected survival, the association between silicosis and lung cancer should be kept in mind. If lung cancer is also theoretically eliminated as a cause of death, the observed survival rate would be still nearer the expected rate.

An interesting point is that in cases of stage II or III silicosis, the same procedure of theoretically eliminating silicosis and tuberculosis as causes of death resulted in practically no difference between the observed and expected survival rates. This result suggests that, in such cases of more advanced disease, the classification of silicosis or tuberculosis as the cause of death was still more accurate. Another relevant observation was the lesser impact of lung cancer in stage II or III than in stage I silicosis. It is conceivable that this lower rate of lung cancer deaths resulted from competition with silicosis and tuberculosis as causes of death.

A general problem in cause-of-death statistics concerns the varying validity of clinical diagnosis. The validity of diagnoses on death certificates in Sweden was investigated by de Faire et al (14) and Britton (10). These writers found that the recorded data were reasonably valid for most forms of cancer, cerebrovascular disease, asthma, accidents, and suicide but not for diabetes mellitus, alcoholism, mental disorders, and heart disease. They also observed that contributory causes of death were clinically underestimated.

The difficulties inherent in selecting the correct main cause of death when several chronic diseases coexist are well known. In studies such as the present one, the best

way to minimize inaccuracies in this respect is to collect all the available documents in each case and have them reviewed consistently by persons with thorough knowledge of the problem area.

A full discussion of all the issues involved in the reliability of death certification is not within the scope of the present study. The analyses were not designed to give quantitative estimates of the effect of all the factors which may introduce bias. For a fuller discussion of these problems the reader is referred to Adelstein (1) and Griffith (20). Although the decisions on causes of death in the population of the present study were based on mixed legal and medical considerations, they can be assumed to have a high degree of consistency, since all were made by the same specialist.

A final point to mention in this discussion is that, not only does silicosis still occur, it can still be fatal. This is a sad

reflection on the inadequacy of preventive measures earlier in this century.

Concluding remarks

In a study population consisting of cases from two occupational categories with silicosis notified in 1931—1969, silicosis and tuberculosis constituted important causes of death. This finding applies to cases notified in 1931—1948 and also to those notified in 1949—1969.

Silicosis and tuberculosis compete with other causes of death, particularly if the silicosis has reached stage II or III at the time of notification.

The analyzed data are consistent with the view that the statements of cause of death from silicosis or tuberculosis in the Register are not unduly biased, although they are based on medicolegal considerations.

Section VIII

Dust exposure after diagnosis and risk of radiographic progression — A case-referent study

A question of great practical importance is whether the natural course of silicosis is influenced by the termination of exposure to silica dust.

This issue was earlier examined in a study by Ahlmark et al (4). According to these authors, removing persons with silicosis or mixed dust fibrosis from exposure to the causal dust failed to improve the outlook concerning progression of the lesions. The problem has also been discussed by Reichel (39), but without reference to published studies. [For additional references the reader can consult Ahlmark et al (4).]

In order to study the relationship between postdiagnostic dust exposure and the risk of radiographic progression, a case-referent study was designed. The data were obtained from the Pneumoconiosis Register.

Material

The main analysis included workers from an iron-ore mining company who, at some time between 1931 and 1950, had been found to have silicosis which had not progressed beyond stage I at the time of diagnosis.

Workers with supervening pulmonary tuberculosis were excluded because of the earlier mentioned difficulties with the radiological assessment of silicosis progression in the presence of tuberculosis. So that the study population would be reasonably homogeneous in regard to dust exposure, only drillers, haulage workers, and loaders (code 09, appendix A) were selected for the follow-up study.

An attempt was also made to study fitters from the steel industry with the same study design.

The routinely collected data on dust exposure and medical examinations were obtained from the Pneumoconiosis Register. They included the duration of dust exposure, the date of the first radiograph showing silicosis, the time when a radiographic progression was registered, the time when exposure ceased, and the time when the case was notified.

Termination of exposure is routinely entered in the Register as the time when a worker retires on pension or is transferred by his employer or at his own request to work which is not considered to involve risk of pulmonary dust disease.

Methods

All cases with radiographically demonstrated progression of silicosis to stage II or III at any time during the follow-up were designated as progression cases. Those without such known progression were used as referents. Each progression case was matched with a referent. The matching was made according to age at the time when silicosis became manifest

(± 4 a) and years of dust exposure preceding that date (± 4 a). When there were several candidates for a matched referent according to these criteria, nearness in time of birth was the decisive factor. Some degree of matching for time of birth was thereby obtained. The total of matched pairs in the analysis was 57.

The progression cases and the referents were classified as + if exposure to dust continued for more than 2 a after silicosis was manifest. If the exposure ceased within this time, the classification was —. The 2-a limit was somewhat arbitrarily chosen as a point of time early in the course of disease when cessation of hazardous exposure could conceivably be rewarding.

The analysis was performed as a matched-pair analysis. The pairs were set up in a 2×2 table according to concordance or discordance of pairs with regard to exposure after the time when silicosis was first observed.

Results

The first analysis is shown in table 64. In it exposure (+) is defined as continued exposure to dust for more than 2 a after the first radiographic signs of silicosis.

Table 64 shows that 34 (28 + 6) pairs were discordant with respect to dust exposure after the manifestation of silicosis. On the null hypothesis that the distribution of discordant pairs is binomial with the parameter $\frac{1}{2}$, test statistics (29) give:

$$Z = (r - n/2 - \frac{1}{2}) / \frac{1}{2} \sqrt{n},$$

where Z is regarded as a standardized normal deviate, n is the total number of discordant pairs, r is the number of dis-

Table 64. Distribution of matched pairs according to continuation or cessation of dust exposure after manifestation of silicosis (1931–1950) — Mining.

Progression cases ^a	Referents ^a		Row totals
	Exposure (+)	Exposure (—)	
Exposure (+)	14	28	42
Exposure (—)	6	9	15
Column totals	20	37	57

^a + = exposure to dust continued after manifestation of silicosis, — = exposure to dust ceased after manifestation of silicosis.

Table 65. Mean age at manifestation of silicosis and mean duration of dust exposure before and after diagnosis in matched pairs — Arithmetic means with standard deviation. (Number of pairs = 57)

	Age (a) at diagnosis		Dust exposure (a)			
			Before diagnosis		After diagnosis	
	Mean	SD	Mean	SD	Mean	SD
Progression cases	42.0	8.1	15.1	7.4	8.7	8.1
Referents	42.9	7.6	15.0	6.5	3.2	6.2

cordant pairs in which the case is + and the referent is — with regard to exposure.

$$Z = \frac{28 - \frac{1}{2} \cdot 34 - \frac{1}{2}}{\frac{1}{2} \sqrt{34}} = 3.6$$

$$p < 0.001$$

An estimate of the risk ratio is obtained as $(28/6) = 4.7$ with 95 % confidence limits of 2.1—10.5 (32).

If, instead of 2 a, the time requirement for exposure after manifest disease is set as 6 a or more to be classified as +, the number of discordant pairs ($r + s$) is $26 + 3$, the point estimate of the risk ratio being $(26/3) = 8.7$.

These analyses of matched pairs showed a statistically highly significant association between risk of silicosis progression and continuation of dust exposure after the manifestation of silicosis. The age and the duration of dust exposure of the cases and referents are shown in table 65 as arithmetic means. The progression cases and the referents were similar with regard to age and prediagnostic exposure time. They differed in exposure time *after* diagnosis, i.e., they differed with respect to the factor under study.

For the majority of the cases and referents the decision to classify radiographically visualized lesions as silicosis was retrospectively made. The mean delay between time of manifest disease and notification was 12.9 a among the progression cases and 10.1 a among the referents.

For the 57 cases with progression the mean time lag between manifestation of silicosis and radiographic progression was 14.8 a (SD = 8.1). For the 57 referents the mean time between manifestation of silicosis and the latest radiographic examination was 17.8 a (SD = 6.3).

In the following years (1951—1975) the Pneumoconiosis Register showed only three cases of progressive silicosis from the same mining company. These were too few to permit an analogous analysis.

Using the same technique, an attempt was made to analyze the association between continued dust exposure and progressive silicosis among steel workers. The group selected for the analysis consisted of fettlers (code 38, appendix A).

From 1931 to 1950 there were 19 fettlers whose exposure to the silica dust contin-

ued for more than 2 a after silicosis became manifest and for whom matched referents (as in the mining group) could be found. They are shown in table 66, along with 19 matched pairs from the later period of the study (1951—1975). For the numerous other cases of progressive silicosis among fettlers, matched referents were not available. Because of the small numbers of matched pairs which could be set up, the analysis was not continued.

Summary of results

There was a statistical association between a continuation of dust exposure and a tendency towards radiographic disease progression for the study population, drawn from one particular mining company.

Discussion

The analysis indicated an association between a continuation of dust exposure after the manifestation of silicosis in 1931—1950 and progression of the disease in drillers, haulers, and loaders in iron-ore mining. The analysis could not be repeated with more recent cases due to the infrequency of progressive silicosis. Nor could such an analysis be satisfactorily conducted for steel fettlers because referents satisfying the matching criteria could not be found.

The use and analysis of matched samples in epidemiologic studies was discussed by Worcester (45). Miettinen (32) reviewed

Table 66. Distribution of matched pairs according to continuation or cessation of dust exposure after manifestation of silicosis — Steel industry.

Silicosis manifest	Progression cases ^a	Referents ^a	
		Exposure (+)	Exposure (—)
1931—1950	Exposure (+)	1	4
	Exposure (—)	5	9
1951—1975	Exposure (+)	—	5
	Exposure (—)	4	10

^a + = exposure to dust continued after manifestation of silicosis, — = exposure to dust ceased after manifestation of silicosis.

the methodology. Issues relevant to this methodology in drug-induced illness were discussed by Jick & Vessey (24). To my knowledge no attempt has previously been made to apply case-referent methodology in investigations of the studied hypothesis.

Basically, the aim in the present study was to note progression of silicosis in relation to continuation of dust exposure after the lesions became radiographically detectable. For this purpose cases with progressive silicosis were matched with referents who showed no progression, and continuation or cessation of dust exposure was recorded for both groups.

In order to ensure homogeneity, the analysis was confined to particular types of dust exposure — the mining of iron ore and steel making. With reasonable confidence, therefore, it could be stated that the cases with progression and their referents had been subjected to equivalent dust exposure and had equal access to diagnostic facilities. The method of collecting information on the dust exposure and on the pulmonary disease was also presumably equivalent for both categories.

Matching was done in order to ensure optimal comparability between the progression cases and their referents. Inter-group differences in age or duration of dust exposure prior to the detection of silicosis could have produced biased estimates of the studied risk. Age and duration of exposure were therefore among the matching factors. Decisions concerning termination of dust exposure following the detection of silicosis in 1931–1950 were based primarily on clinical considerations. The documentation concerning these decisions does not permit a closer examination of the presence or absence of confounding factors.

The association that was found between the postdiagnostic continuation of exposure to dust and a tendency towards a progression of silicosis is statistically strong. Consequently, it can scarcely be attributed to chance.

The main questions evoked by the available data concern the possibilities that (i) the cases showing progression differed from the referents before progression occurred, and that this difference was relevant to the future course of the disease, and (ii) there was a failure to detect tran-

sition from referent to progression status.

As regards the first possibility, an inter-group difference could conceivably have existed for exposure to dust, eg, more intense exposure among the cases with progression than among the referents. This difference could then have been responsible for the early progression of the disease without being related to termination of dust exposure. The course of the disease would thus have been set already at the time of diagnosis. A scrutiny of the registered information revealed no difference of this kind between the groups.

All of the 57 miners with a progression of silicosis and all of their matched referents had, at some time, been drillers in iron-ore mines; 43 of the former and 45 of the latter were actually employed as drillers when silicosis was detected. There was nothing in the available data on dust exposure, ie, the individual occupational histories, to suggest that the progression cases differed from the referents with regard to the intensity of dust exposure. One must nevertheless recognize that the validity of the analysis rests on the assumption that the duration of exposure to dust in the work processes concerned is a relevant matching criterion.

The progression of silicosis is common and may occur after many years. In order to eliminate the time factor as a source of error, the length and intervals of follow-up must be similar for the cases with progression and for the referents. Otherwise referents may have unobserved progression in the late follow-up period. A relevant point is that the latest radiographic examinations of the referents were, on the average, made after progression had been radiographically visualized in the progression cases.

The data in the Pneumoconiosis Register thus permitted the conclusion that the silicosis cases with progression and the reference group did not appreciably differ with regard to characteristics of dust exposure and length of postdiagnostic observation.

In interpreting the observed association between continued dust exposure and the progression of silicosis manifested between 1931 and 1950 among the mine workers, one must keep in mind that the data had been obtained from a single company. An

attempt to perform a corresponding analysis for steel fitters failed because the sample was too small and difficulties arose in extracting matched referents. Although one cannot generalize from findings restricted to one mining company, such findings may be used as a basis for discussing the practical implications of a relationship between silicosis progression and the continuation of exposure to dust. One such implication is that the course of silicosis may be favorably influenced by removal of the worker from the causal dust, even at a relatively late stage, and thus additional pulmonary dust load is avoided. In a somewhat analogous fashion, Peto (37) discussed preventive value in regard to lung cancer risk and the termination of many years of cigarette smoking.

The analysis of routinely collected data on the radiographic course of silicosis and exposure to dust in iron-ore miners suggests that a preventive effect may be achieved if dust exposure is terminated. Since no other explanation is available, it is tempting to interpret the association as causal. From a scientific point of view, however, some caution is advisable. The routine data on which the analysis was based were not collected with this kind of study in mind. They concern past events and must be accepted at face value, since precise information on dust exposure in individual cases cannot be obtained many years later. As far as the available infor-

mation is concerned, there is no reason to suspect that the association was produced by some unrevealed biasing factor. To exclude the possibility of such a factor would require a special study however.

Removal of a worker from the dusty environment as the sole preventive measure has many disadvantages. Protective measures against silicosis must instead be primarily technical, aimed at the dust as such.

If the methodological aspects are put aside, although they deserve a discussion in the application of the present use of the case-referent method, the observed association between continued exposure to dust and the progression of silicosis has important merit with respect to biological coherence. Thus, it is plausible that easing the burden of dust on the lungs by stopping the hazardous work may be beneficial in that it at least delays progression. This action may apply to exposure to silica dust in some occupational settings but not necessarily in others.

Concluding remarks

An association was found between post-diagnostic dust exposure and the risk of radiographic progression in cases of silicosis drawn from a Swedish mining company and diagnosed in 1931—1950. The causal nature of this association was suggested but not proved.

Section IX

Case reports

One of the functions of the Pneumoconiosis Register is to draw attention with the least possible delay to cases which are remarkable by reason of causal circumstances or the early age of the afflicted person. The Pneumoconiosis Register contains a condensed form of the occupational history of each notified case. To illustrate the importance of this particular function, I have collected some cases of silicosis arising

after relatively brief exposure to dust in recent years, and also of silicosis which became manifest in workers at a remarkably early age.

This section presents some data from the histories in a few individual silicosis cases and summaries of their exposure to silica dust. These cases were drawn from among the notifications to the Pneumoconiosis Register between 1971 and 1977. Table 67

Table 67. Cases reported in 1971—1977 (30 June)
— All occupational categories.

First exposure	Number	Cumulated %
≤ 1910	2	0.5
1911—1920	13	3.9
1921—1930	77	24.2
1931—1940	109	52.9
1941—1950	121	84.7
1951—1960	47	97.1
≥ 1961	11	100.0
Total	380	

shows all the cases notified in this period in relation to time when the dust exposure began.

There was a wide range in the time when these recently reported cases were exposed to silica dust. In approximately half of them the dust exposure began after 1940 (table 67).

Tables 68 and 69 (both in appendix C) present cases with silicosis manifest before the age of 40 a (table 68) and cases with manifest silicosis preceded by less than 10 a of exposure to silica dust (table 69).

Discussion

The cases in tables 68 and 69 demonstrate one aspect of the usefulness of a central case register. Focusing attention on individuals rather than on groups can help to elucidate silicosis risks in workplaces, environments, and processes which earlier were underestimated or even disregarded in this respect. One must of course recog-

nize that, because of the interval between commencement of dust exposure and the radiographic manifestation of silicosis, the disease in many of the notified cases could be attributed to environmental conditions existing some years earlier.

The tabulated information is essentially anecdotal. Because it was intended to underline the persistent importance of silicosis, only persons with a less than 10-a history of exposure to silica dust or a statistically long life expectancy (younger than 40 at diagnosis) were selected. Though the occupational histories were greatly condensed, they and the other data serve as reminders that silicosis is not yet eradicated. There is still no room for complacency as regards our efforts to prevent silicosis. This basically man-made disease may still result from relatively brief exposure to silica dust, and it may still affect relatively young workers.

If table 67 is valid for analogous projections into the future, it may confidently be forecast that towards the end of the present century silicosis will be detected in persons whose exposure to silica dust occurred some 20 or 30 a earlier. Among them will indeed be workers performing dusty work while this monograph is being written.

Eradicating silicosis as a community health problem is a long-term project. Even if all known risks of the disease could be prevented by immediate action, earlier dust conditions in many occupations will ensure that cases of silicosis will long continue to become manifest.

Section X

General trends in the occupational environment

A comprehensive analysis of the occurrence and distribution of pneumoconiosis in Sweden was published in 1942 (12). The analysis showed that the disease was particularly common in the mining of iron ore, in iron and steel industries, in the ceramic industry, and in stone crushing and

cutting. The disease had arisen in the environmental conditions which prevailed in the century's early decades. Since then, technical and other measures have resulted in substantial changes in the environment. Such measures, though primarily aimed at increased production, nevertheless often

resulted in reduction of the injurious dusts. Sometimes they resulted in an increase in dust pollution. The sources of dust, its pathways of dissemination, and contact with workers are today the main objects of attack. Some important preventive measures in a variety of occupations and environments are substitution of safer materials for quartz-containing substances, relative or total isolation of processes in which inhalable quartz dust is released, the binding or absorption of dust, improvement of ventilation to remove dust, evolution of facilities for maintaining good general and personal hygiene, provision of protective clothing, masks, etc, and refinement of methods for monitoring hygienic standards at work sites.

These measures, singly or in combination, have been increasingly and often intensively applied in many hazardous situations in Swedish industry.

Technical progress in most occupations with inherent silicosis risk is dominated by a switch from manual to mechanized or automated procedures. Such transition often leads to increased production, with a concomitant increase in waste products and pollutants, among which may be silica dust. If the increased emission of pollutants is not counterbalanced by preventive action, more intensive dust exposure occurs and results in a heightened risk of silicosis.

Mechanization and other production-promoting measures may have additional consequences. As a result of increased mechanization, fewer workers may be needed, ie, the population at risk diminishes. In addition the reduction or elimination of manual procedures usually entails less physical exertion, and therefore the inhalation of dust may decrease.

Another pertinent point is the progressive reduction of workhours. In Sweden this reduction has primarily been achieved by legislation. In comparison with the 1920s and 30s the reduction is on the order of 25 to 30 %. In industries with pneumoconiosis risk, the shorter workweek as such means less exposure to dust.

The hygienic situation that has resulted from all of these factors has varied with time and between and within different industries; it has also involved many occupa-

tional groups throughout the country.

Thus changes have occurred in somewhat uneven stages over the past 50 a, ie, the time during which most of the silicosis in this study was generated. The population at risk — from which the study population derived — was therefore exposed to environmental conditions which underwent many changes caused by a wide variety of factors. In fact, a complex problem of exposure dynamics is being dealt with, a problem which cannot be analyzed solely from data in the Pneumoconiosis Register. One must keep in mind, however, that the changes involved various subgroups of the risk population at different points in time and that the exposure pattern varied accordingly.

The determinants of risk in the present study were limited to the nature of the occupation and the length of time spent in it. In evaluating silicosis risk, therefore, the important questions become "where," "at what time," "for how long," and "at what age" the work was done.

Mining industry

The underground extraction of minerals has a long history in Sweden. Workers have been, and still are, exposed to the risk of pneumoconiosis in mines. The ores, and the rock in which they are embedded, vary greatly as regards quartz content. The risk of pneumoconiosis from dust raised in the mining processes also varies. Iron and sulfide ores constitute the bulk of the extracted minerals. Since 1970 there has been no coal mining in Sweden.

An example of a sulfide ore with an extremely high quartz content (50—90 %) is the lead ore mined in Laisvall, in Lapland. The Lapland iron ores contain 5—70 % quartz, and the enclosing rock 3—40 %.

The sulfide ores mined in the Skellefteå district in northern Sweden contain approximately 50 % quartz. In the Bergslagen district of central Sweden iron ores with quartz content ranging from 30 to 70 % are extracted from rock containing 20 to 40 % quartz. The coal mining that

earlier took place in southern Sweden carried a small risk of pneumoconiosis in comparison with coal mining in other countries. The quartz content in the coal seams and enclosing strata was often less than 10 %. Many coal seams were enclosed in clay, and the resultant dampness tended to counteract the risk of dust disease.

A number of processes in the extraction of ore, mainly drilling, blasting, cutting, loading, and transporting, involve a risk of pneumoconiosis. In drilling, the initial dry procedure has gradually been superseded by methods using water to bind the dust, beginning in the 1920s. The suppression of dust by water spraying has been extended to other mining processes. A practical problem with water spraying in cold climates such as Sweden's is the freezing of the water, and experience has shown that it can lead to increased atmospheric dust during the cold season. Another important measure for dust control is ventilation. Constructing and maintaining ventilation systems in mines can be considerably difficult, however, because of the need to transport fresh and exhaust air over long distances and extend the system in order to keep pace with the extension of mine workings.

Iron and steel industries

The risk of silicosis arises mainly in foundry processes. Preparing molding and core sand involves such a risk, as also do the molding of castings, the knocking-out of molds and cores, and the cleaning of castings.

These processes have undergone many modifications in the past 50 a, and the emission and dissemination of their injurious dust has, as a consequence, changed. In many instances the mechanization and automation of the processes have reduced the time during which dust is generated. The replacement of siliceous by nonsiliceous materials, for instance, quartz sand by olivine sand in fettling shops and sand-blasting activities, has also contributed to a reduction in silicosis risk. The enclosure of dusty processes and refinements in ventilating systems are other factors that have contributed to general improvement.

Ceramic industry

The ceramic industry covers a wide range of products, from silica bricks and tiles to sanitary ware and domestic porcelain. The raw materials include quartz. Dust may be released in the crushing, grinding, and sifting of these materials and in their further processing. Moreover, some quartz-containing waste products are recycled for use in the production processes.

In the ceramic industry the general principles for technical prevention against dust disease have been followed. Considerable efforts have been made to develop wet techniques adapted to the industry's special needs. Already during the late 1930s programs for technical preventive action were required by the supervising authorities.

Quarrying and working of stone

Sandstone and granite are the main types of stone cut and quarried in Sweden. They are used in the building industry, in road construction, for monumental masonry, etc. The risk of silicosis is largely dependent on the content of quartz in the raw stone and on the nature of the processing.

Manual drilling and other manual procedures in stone quarries have gradually been mechanized. Mechanical drills were introduced around 1910—1920. Dust suppression by water has a long history in many quarries and has increasingly been adopted. Pneumatic drills replaced steam-driven ones after the 1920s, and about 20 a later the transition was complete. Technical improvements have resulted in an increased durability of the component parts of drills and greater production capacity. Improvements in exhaust ventilation came relatively late — towards the end of the 1960s.

In the processing of stone there has been a comparable evolution, with transition from manual to mechanized procedures in the 1920s and 30s and subsequent refinements leading to increased productivity. Exhaust ventilation was introduced in the early 60s.

The technical measures for reducing exposure to dust in many industries have been reviewed by Gerhardsson (18), Gerhardsson et al (19), and Öhman (35).

Section XI

Measurements of environmental dust

Generally speaking, measurements of air pollutants are important in the assessment of hygienic standards. The measurements may be useful also for describing exposure to pollutants. The problems in setting a threshold limit value (TLV) for quartz based on estimations of the previous exposure of workers and on epidemiologic studies of dose-response relationships have been discussed by Jahr (23). Öhman has, in a similar manner, discussed the concept of an acceptable life dose for silica dust (35).

The Pneumoconiosis Register does not include measurements of silica dust. Comments on some Swedish field projects in which data on dust were collected from various industries nevertheless seem to be appropriate. It is essential to recognize that dust conditions prevailing early in the present century are difficult to reproduce and, accordingly, to measure. The data discussed thus illustrate relatively recent situations. They cannot unreservedly be projected into either the future or the past.

Before the 1950s there were only sporadic measurements of dust levels in Swedish workplaces with silicosis risk. An account of early dust measurements in Swedish mines, performed with a direct microscopic technique, was published by Pontén in 1939 (38). One of the early surveys was that performed by the National Institute of Public Health from 1947 through 1957. An account of this survey, including the dust measurements, was published by Ahlmark & Öhman (6). These surveys gave valuable insight into the identification of dusty occupations and provided helpful guidance for subsequent studies.

From the 1960s on, measurements of industrial dust have been performed on an increasing scale. Between 1963 and

1967 the National Institute of Public Health (since 1966 the National Institute of Occupational Medicine) undertook an extensive continuation of the earlier survey, aimed at a more systematic collection of information on current silicosis hazards in Swedish industry. This "Silicosis Investigation" was made in collaboration with the National Board of Occupational Safety and Health. The results were published by Ahlmark (2).

The Silicosis Investigation was followed by the "Silicosis Project," conducted from 1968 through 1971 by the National Institute of Occupational Medicine. This project aimed at defining the risk of silicosis in occupational settings with quartz dust. The results were published by Gerhardsson et al (19).

Some data abstracted from the reports are presented and discussed in this section. The techniques of sampling and analysis used in them were described by Öhman (34) and Gerhardsson et al (19).

Tables 70 and 71 show results from measurements which were made at different times. The results are comparable because the technique was the same — the gravimetric membrane filter method. This filter effectively separates dust particles > 0.0001 mm in diameter from the sampled air. The fraction with a diameter of < 0.005 mm is determined with a fractionated sedimentation technique after dispersion of the filter contents in ethyl alcohol (34).

In tables 70 and 71 the sampling times are presented as 1963 (the Silicosis Investigation) and 1968 (the Silicosis Project). The abstracted data include only procedures which could be identified under the same headings in both reports. Only measurements carried out with fixed sampling points are included. These measurements were as a rule carried out during 24 h,

three 8-h shifts. Mobile equipment to estimate levels of dust exposure for individual workers underwent changes between 1960 and 1970, and comparisons of such measurements are difficult. Measurements made with mobile equipment were therefore excluded.

In table 70 total dust concentrations in iron foundries are presented as arithmetic means and as median readings. [For the numbers of samples and other details the reader is referred to the original publications.] Table 71 gives corresponding data from the mining industry. The results presented in tables 70 and 71 indicate levels of silica dust which were, by and large, the same in 1963 and 1968.

Discussion

The figures in tables 70 and 71, which were abstracted from earlier reports, cannot be interpreted as unanimously reflecting improvements in occupational environments between 1963 and 1968. Indeed, some figures suggest a deterioration of hygienic

standards, whereas others suggest an improvement.

In any attempt to interpret these readings, a few principal aspects of the problems of measurement should be considered.

The atmospheric content of dust in workplaces varies greatly. These variations are caused by many factors. The work process may be intermittent, and climatic conditions such as temperature, humidity, and air flow may be inconstant. If measurements are made at intervals, such factors may result in differences in the readings. To select samples to produce a representative picture of the true work situation is therefore a complex and difficult task. Consequently, discrepancies between results of measurements made by different persons at different times at the same or different workplaces should be circumspectly approached. The divergencies may have arisen from variations in strategies of sampling, in the selection of measuring sites, or in other factors and thus by no means reflect true differences in the compared situations. Conversely,

Table 70. Dust characteristics of stationary sampling points measured in 1960 and 1970 — Iron foundries.

Occupation	Mean/median total dust concentration (mg/m ³)		Quartz in dust (%)		Particles ≤ 5 μ (% of weight)	
	1963	1968	1963	1968	1963	1968
Fettling	12.8/10.0	12.7/8.8	14	13	19	22
Sand preparation	11.4/ 9.3	16.3/9.5	8	6	28	30
Knocking out of castings	11.7/ 8.8	15.4/10.8	8	8	20	23

Table 71. Dust characteristics of stationary sampling points measured in 1960 and 1970 — Mining.

Occupation	Mean/median total dust concentration (mg/m ³)		Quartz in dust (%)		Particles ≤ 5 μ (% of weight)	
	1963	1968	1963	1968	1963	1968
Drift mining (unspecified)	8.3/5.8	5.3/3.8	17	13	27	32
Ore mining (unspecified)	7.8/5.0	11.3/3.3	16	8	23	18
Haulage	3.9/3.4	6.4/4.4	22	7	34	18
Crushing	4.5/3.7	6.9/4.2	31	12	33	37

identical results in separate measurements may in fact derive from widely differing environments.

The two projects from which the data in tables 70 and 71 were extracted were not designed to provide material for an analysis of trends. Their objective was primarily to obtain a basis for preventive measures in specific industries. The results were used locally. In some instances they were brought to the attention of the national Factory Inspectorate, when intervention at that level was judged to be appropriate. The wealth of data accumulated in the projects also included descriptions of work processes and other circumstances regarded as relevant for an overall assessment of local silicosis risk.

The projects were thus intended to provide a cross-sectional view of the situation at the studied workplaces. They were service-oriented in the sense that they were meant to provide an incentive for preventive action at the individual workplaces. Great pains were taken to ensure uniformity in sampling, general strategy, and measurement techniques. The procedure was carefully rehearsed and closely supervised. In some plants the data were used as a basis for evaluating specific preventive measures at the company level (35). The measurements were made at two different, though undoubtedly to a

large extent overlapping, sets of workplaces.

If one considers how the data have been generated in the cited projects, it is obvious that they are of limited usefulness for estimating changes in environmental risk factors. The measurements do not preclude the probability that hygienic standards, in regard to risk of silicosis, have improved in important respects. On the contrary, the likelihood is great that progress has been made from hazardous to safer conditions. But this is a likelihood that, despite its credibility, must be taken on trust. The contention that environments responsible for the silicosis dealt with in this study have recently undergone nationwide changes thus has not been scientifically proved by the surveys hitherto published.

It is, of course, possible that individual companies have performed measurements which permit a more reliable judgement of environmental conditions and which have not been published.

A relevant point is that practically all the workers in the Pneumoconiosis Register were exposed to dust long before the dust measurement surveys were undertaken. One can only say that the dust situations in those years were very likely at least of the levels reflected in tables 70 and 71. In many instances the conditions were probably much worse.

General discussion

This section deals with some of the broader implications of the use of a case register for analytical purposes. The present study is based on data collected in a system of routine reporting. Systems for the routine collection of data relating to aspects of health constantly require critical evaluation for their correct maintenance and further development.

In a review of various types of registers, Weddell (44) emphasized the importance of

clear-cut objectives. The needs that the information system is intended to satisfy, and that cannot be met in any other way, must be identified. The issues involved in the planning of disease registers for various uses were surveyed by Brooke (11). She pointed out that the register's purpose is a determinant for the data to be collected and for the methods of analysis to be employed.

Main problems in the routine reporting

of diseases are the deficiencies regarding the completeness and accuracy of the collected data (7). The completeness of registration depends upon the following factors:

- (a) existence of medical services,
- (b) ease of access to such services,
- (c) general attitudes towards seeking medical advice,
- (d) standards of diagnostic accuracy,
- (e) notification coverage of detected cases.

The specificity of routinely collected data relative to the needs of special studies is another problem area. It is common experience that data routinely collected for more general purposes do not meet requirements for special studies. Such experience has sometimes brought routine data into undeserved disrepute. Routine collection of data does not as a rule obviate needs for special surveys in order to examine problems more closely.

Defining a basis of comparison for a study population may also constitute a problem. The data collected by a disease register, in this instance the Pneumoconiosis Register, concern persons with a particular disease. The healthy population from which these cases are drawn, the denominator, remains unknown unless direct efforts are made to obtain the requisite knowledge. The better the total risk population is known with regard to risk factors, the more rewarding the study of these factors will be with respect to the cases comprising the numerator (42).

In a discussion of the need to register incident cases of pneumoconiosis, the general problems of incomplete and inaccurate registration, absence of population basis, and inability to satisfy the needs of special studies should all be recognized. They also apply to pneumoconioses other than silicosis.

The following features of silicosis create particular difficulties: (i) silent clinical course, (ii) late manifestation, and (iii) nonspecific radiographic signs of early disease.

As a consequence of these features, the records in a pneumoconiosis register consist of what actually is reported and not necessarily of what ought to be reported. The incidence rates are dependent on the

adequacy of the case-finding procedures, including the diagnostic standards.

Full identification of existing cases requires extensive screening. Such screening must include persons whose dust exposure ceased many years earlier, particular attention being paid to elderly people. The true epidemiologic picture of silicosis is difficult — if not impossible — to extract from a case register, since there will always remain undetected cases of the disease.

On the other hand there are features which should also be taken into account in a discussion of disease registration. They are specificity in causal association and diagnostic reliability in developed stages of silicosis.

Causal specificity refers to the single-factor etiology. The single risk factor in silicosis is quartz dust, including some of its crystalline modifications. Therefore the risk population, or at any rate the part of it currently exposed to quartz dust, should be possible to identify.

The reliability of diagnosis refers to the radiographic manifestations of fully developed disease which can be described and identified with reasonable uniformity by specialists.

A medicolegal aspect with some relevance to this discussion is that the registration of silicosis is of interest to persons with the disease. Silicotic persons may be eligible for disability benefits. Registration can therefore be regarded as part of the social security system. Routines for accurate basic reporting from populations at risk are easy to establish, by legal means if necessary, and the routines can readily be extended to the notification of cases detected outside of regular monitoring programs.

A discussion of the need to register incident cases of pneumoconiosis cannot disregard the possible uses of the registered information. One should keep in mind the statement made in 1967 by a WHO Expert Committee on Health Statistics:

The term "registration" implies something more than "notification." A register requires that a permanent record be established, that the cases be followed-up, and that basic statistical tabulations be prepared both on frequency and survival. In addition, the patients on a register should frequently be the subjects of special studies [46].

As regards silicosis, follow-up programs serve not only scientific ends, but also are in the direct interests of affected persons. Apart from financial considerations, the early recognition and treatment of possible complicating disorders such as tuberculosis is advantageous.

In this particular aspect — the follow-up function of a register — one relevant factor is the facilities for follow-up. In Sweden, as in the other Scandinavian countries, the system of unique personal identity numbers facilitates the tracing of individuals and the linkage of records. Ease of tracing is an important prerequisite for successful follow-up.

When considering the question of how the registration of a disease such as silicosis should best be done, one is faced with various alternatives. The final answer will depend on what needs the registration is intended to satisfy.

A pneumoconiosis register can provide a means for the following:

- (a) an estimation of the extent of the disease problem and description of time trends,
- (b) a facilitation and operation of follow-up procedures,
- (c) a study of the natural course of the disease in subgroups of the case population,
- (d) a study of the mortality, total and cause-specific, in the subgroups, and
- (e) a study of association between pneumoconiosis and coexisting diseases of the lungs, such as tuberculosis and cancer.

The register can be a useful tool for these objectives, but it depends on completeness in registration, adequacy in diagnostic standards, facilities for follow-up, and possibilities for defining populations at risk.

Deficiencies with regard to these requirements result in limitations in the usefulness of the collected data. The possibilities of satisfying the requirements can be expected to vary between and within countries and also within different industries.

The value of centralized registration lies partly in the statistical aspects of the disease. A central disease registration can,

if circumstances are favorable, be made to provide a nationwide panoramic view of the disease occurrence. The centralized collection and processing of cases from small risk populations throughout the country gives numbers amenable to statistical analysis. The issue of central versus peripheral registration also has a quality aspect. Diagnostic and environmental data are more easily standardized in a central processing of information than in a system where it is done by a number of peripheral units, particularly for a disease with a relatively low incidence in the general population and one that requires the skills of specialists for correct diagnosis. Pneumoconiosis is a such disease.

Data on registered cases of a disease such as pneumoconiosis thus have many potential uses, particularly if the data are properly handled and their limitations are kept in mind. They can describe the current position in a problem area and also reinforce previously acquired knowledge. Moreover, the routinely collected data can point to problems which warrant more penetration in special studies.

By focusing on individual cases, one can assemble study populations with special characteristics. Such characteristics may, for instance, concern occupational circumstances which were previously unrecognized as carrying a risk for pneumoconiosis.

On the other hand, one must recognize that disease risk in a population exposed to risk factors cannot be investigated solely from the data of a case register. Truly population-based studies are essential for measuring and monitoring such risks. The analyses must then include information on exposure parameters in both the total exposed populations and the cases of disease.

The present study is based on cases of silicosis centrally registered in 1931—1975. In the register particular care has been taken to maintain high quality for all recorded information. Each registered case has been assessed with a uniform procedure by specialists during both the registration and the follow-ups. Without these efforts over the many years, this study would not have been possible.

I must concede that in the present study the problem of incomplete reporting is not under full scientific control. It is in fact

hard to see how it ever could be — at least on a nationwide scale — due to the difficulties described earlier.

However, I would like to emphasize that data retrieved from industries aware of the risk and with access to medical in-plant services can be assumed to be reasonably complete. I have accepted this assumption as valid enough for the conclusions I have drawn.

A pneumoconiosis register should be regarded as part of a health information system on pneumoconiosis. The register is justifiable if the collected information is as complete as possible and if it is used to increase knowledge of pneumoconiosis and to prevent the disease or its complications. Data collected without such use in view are of doubtful value.

Silicosis and many other types of pneumoconiosis, one must remember, are pre-

ventable, man-made diseases. As the present study has demonstrated, silicosis still occurs and it or its complications may be fatal. The effective prevention of silicosis is achievable only with the elimination of silica dust. This prevention demands strategies. As Tunstall Pedoe (42) pointed out. "If there is no strategy, no manoeuvring, but simply hand-to-hand combat using any weapon available at any point with no questions asked, then no intelligence system is needed."

If silicosis is to be eradicated in a country, the scale of measures aimed at such elimination must be nationwide and involve many industries and occupations. Whatever strategy is chosen with a view to total eradication, an information system is required to describe and monitor occurrence of the disease and to provide opportunities for analyzing its consequences.

General summary

The monograph provides a picture of some trends and developments of silicosis in Sweden, as they are reflected in a central register for notified cases. The number of notifications of silicosis per year is, at present, 80—100.

The basic study population consisted of 4,590 cases of silicosis, including mixed-dust fibrosis, diagnosed during the time periods 1931—1950, 1951—1960, 1961—1975. The study population is presented as basic tabulations in section II. The section provides a descriptive account of the distributions and trends with regard to age at the radiographic manifestation of silicosis, latency time (time from commencement of dust exposure to silicosis manifestation), and radiographic disease stage at diagnosis. Data are presented for the total study population and for occupational categories.

The monograph presents the results of follow-up investigations and analyses focused on topics of particular interest. They concern the radiographic progression of

silicosis (section III), complicating pulmonary tuberculosis (section IV), overall mortality (section V), lung cancer (section VI), cause-specific mortality and competing mortality risks (section VII), and the possibility of an association between continued dust exposure after diagnosis and radiographic disease progression (section VIII). The occupational environments with risk of silicosis are dealt with in sections X and XI. The implications of using a disease register as a source of information in analyses are presented in the general discussion.

Regarding *basic tabulated variables*, the registered data showed the following trends during the observation period:

- (a) a rising mean age at the manifestation of silicosis,
- (b) an increasing mean length of prediagnostic exposure time,
- (c) a diminishing number of cases diagnosed in advanced stages of disease.

These trends are consistent with the concept of an improving environment. They should not be accepted as evidence of such improvement since other possible explanations exist.

A long-term observation of the tendency towards *radiographic progression*, as measured with the Johannesburg classification of silicosis, showed differences between five occupational groups with regard to rates of verified progression. The rates ranged between 25 and 70 % over a 20-a period of observation. Progression may occur as a late event.

Tuberculosis continues to be an important complication in silicosis. The overall risk of contracting pulmonary tuberculosis in cases diagnosed in 1951—1969 and followed through 1970 was observed to be seven times greater than the risk in the general population. The true risk is probably even greater, 10- to 20-fold. The data indicate differences in risk between various occupational groups. Complicating tuberculosis may occur as a late event.

The *overall mortality* was observed to be higher in the study population than in the general population. This excess risk was observed both for cases notified in 1931—1948 and for cases notified in 1949—1969. The excess risk was particularly high for cases notified in stage II or III of silicosis and for cases with verified or radiographically suspected tuberculosis of the lungs.

Subgroups of the study population showed a decrease in mortality risk when cases notified in 1931—1948 were compared with those notified in 1949—1969. This finding is consistent with the concept of an improving environment resulting in a diminished impact of disease. It should not be accepted as evidence of such improvement since other possible explanations exist.

An analysis of *cause-specific mortality* showed that silicosis and tuberculosis are important registered causes of death in the study population, particularly in cases notified in advanced stages of disease. The data indicate that the excess mortality risk is attributable to silicosis and tuberculosis.

Silicosis and tuberculosis compete with

other causes of death. This competition should be taken into account in attempts to assess the full impact of the disease.

Cases of silicosis notified from mining, quarrying, and tunneling, and from the iron and steel industries, were under greater risk of death from *lung cancer* than the general population.

In a study population selected from the mining industry an *association* was observed between continued exposure to silica dust after time of diagnosis and a tendency towards radiographic progression. The causal nature of the association was suggested but not proved.

Dust measurements obtained in nationwide surveys from some of the occupational settings in which silicosis cases were generated did not support the concept of improving environmental conditions during recent years.

The present study also illustrates the many problems associated with studies of the trends and developments concerning a disease such as silicosis and the limitations in analyses with a case register as the main source of information.

Data collected in a pneumoconiosis register can provide a cross-sectional view of the situation at any given time. They can indicate trends and suggest hypotheses to be explored in special studies. Furthermore, the data can provide a basis for longitudinal studies of the disease course (progression), complications (tuberculosis, etc), and mortality risks. Such studies may reveal differences between silicosis arising in different occupations. The register data alone do not, however, permit comparative estimates of silicosis risk among groups with different degrees of dust exposure. For such assessments, population-based studies are needed.

A register for the collection of data on cases of silicosis should be regarded as part of an information system concerning a chronic and irreversible occupational disease which has not yet been eradicated.

The value of a silicosis register depends ultimately on how the collected data are used to combat the occurrence of the disease and its complications.

Acknowledgments

The present study was carried out at the Department of Hygiene, Umeå University, Umeå, and the National Swedish Board of Occupational Safety and Health, Stockholm, Sweden.

My thanks are due to all those who, in many ways, gave me support during the study and the writing of this monograph.

I wish to express my gratitude to Professor Axel Ahlmark and Professor Torsten Bruce, both veterans who have achieved high distinction in the battle against silicosis and both of whom were my tutors, for their unfailing support in every way and at all stages of the work.

I also wish to thank:

Mr Rein Maasing, BSc, for his coauthorship, statistical advice and help, including all the computer processing involved.

Mr Lars Holmlund and Ms Inga Segelberg, BSc, for their assistance with some of the statistical analyses.

Ms Inga Sjödin for her dedicated and indispensable help with the abstraction and arrangement of the data from the Pneumoconiosis Register for the analyses.

In the course of my writing, I took counsel with friends whose advice I value. I accepted many of their suggestions and rejected others. My sincere thanks are due to:

My wife, Assistant Professor Barbro Westerholm and Assistant Chief Physician Henrik Nordman for reading the full manuscript and for providing much constructive criticism.

Professor Gunnar Eklund for his valuable discussions and suggestions regarding the use of the case-referent method.

Professor Gideon Gerhardsson and Mr Harry Öhman, both occupational hygienists, for reading and criticizing the chapters describing the environment where silicosis hazards are to be found.

The writing-up of a study comprising the description and analysis of data collected in a register over a long time is very much dependent on editorial and secretarial work. My sincere thanks are due to:

Ms Ingrid Nordenstam for her proficient and painstaking editing and typing of the manuscripts and for her infinite patience with my innumerable amendments.

Ms Gun Berglund and my daughter Ms Anna Westerholm for drawing the diagrams.

Mr Ove Larsson, Ms Anita Lindwall, Ms Maritha Lundgren, Ms Marianne Mörner, Ms Gun Sjöö, and Ms Ann-Christine Wetterstål for their secretarial help at various stages of the work.

Ms Georgianna Oja, BA, for having prepared the manuscript for publication.

The efforts of many have gone into the preparation of this monograph.

The responsibility for the conclusions and the views expressed remains mine.

The study was supported financially by the National Swedish Board of Occupational Safety and Health and by the Swedish Working Environment Fund.

Peter Westerholm

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Appendix A

Occupational codes

Mining, quarrying and tunneling

Mines A. Quartz > 50 %

Laisvall — 90 % (lead ore)

- 01 Drillers, haulers, loaders
- 02 Other workers below ground
- 03 Concentrating-plant workers
- 04 Persons with varying occupations

Skellefte mines — 50—60 % (sulfide ores)

- 05 Drillers, haulers, loaders
- 06 Other workers below ground
- 07 Crushing-plant workers above ground
- 08 Persons with varying occupations

Copper, zinc, sulfide pyrite, etc, mines

- 14 Drillers, haulers, loaders
- 15 Others workers below ground
- 16 Persons with varying occupations

B. Quartz < 50 %

Iron ore mines (Lapland and central Sweden)

- 09 Drillers, haulers, loaders
- 10 Other workers below ground
- 11 Crushing-plant workers above ground
- 12 Sorters and concentrating-plant workers
- 13 Persons with varying occupations

Coal mines

17 Drillers, haulers, loaders

C. Work in two or more types of mine

18 All workers

Quarrying, crushing and grinding of quartz

19 Drillers, loaders, sorters below ground

20 Drillers, cutters above ground

21 Crushing-plant workers (including mixers and packers)

84 Clay preparation (quartz content in clay > 25 %)

22 Persons with varying occupations

Quarrying and working of sandstone

23 All workers

Quarrying and crushing of feldspar

24 All workers

Quarrying and working of granite, tunneling in rock

25 Tunnelers

26 Crushing-plant workers

27 Cutters and dressers

28 Persons with varying occupations

29 (original category altered)

30 Crushing and grinding of flint-containing limestone

31 All workers, other occupations not classifiable under numbers 1—30

Metal foundries

Steel foundries

32 Ladlemen

33 Furnace masons

34 Sand preparation workers

35 Molders

36 Knock-out men

37 Persons with varying occupations in molding shops

38 Fettlers

76 Slag sorters (nonmechanized sorting)

40 Workers in more than one of categories 32—33, 34—37, and 38—39

Iron foundries

41 Furnace masons

42 Sand preparation workers

43 Molders

44 Fettlers

45 Other workers in molding and fettling shops

46 Persons with varying occupations

Silicon alloy smelting works

47 All workers

More than one foundry type

48 All workers

Ceramic industry

Potteries

49 Sliphouse workers

50 Forming-shop workers (throwers, turners, finishers, etc)

51 Furnacemen

52 Glazers

53 Persons with varying occupations

Making of silica bricks

54 All workers

Making of other bricks

81 Brickyard and tilery workers, including furnace workers

Glassworks

55 Potmakers

82 Furnace repairers

85 Blenders of quartz-containing constituents of glass making

Work in two or more branches of the ceramic industry

87 All workers

Other industries and occupations

(code numbers not given)

Sand blasting (metal, glass, earthenware, etc)

All workers

Metal grinding against stone

Against sandstone

Against quartz-containing synthetic stone

Against synthetic stone and sandstone

Sand blasting plus grinding

All workers

Filing and polishing of dental prostheses

All workers

Enameling

All workers

Drilling in concrete

All workers

Quartz filler work in rubber making

All workers

Scouring powder making (with quartz content)

All workers

Furnace repairing in gas works

All workers

Tile stove making and repairing

All workers

Calcining and milling of diatomaceous earth

All workers

Furnace repairing in papermills

All workers

Roofing slate work (Norwegian slate, 20 % quartz)

All workers

Work in two or more of the foregoing main categories
(code numbers not given)

Mining, quarrying and tunneling + foundries

Mining, quarrying and tunneling + ceramic industry

Foundries + ceramic industry

Foundries + other industries

Mining, quarrying and tunneling + foundries + ceramic industry

Mining, quarrying and tunneling + other industries

Ceramic industry + other industries

Mining, quarrying and tunneling + ceramic industry + other industries

Appendix B

Statistical methods

by Rein Maasing, Peter Westerholm

The general background of the study was described in the introductory chapter of the monograph.

The cases from the Pneumoconiosis Register were followed from their date of entry into the study, ie, from notification of silicosis, until the closing date of the study or until death, whichever occurred first. The follow-up is illustrated schematically in fig B-1 where each line denotes an individual case. The heavy portion of the line indicates the period in which the case was observed during the study (beginning at notification of silicosis).

Fig B-1. Schematic presentation of the age-time relationship for follow-up of cases from the Pneumoconiosis Register. (Death is denoted by †)

The possibility that the mortality risks among the workers with silicosis differed from those in a reference or "standard" population was statistically analyzed. The null hypothesis (H_0) was that the risk of death in the *index population* (silicosis cases) did not differ from the risk of death in the *reference population*. The corresponding alternative hypothesis (H_a) was that the risk of death differed between the index and the reference populations.

Two methods were used for these comparisons. In the first approach the unit of time was the calendar year. For each separate year of the study the deaths among the index population were counted. They were compared with the deaths in the corresponding sector of the reference population. The variable thus was number of deaths — observed and expected.

In the second approach the time unit was the year of follow-up. The starting point in each case was date of entry into the study. The variable in this method was the probability of death or survival. This is an actuarial method, usually referred to as a life-table method.

Population rates

Since the silicosis cases in the Register had been reported from all parts of Sweden and from a wide range of occupations, it was deemed appropriate to use the population of the whole country as the reference.

For calculation of the expected number of deaths (expected mortality risk) the matrices illustrated in fig B-2 were used.

The risks in category A (years 1901 through 1970 and ages up to and including 94 a) were extracted from the annual mortality statistics (5) published by the Central Bureau of Statistics.

The risks in category B (1971 through 1974 and ages up to and including 94 a) were calculated as the ratio of annual numbers of deaths to the midyear population in age classes with 5-a intervals.

The source was again the Central Bureau of Statistics.

In category C (1901 through 1974 and ages 95 through 99 a) the annual mortality rates were calculated on the assumption

of an annual mortality risk increase of 10 % at ages 95 to 99 a. These small computational differences were due to changes in the contents and presentation of the mortality statistics published by the Central Bureau of Statistics.

Calculations based on calendar years

Individual contributions to the number of expected deaths in each year were calculated according to the formula

$$e_{xy} = q_{xy} \pi p_{ij}; a < x; b < y; e_{ab} = 1/2 q_{ab},$$

$$i = a$$

$$j = b$$

where e_{xy} is the contribution to expected deaths during the calendar year x and at age y and q_{xy} is the mortality risk in the general Swedish population for persons who are y years old during year x . The probability of survival is

$$p_{xy} = 1 - q_{xy}.$$

For this first calendar year of follow-up the survival probability (p_{ab}) was calculated as

$$p_{ab} = 1 - 1/2 q_{ab},$$

since it could reasonably be assumed that entries were evenly distributed during the year, with the average at the midyear point.

The individual e_{xy} were added to give E_{xy} as the expected number of deaths among those who reached or would have reached age y years during year x .

A summation of figures from the age groups and/or calendar years gives expected numbers of deaths during these years and/or in the age groups. Since the sums for each calendar year were rela-

Fig B-2. Schematic example of risk matrices used for calculating expected mortality.

tively small, they were cumulated in the tables and diagrams. In order to illustrate the distribution of entry points for the groups under study, the numbers in the tables and diagrams were transformed into cumulative percentages. These denote the proportion of the group under study which was observed from entry until that particular year.

Observed numbers of deaths

The observed deaths were classified according to year of occurrence and age, giving O_{ij} .

Summations of observed deaths according to categories i and/or j gives

$$O_{.j} = \sum_i O_{ij} \text{ (age category total),}$$

$$O_j = \sum_j O_{ij} \text{ (year total),}$$

and

$$O_{..} = \sum_i \sum_j O_{ij} \text{ (grand total).}$$

Comparison between observed and expected numbers of deaths

For simplicity, the expected number of deaths was denoted as E , and the observed number with the letter O . Since the risk of dying was small and the number of deaths fairly proportional to the length of the time interval, it is reasonable to assume that the number of deaths follows a poisson distribution. For the determination of whether O was a probable outcome in a poisson distribution with the mean E , the chi-square test was used, giving

$$\chi^2 = (O-E)^2/E.$$

For small values of E the poisson distribution was applied directly.

The relationship between O and E in each time interval can be expressed as an index ($SMR = \text{standardized mortality ratio}$) as

$$100 \times O/E.$$

This percentage can be regarded as the degree to which the expected mortality in the population during that particular interval has been utilized.

When E is not very small, the hypothesis

$$H_0 : SMR = 100$$

can be tested by

$$Z = \frac{O/E - 1}{1/\sqrt{E}},$$

where Z is a standardized normal deviate (1).

When the same standard population is used in calculations of E in different sub-groups, as in this study, the various SMRs can be compared. The variance of SMR is then

$$\text{Var (SMR)} = 100^2 \times \text{Var (O/E)} = 100^2 \times O/E^2.$$

The test variable for hypothesis H_0 ; $SMR_1 = SMR_2$ when E is fairly large is

$$Z = \frac{SMR_1 - SMR_2}{\sqrt{\text{Var (SMR}_1) + \text{Var (SMR}_2)}} = \frac{O_1/E_1 - O_2/E_2}{\sqrt{O_1/E_1^2 + O_2/E_2^2}}$$

Graphic presentation of the data

In order to illustrate the developments up to year i in mortality, E_i and O_i were calculated. Since the individual values of E_i were small, the expected and the observed numbers were cumulated over periods of some years. The cumulated sums in a specific year are most appropriately regarded as the result that would have been declared if the cohort study had been closed that year.

For each calendar year the index

$$\frac{\text{Observed cumulated number of deaths}}{\text{Expected cumulated number of deaths}} \times 100$$

was calculated and used in diagrams for descriptions of the mortality experience of the index population.

Proportional mortality

In section VI (on lung cancer) a proportional mortality technique of analysis was used. The objective was to examine whether there was a demonstrable shift in the proportion of cases dying from lung cancer in the index population as compar-

ed with the general population. The following table was set up:

Cause	Observed	Expected
C ₁	O ₁	E ₁
C ₂	O ₂	E ₂
⋮	⋮	⋮
C _r	O _r	E _r
⋮	⋮	⋮
$\sum_i$	O	E

The conventional test for goodness of fit to the chi-square distribution is:

$$\chi^2 = \frac{\sum (O_i - E_i)^2}{E_i}$$

This test is applicable when $\sum O_i = \sum E_i$, that is when $O = E$.

One of the problems encountered in this study was that $\sum O_i$ was greater than $\sum E_i$. There was high mortality in the index population. This occurrence meant that the observed pattern of causes of death was compared with an expected pattern which was not equivalent with regard to age and time. An increased mortality rate by definition means that persons in the index population die earlier and, consequently, younger than the general population, and a correction of values in the E column is needed.

In the present study this correction was achieved with the calculation of E_{ij}^r as

$$E_{ij}^r = O_{ij} \cdot \frac{A_{ij}^r}{A_{ij}}$$

where O_{ij} = total number of observed deaths in the age and time category ij of the index population, A_{ij}^r/A_{ij} = proportion of all deaths due to cause r in category ij in the general population, A_{ij}^r = number of deaths from cause r in category ij , and A_{ij} = number of deaths from all causes in category ij .

This correction results in expected numbers of deaths due to different causes, distributed according to the pattern existing when the deaths in fact occurred.

Comments

This method of statistical analysis differs in one important respect from the most commonly used methods (4). The difference concerns the calculation of expected numbers of deaths.

In many studies, expected mortality is calculated from sex-specific matrices where each element, M_{ij} , denotes the number of person-years at risk during year i , in age group j . The elements are multiplied by the risk value, q_{ij} , and the summed up results yield E = expected total deaths.

Theoretically this procedure is debatable. It involves a summation of outcomes which are not independent since the q_{ij} 's are conditional probabilities. In short follow-up and young age groups this is of no great practical importance. With a long follow-up into a higher age it may have consequences. The annual mortality risk in old age is considerable. Therefore persons remaining in the study into old age may contribute to the expected mortality with even more than 1, while they can only contribute to the observed number of deaths with 0 or 1. In extreme situations one may find an expected number of deaths greater than the size of the cohort.

With the method used in the present study, which is simply the multiplication rule for conditional probabilities, expected and observed mortality rates were strictly separated. Observed events thus were not allowed to influence expected events. The contribution made by each individual to the expected mortality during the total years of observation does not exceed 1.

Calculations based on years of follow-up

In order to estimate the probability of dying, a basic table was set up, with time intervals of 1 a (2).

←— No withdrawal —→										←— Withdrawal									
N _x	m _x	s _x	d _x	d _{1x}	...	d _{rx}	n _x	w _x	d' _x	d' _{1x}	...	d' _{rx}							
Interval																			
0—1 year																			
1—2 years																			
2—3 years																			
etc																			

Each person entering the study started at the top of the table [time (x) = 0]. The symbols used in the table are illustrated in fig B-3, which covers the time intervals [x, x + 1] and [x + 1, +2]. The oblique line represents the closing date of the study. The shaded areas are observation times < 1 a.

Thus, $w_x + d'_x = n_x$ = cases withdrawn in interval [x, x + 1], since they did not complete a full year of observation before the closing date of the study; $s_x + d_x = m_x$ = cases not withdrawn in interval [x, x + 1], since they could complete a full year of observation before the closing

date; $d_x = \sum_r d_{rx}$ = total deaths, where r is a specific cause of death (r = 1,).

The probability of survival at x + 1 for a person alive at X was calculated as

$$p_x = \frac{(\frac{1}{2}d_x + \sqrt{\frac{1}{4}d_x^2 + 4(N_x - \frac{1}{2}n_x)(s_x + \frac{1}{2}w_x)^2}}{2(N_x - \frac{1}{2}n_x)}$$

the conditional risk of death being

$$q_x = 1 - p_x.$$

The approximate error of this estimate is

$$s(p_x) = \sqrt{\frac{p_x q_x}{M_x}},$$

where $M_x = m_x + n_x / (1 + \sqrt{p_x})$.

This expression differs from the standard error of a proportion by the correction

Fig B-3. Illustration of symbols used in calculating mortality in relation to duration of follow-up. Each line = 1 case. (Intervals [x, x + 1] and [x + 1, x + 2] = 1 year; w_x = survived closing date of study; d'_x = died in time interval [x, x + 1], which was interrupted by the closing of the study before reaching length of 1 year; s_x = survived time interval [x, x + 1]; and d_x = died in time interval (x, x + 1] reaching length of 1 year before the closing of the study.

term $1/(1 + \sqrt{p_x})$, applied to the withdrawals.

The cumulative probability of survival at x + 1 is the product of the conditional probabilities

$$p_{ox} = \prod_{u=0}^x p_u$$

with the error

$$S(p_{ox}) = \sqrt{p_x^2 \sum_{u=0}^{x-1} \frac{1}{p_u^2} \cdot s_{p_u}^2}$$

Expected mortality risks

The expected risk of death in the interval [x, x + 1] was calculated from the general mortality risk in Sweden, q_{ij} . This is the probability of death during year i for a person of age j. For a person entering the study in year b and at age a, the risk series will be

$$q'_1 = (1 - w_t) q_{b, a} + w_t q_{b+1, a}$$

$$q'_2 = (1 - w_t) q_{b+1, a} + w_t q_{b+2, a+1}$$

etc,

where q'_1 = the risk of death in the interval [0, 1]. For practical purposes it was assumed that all entries to the study occurred on 1 July of the respective years and that this was the date of birth of all entrants. Thus, $w_t = 1/2$.

For each time interval the expected risk was calculated as the arithmetic mean of the individual q 's. For withdrawals the weight $1/2$ was used both in the numerator and in the denominator.

The life-table method was also used to study differences between the index and the reference or standard population in regard to the risk of contracting tuberculosis. The population risk matrices for tuberculosis were constructed from data in the annual statistics of the Swedish Tuberculosis Index (6).

Median survival time

When differences in survival times are considered, mean values are useful. Accurate means, however, require knowledge of the complete survival experience of all studied cases. For this reason, we calcu-

lated the median survival time (3). Fig B-4 shows the median survival time as the point on the graph of cumulative survival where the probability of survival is 50 %.

In fig B-4 the 50 % level of observed survival is at a years. The corresponding intercept for expected survival is at b years. The difference thus is b — a. The graph of observed survival was based on a sample and therefore was subject to random variation. To make allowance for this, a conservative interval was constructed, setting two limits for the graph of cumulated observed survival probability. These limits were estimated to lie at ± 2 standard errors from the curve, and the interval estimate (b — d) — (b — c) of the difference was produced. The true statistical confidence of such an interval is not known, but presumably it is > 95 %.

In subgroups of the study population in which the 50 % survival probability could not be measured, due to short follow-up time, the estimates were based on 75 % levels.

Competing risks

Mortality during a follow-up study may have various causes. These causes (risks) are competing (2, 3). For instance, a man may have a malignant disease which is expected to be fatal soon, but he may be killed in a traffic accident. Cancer will not be recorded as the cause of death. Similarly, a man with silicosis who dies at an early age from cardiovascular disease is no longer at risk of contracting tuberculosis in later life.

Competing risks are of particular interest in follow-up studies of mortality from occupational disease. The question is how far mortality risk could be reduced by elimination of a cause of death that is closely related to occupational factors. Survival obviously would not be guaranteed, since there are also nonoccupational risks. If, for instance, in interval $x, x + 1$ cause r is eliminated, the result will apparently be increased mortality rates from other causes in the studied population.

Fig B-5 illustrates the effect of eliminating a mortality risk factor. The sym-

bols are as follows:

q_x = probability of death in interval $[x, x + 1]$,

$p_x = 1 - q_x$ = probability of surviving interval $[x, x + 1]$,

$q_{x,r}$ = net probability of death in interval $[x, x + 1]$ after the elimination of cause r, which implies the net risk of death from any cause other than r.

$q_{x,r}$ can be estimated from the formula:

$$q_{x,r} = 1 - p_x (D_x - D_{xr}) / D_x.$$

In this formula

D_x = number of deaths in interval $[x, x + 1]$ and

D_{xr} = number of deaths from cause r in interval $[x, x + 1]$.

The relative importance of mortality factor r, expressed as an upper limit of the potential gain in terms of reduced mortality risk, can be estimated from the formula:

$$\text{reduction in death risk (\%)} = 100 \cdot \frac{q_x - q_{x,r}}{q_x} \%$$

Fig B-4. Median survival time. (a = observed, b = expected, c—d = conservative interval for observed survival)

Fig B-5. Schematic illustration of influence on mortality of eliminating a causal factor (r).

This estimate of reduction in the probability of death is hypothetical. It is, moreover, based on the assumption that the eradication of factor *r* in the occupational environment results in zero exposure to that factor. This assumption of course is valid only for factors strictly confined to that environment.

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Appendix C

Tables and figures referred to, but not included, in the text

Section II

Table 6. Distribution of silicosis cases according to age at silicosis manifestation — Mining.

Silicosis manifest	Age (a)										Total	Weighted arithmetic mean
	21—30		31—40		41—50		51—60		≥ 61			
	N	%	N	%	N	%	N	%	N	%		
1931—1950	12	2.0	84	14.2	195	33.0	200	33.9	99	16.8	590	50.4
1951—1960	7	2.5	38	13.8	93	33.7	95	34.4	43	15.6	276	50.2
1961—1975			8	2.9	63	22.8	150	54.3	55	19.9	276	54.6

Table 7. Distribution of silicosis cases according to duration of dust exposure preceding silicosis manifestation — Mining.

Silicosis manifest	Prediagnostic exposure (a)											Total
	1—5	6—10	11—15	16—20	21—25	26—30	31—35	36—40	41—45	46—50	≥ 51	
1931—1950	18	53	78	115	101	87	64	41	25	6	2	590
1951—1960	17	39	34	57	38	37	24	20	6	4	—	276
1961—1975	—	15	27	36	51	54	39	28	22	3	1	276

Table 8. Distribution of silicosis cases in the age groups ≤ 50 and ≥ 51 a: Prediagnostic duration of dust exposure — Mining.

Silicosis manifest	Age ≤ 50 a			Age ≥ 51 a			Total	Weighted arithmetic mean
	Number of cases	%	Mean exposure (a)	Number of cases	%	Mean exposure (a)		
1931—1950	291	49.3	16.7	299	50.7	28.6	590	22.7
1951—1960	138	50.0	15.0	138	50.0	26.7	276	22.8
1961—1975	71	25.7	16.3	205	74.3	29.1	276	25.8

Table 9. Distribution of silicosis cases in the age groups ≤ 50 and ≥ 51 a: Mean latency time — Mining.

Silicosis manifest	Age ≤ 50 a			Age ≥ 51 a		
	Number of cases	Mean (a)	SD	Number of cases	Mean (a)	SD
1931—1950	291	18.1	7.2	299	33.4	9.9
1951—1960	138	17.0	7.4	138	32.9	10.0
1961—1975	71	21.6	6.1	205	33.4	9.2

Table 10. Distribution of silicosis cases according to age at silicosis manifestation — Steel industry.

Silicosis manifest	Age (a)					Total	Weighted arithmetic mean
	21—30	31—40	41—50	51—60	≥ 61		
1931—1950	10	70	153	127	51	411	48.9
1951—1960		23	65	63	28	179	50.8
1961—1975		7	42	70	38	157	54.4

Table 11. Distribution of silicosis cases according to duration of dust exposure preceding silicosis manifestation — Steel industry.

Silicosis manifest	Prediagnostic exposure (a)											Total
	1—5	6—10	11—15	16—20	21—25	26—30	31—35	36—40	41—45	46—50	≥ 51	
1931—1950	14	55	87	55	48	53	47	23	7	7	5	401 ^a
1951—1960	8	40	31	29	23	11	14	12	7	2	—	177 ^b
1961—1975	2	21	34	27	37	13	8	9	5	1	—	157

^a In ten additional cases the duration of prediagnostic exposure could not be ascertained.

^b In two additional cases the duration of prediagnostic exposure could not be ascertained.

Table 12. Distribution of silicosis cases in the age groups ≤ 50 and ≥ 51 a: Prediagnostic duration of dust exposure — Steel industry.

Silicosis manifest	Age ≤ 50 a		Age ≥ 51 a		Total	Weighted arithmetic mean
	Number of cases	Mean exposure (a)	Number of cases	Mean exposure (a)		
1931—1950	233	16.6	168	27.6	401	21.2 ^a
1951—1960	88	15.6	89	23.0	177	19.3
1961—1975	49	17.9	108	21.4	157	20.3

^a In ten additional cases the prediagnostic duration of exposure was unknown.

Table 13. Distribution of silicosis cases in the age groups ≤ 50 and ≥ 51 a: Mean latency time — Steel industry.

Silicosis manifest	Age ≤ 50 a			Age ≥ 51 a		
	Number of cases	Mean (a)	SD	Number of cases	Mean (a)	SD
1931—1950	233	17.8	8.1	178	30.0	11.3
1951—1960	88	18.7	8.5	90	28.0	12.9 ^a
1961—1975	49	19.7	7.3	108	26.2	10.9

^a In one case the age at commencement of exposure was unknown.

Table 14. Distribution of silicosis cases according to age at silicosis manifestation — Iron industry.

Silicosis manifest	Age (a)					Total	Weighted arithmetic mean
	21—30	31—40	41—50	51—60	≥ 61		
1931—1950	1	25	83	100	66	275	53.0
1951—1960	1	12	65	119	69	266	54.6
1961—1975		9	59	166	104	338	56.2

Table 15. Distribution of silicosis cases according to duration of dust exposure preceding silicosis manifestation — Iron industry.

Silicosis manifest	Prediagnostic exposure (a)											Total
	1—5	6—10	11—15	16—20	21—25	26—30	31—35	36—40	41—45	46—50	≥ 51	
1931—1950	1	9	25	27	40	42	46	35	18	24	8	275
1951—1960	—	11	18	28	32	39	49	36	34	11	7	265 ^a
1961—1975	4	16	23	48	47	53	44	43	40	13	7	338

^a In one case the duration of exposure could not be ascertained.

Table 16. Distribution of silicosis cases in the age groups ≤ 50 and ≥ 51 a: Prediagnostic duration of dust exposure — Iron industry.

Silicosis manifest	Age ≤ 50 a			Age ≥ 51 a			Total	Weighted arithmetic mean
	Number of cases	%	Mean exposure (a)	Number of cases	%	Mean exposure (a)		
1931—1950	109	39.6	21.1	166	60.4	34.9	275	29.4
1951—1960	78	26.7	21.4	187	70.6	33.9	265	30.5
1961—1975	68	20.1	21.1	270	79.9	30.5	338	28.6

Table 17. Distribution of silicosis cases in the age groups ≤ 50 and ≥ 51 a: Mean latency time — Iron industry.

Silicosis manifest	Age ≤ 50 a			Age ≥ 51 a		
	Number of cases	Mean (a)	SD	Number of cases	Mean (a)	SD
1931—1950	109	22.5	7.5	166	36.7	10.2
1951—1960	78	22.2	7.3	188	35.8	9.8
1961—1975	68	22.4	8.3	270	33.8	11.0

Table 18. Distribution of silicosis cases according to age at silicosis manifestation — Ceramic industry.

Silicosis manifest	Age (a)					Total	Weighted arithmetic mean
	21—30	31—40	41—50	51—60	≥ 61		
1931—1950	7	68	88	64	35	262	47.5
1951—1960	2	17	30	26	10	85	48.4
1961—1975	—	4	19	38	21	82	54.7

Table 19. Distribution of silicosis cases according to duration of dust exposure preceding silicosis manifestation — Ceramic industry.

Silicosis manifest	Prediagnostic exposure (a)											Total
	≤ 5	6—10	11—15	16—20	21—25	26—30	31—35	36—40	41—45	46—50	≥ 51	
1931—1950	1	7	35	32	66	50	28	22	16	5	—	262
1951—1960	3	5	12	16	13	10	12	9	2	1	2	85
1961—1975	—	9	9	6	16	12	11	8	3	8	—	82

Table 20. Distribution of silicosis cases in the age groups ≤ 50 and ≥ 51 a: Prediagnostic duration of dust exposure — Ceramic industry.

Silicosis manifest	Age ≤ 50 a			Age ≥ 51 a			Total	Weighted arithmetic mean
	Number of cases	%	Mean exposure (a)	Number of cases	%	Mean exposure (a)		
1931—1950	163	62.2	21.7	99	37.8	31.9	262	25.6
1951—1960	49	57.6	20.0	36	42.4	29.7	85	24.1
1961—1975	23	28.0	14.5	59	72.0	28.8	82	26.5

Table 21. Distribution of silicosis cases in the age groups ≤ 50 and ≥ 51 a: Mean latency time — Ceramic industry.

Silicosis manifest	Age ≤ 50 a			Age ≥ 51 a		
	Number of cases	Mean (a)	SD	Number of cases	Mean (a)	SD
1931—1950	163	23.5	6.9	99	33.6	8.3
1951—1960	49	24.3	8.2	36	34.5	11.6
1961—1975	23	22.9	8.6	59	34.5	12.0

Table 23. Mean age at silicosis manifestation and mean duration of preceding dust exposure — Mining.

Silicosis manifest	Silicosis stage								
	I			II			III		
	Number of cases	Exposure (a)	Age (a)	Number of cases	Exposure (a)	Age (a)	Number of cases	Exposure (a)	Age (a)
1931—1950	421	21.5	48.4	106	25.4	54.0	63	37.4	57.1
1951—1960	241	20.5	49.3	27	19.7	55.6	8	23.0	57.8
1961—1975	257	26.1	54.2	17	33.2	58.8	2	38.0	65.0

Table 25. Mean age at silicosis manifestation and mean duration of preceding dust exposure — Steel industry.^a

Silicosis manifest	Silicosis stage								
	I			II			III		
	Number of cases	Exposure (a)	Age (a)	Number of cases	Exposure (a)	Age (a)	Number of cases	Exposure (a)	Age (a)
1931—1950	289	22.7	47.2	68	23.5	51.8	44	25.2	54.6
1951—1960	154	19.4	50.0	22	19.1	56.2	1	24.0	44.0
1961—1975	153	20.3	54.0	4	19.3	60.1	—		

^a Cases in which the duration of exposure could not be confidently ascertained were excluded.

Table 27. Mean age at silicosis manifestation and mean duration of preceding dust exposure — Iron industry.

Silicosis manifest	Silicosis stage								
	I			II			III		
	Number of cases	Exposure (a)	Age (a)	Number of cases	Exposure (a)	Age (a)	Number of cases	Exposure (a)	Age (a)
1931—1950	244	29.2	52.3	23	34.7	58.1	8	33.0	56.8
1951—1960	248 ^a	29.6	54.3	16	38.6	58.6	1	38.0	65.0
1961—1975	309	29.1	55.7	28	30.5	61.9	1	18.0	35.0

^a One case was excluded as duration of exposure could not be confidently ascertained.

Table 29. Mean age at silicosis manifestation and mean duration of preceding dust exposure — Ceramic industry.

Silicosis manifest	Silicosis stage								
	I			II			III		
	Number of cases	Exposure (a)	Age (a)	Number of cases	Exposure (a)	Age (a)	Number of cases	Exposure (a)	Age (a)
1931—1950	157	23.4	45.4	72	29.3	50.1	33	27.4	51.8
1951—1960	68	23.4	42.2	17	26.5	54.3	1	8.0	55.0
1961—1975	76	26.8	54.8	5	26.0	53.5	1		

Section III

Table 30. Silicosis with (+) and without (—) progression according to mean age and mean duration of dust exposure at time of first positive radiograph — Mining.

Silicosis manifest	Progression +					Progression —				
	Number of cases	Mean age ^a (a)	SD	Mean exposure ^a (a)	SD	Number of cases	Mean age ^a (a)	SD	Mean exposure ^a (a)	SD
1931—1950	159	46.6	10.0	20.8	10.0	206	49.0	9.6	21.3	10.3
1951—1975	149	50.4	8.3	21.3	10.2	232	51.8	9.3	23.2	10.5

^a Weighted arithmetic means based on 5-a intervals.

Table 31. Silicosis with (+) and without (—) progression according to mean latency time — Mining.

Silicosis manifest	Progression +			Progression —		
	Number of cases	Latency time ^a (a)	SD	Number of cases	Latency time ^a (a)	SD
1931—1950	159	22.4	10.6	206	24.5	11.7
1951—1975	149	24.5	10.5	232	27.0	10.6

^a Weighted arithmetic means based on 5-a intervals.

Table 32. Silicosis with (+) and without (—) progression according to mean age and mean duration of dust exposure at time of first positive radiograph — Steel industry.

Silicosis manifest	Progression +					Progression —				
	Number of cases	Mean age ^a (a)	SD	Mean exposure ^a (a)	SD	Number of cases	Mean age ^a (a)	SD	Mean exposure ^a (a)	SD
1931—1950	166	46.0	8.9	18.3	10.0	79	49.9	10.0	23.1	11.5
1951—1975	66	49.6	8.6	18.0	11.1	173	51.9	8.4	20.1	10.3

^a Weighted arithmetic means based on 5-a intervals.

Table 33. Silicosis with (+) and without (—) progression according to mean latency time — Steel industry.

Silicosis manifest	Progression +			Progression —		
	Number of cases	Latency time ^a (a)	SD	Number of cases	Latency time ^a (a)	SD
1931—1950	166	19.5	9.9	79	24.3	11.5
1951—1975	66	21.0	11.0	173	22.4	10.7

^a Weighted arithmetic means based on 5-a intervals.

Table 35. Progression of stage I silicosis in the 20-a follow-up. (Mean age at start of observation = 48.7 a, SD = 9.9) — Quarrying, tunneling, and granite work.

Observation time (a)	Silicosis stage			Cumulated deaths	Nonattendance at follow-up
	I	II	III		
0	65				
5	42	17	4	2	—
10	27	20	8	10	—
15	14	19	14	18	—
20	2	8	6	48	1

Table 36. Progression of stage I silicosis in the 20-a follow-up. (Mean age at start of observation = 47.8 a, SD = 9.2) — Steel industry.

Observation time (a)	Silicosis stage			Cumulated deaths	Nonattendance at follow-up ^a
	I	II	III		
0	241				
5	178	51	6	6	—
10	121	75	23	19	3
15	74	82	33	40	9 (3)
20	13	34	24	146	12 (5)

^a Numbers within parentheses give number of cases with known progression among nonattenders.

Table 37. Progression of stage I silicosis in the 20-a follow-up. (Mean age at start of observation = 52.4 a, SD = 9.0) — Iron industry.

Observation time (a)	Silicosis stage			Cumulated deaths	Nonattendance at follow-up ^a
	I	II	III		
0	219				
5	203	8	—	7	1
10	161	18	2	35	2
15	110	22	4	66	14
20	25	15	3	134	25 (3)

^a Numbers within parentheses give number of cases with known progression among nonattenders.

Table 38. Progression of stage I silicosis in the 20-a follow-up. (Mean age at start of observation = 45.4 a, SD = 9.5) — Ceramic industry.

Observation time (a)	Silicosis stage			Cumulated deaths	Nonattendance at follow-up ^a
	I	II	III		
0	131				
5	115	10	—	5	1
10	83	33	6	6	2
15	43	48	13	18	6 (2)
20	5	18	23	72	4 (2)

^a Numbers within parentheses give number of cases with known progression among nonattenders.

Fig 2. Quarrying, tunneling, and granite work — Progression of stage I silicosis. Height of columns shows number of survivors after 5-a intervals. [□ number without progression, ▨ number with radiographic progression (form I to II or III), ● cumulative percentage of known radiographic progression including progression in subjects who died in the 5-a interval and progression in nonattenders]

Fig 3. Steel industry — Progression of stage I silicosis. Height of columns shows number of survivors after 5-a intervals. [□ number without progression, ▨ number with radiographic progression (from I to II or III), ● cumulative percentage of known radiographic progression including progression in subjects who died in the 5-a interval and progression in nonattenders]

Fig 4. Iron foundries — Progression of stage I silicosis. Height of columns shows number of survivors after 5-a intervals. [□ number without progression, ▨ number with radiographic progression (from I to II or III), ● cumulative percentage of known radiographic progression including progression in subjects who died in the 5-a interval and progression in nonattenders]

Fig 5. Ceramic industry — Progression of stage I silicosis. Height of columns shows number of survivors after 5-a intervals. [□ number without progression, ▨ number with radiographic progression (from I to II or III), ● cumulative percentage of known radiographic progression including progression in subjects who died in the 5-a interval and progression in nonattenders]

Section IV

Table 40. Silicosis plus tuberculosis (TB), distribution according to stage of silicosis on first positive radiograph — Mining, quarrying, tunneling, etc.

Silicosis manifest	Stage I			Stage II			Stage III			Total		
	Total	TB	%	Total	TB	%	Total	TB	%	Total	TB	%
1931—1950	506	62	12.3	155	34	21.9	102	23	22.5	763	119	15.6
1951—1960	385	43	11.2	52	7	13.5	9	2	22.2	446	52	11.7
1961—1975	634	29	4.5	90	11	12.2	21	1	4.8	745	41	5.5

Table 41. Silicosis plus tuberculosis (TB), distribution according to stage of silicosis on first positive radiograph — Steel industry.

Silicosis manifest	Stage I			Stage II			Stage III			Total		
	Total	TB	%	Total	TB	%	Total	TB	%	Total	TB	%
1931—1950	292	42	14.4	72	14	19.4	47	5	10.9	411	61	14.9
1951—1960	155	7	4.5	23	1	4.3	1	—	—	179	8	4.5
1961—1975	153	1	0.7	4	—	—	—	—	—	157	1	0.6

Table 42. Silicosis plus tuberculosis (TB), distribution according to stage of silicosis on first positive radiograph — Iron industry.

Silicosis manifest	Stage I			Stage II			Stage III			Total		
	Total	TB	%	Total	TB	%	Total	TB	%	Total	TB	%
1931—1950	281	15	5.3	31	4	12.9	12	1	8.3	324	20	6.2
1951—1960	271	20	7.3	19	3	15.8	1	—	—	291	23	7.9
1961—1975	356	4	1.3	29	1	3.6	2	—	—	387	5	1.3

Table 43. Silicosis plus tuberculosis (TB), distribution according to stage of silicosis on first positive radiograph — Ceramic industry.

Silicosis manifest	Stage I			Stage II			Stage III			Total		
	Total	TB	%	Total	TB	%	Total	TB	%	Total	TB	%
1931—1950	157	17	10.8	72	17	23.6	33	4	12.1	262	38	14.5
1951—1960	68	2	2.9	17	—	—	1	—	—	86	2	3.5
1961—1975	76	2	2.6	5	—	—	1	—	—	82	2	2.4

Table 44. Silicosis with tuberculosis (TB), distribution according to stage of silicosis on diagnostic radiograph — Other industries or multiple-category dust exposure.^a

Silicosis manifest	Stage I			Stage II			Stage III			Total		
	Total	TB	%	Total	TB	%	Total	TB	%	Total	TB	%
1931—1950	154	22	14.3	63	12	19.0	27	3	11.1	244	37	15.2
1951—1960	95	9	9.5	29	1	3.4	7	—	—	131	10	7.6
1961—1975	181	5	2.8	16	2	12.5	7	1	14.3	204	8	3.9

^a Does not include six cases without known tuberculosis and with silicosis stage unclassified at diagnosis.

Table 47. Analysis of tuberculosis risk according to numbers of observed persons and observed cases with tuberculosis — Mining.

Observation time (a)	Number entering ^a	Observed number of tuberculosis cases
0— 1	499	6
1— 2	417	3
2— 3	414	1
3— 4	397	2
4— 5	382	3
5— 6	338	1
6— 7	320	1
7— 8	290	0
8— 9	267	1
9—10	241	0
10—11	216	1
11—12	189	2
12—13	167	1
13—14	153	0
14—15	130	0
15—16	118	0
16—17	100	0
17—18	76	1
18—19	57	0
19—20	37	0

^a Date of entry = year of established silicosis diagnosis.

Table 48. Analysis of tuberculosis risk, calculated observed and expected risks; observed risk values smoothed (3-point moving average) — Mining.

Observation time (a)	Observed tuberculosis risk (%)	Expected tuberculosis risk (%)	Ratio observed/expected
0— 1	—	0.103	—
1— 2	0.756	0.099	7.61
2— 3	0.493	0.095	5.17
3— 4	0.529	0.092	5.73
4— 5	0.549	0.090	6.11
5— 6	0.705	0.087	8.10
6— 7	0.428	0.086	5.00
7— 8	0.458	0.085	5.40
8— 9	0.277	0.085	5.20
9—10	0.441	0.085	5.20
10—11	0.682	0.083	8.23
11—12	0.951	0.081	11.78
12—13	0.787	0.080	9.90
13—14	0.415	0.079	5.28
14—15	0	0.078	0
15—16	0	0.078	0
16—17	0.498	0.077	6.46
17—18	0.498	0.077	6.49
18—19	0.498	0.079	6.32
19—20	—	0.082	—

Table 49. Analysis of tuberculosis risk according to the numbers of observed persons and observed cases with tuberculosis — Iron industry.

Observation time (a)	Number entering ^a	Observed number of tuberculosis cases
0— 1	524	6
1— 2	442	3
2— 3	439	1
3— 4	422	2
4— 5	407	3
5— 6	363	1
6— 7	343	1
7— 8	310	0
8— 9	284	1
9—10	256	0
10—11	229	1
11—12	201	2
12—13	178	1
13—14	163	0
14—15	139	0
15—16	124	0
16—17	104	0
17—18	80	1
18—19	60	0
19—20	40	0

^a Date of entry = year of established silicosis diagnosis.

Table 50. Analysis of tuberculosis risk, calculated observed and expected risks; observed risk values smoothed (3-point moving average) — Iron industry.

Observation time (a)	Observed tuberculosis risk (%)	Expected tuberculosis risk (%)	Ratio observed/expected
0— 1	—	0.103	—
1— 2	0.715	0.099	7.21
2— 3	0.464	0.095	4.89
3— 4	0.497	0.092	5.39
4— 5	0.513	0.089	5.74
5— 6	0.658	0.087	7.59
6— 7	0.400	0.085	4.68
7— 8	0.429	0.085	5.07
8— 9	0.261	0.085	3.02
9—10	0.415	0.084	4.94
10—11	0.642	0.082	7.80
11—12	0.894	0.080	11.13
12—13	0.793	0.079	9.36
13—14	0.389	0.078	4.99
14—15	0	0.078	0
15—16	0	0.078	0
16—17	0.473	0.077	6.14
17—18	0.473	0.077	6.14
18—19	0.473	0.079	6.01
19—20	—	0.082	—

MINING

CASES ADMITTED 1951-70 N=499

Fig 7. Observed and expected freedom from tuberculosis in silicosis contracted in mining.

IRON FOUNDRIES

CASES ADMITTED 1951-70 N=524

Fig 8. Observed and expected freedom from tuberculosis in silicosis contracted in the iron industry.

STEEL FOUNDRIES

CASES ADMITTED 1951-70 N=165

Fig 9. Observed and expected freedom from tuberculosis in silicosis contracted in the steel industry.

QUARRYING
CASES ADMITTED 1951-70 N=189

Fig 10. Observed and expected freedom from tuberculosis in silicosis contracted in stone quarrying.

Section V

Table 54. Observed and expected mortality according to stage of silicosis and presence (TB+) or absence (TB-) of suspected or verified tuberculosis — Mining, quarrying, and tunneling.

Subcategory	Years	Sample size at end of period	Deaths		Ratio observed/ expected
			Observed (N)	Expected (N)	
1931—1948	1931—1940	36	5	1.6	3.1 *
Stage I	1941—1950	108	17	14.7	1.2
TB—	1951—1960	108	37	30.4	1.2
	1961—1969	108	29	27.7	1.0
1931—1948	1931—1940	56	29	3.2	9.1 **
Stage II—III	1941—1950	119	40	18.4	2.2 **
TB—	1951—1960	119	29	34.9	0.8
	1961—1969	119	15	31.8	0.5
1931—1948 ^a	1931—1940	11	3	0.3	10.0 **
Stage I	1941—1950	47	17	5.0	3.4 **
TB+	1951—1960	47	14	10.9	1.3
	1961—1969	47	8	12.2	0.7
1931—1948 ^b	1931—1940	35	19	1.6	11.9 **
Stage II—III	1941—1950	93	39	10.1	3.9 **
TB+	1951—1960	93	22	23.5	0.9
	1961—1969	93	7	25.9	0.3 **
1949—1969	1949—1950	72	2	0.9	2.2
Stage I	1951—1960	275	41	24.8	1.7 **
TB—	1961—1969	492	80	63.6	1.3 *

^a One postmortem notification, cause of death tuberculosis.

^b Seven postmortem notifications, cause of death tuberculosis.

* $p < 0.05$, ** $p < 0.01$.

Table 55. Observed and expected mortality according to stage of silicosis and presence (TB+) or absence (TB-) of suspected or verified tuberculosis — Steel and iron industries.

Subcategory	Years	Sample size at end of period	Deaths		Ratio observed/ expected
			Observed (N)	Expected (N)	
1931—1948	1931—1940	34	2	1.0	2.0
Stage I	1941—1950	177	24	16.7	1.4
TB—	1951—1960	177	53	39.5	1.3
	1961—1969	177	46	44.2	1.0
1931—1948	1931—1940	47	13	2.3	5.7 **
Stage II—III	1941—1950	98	27	15.6	1.7 *
TB—	1951—1960	98	31	28.2	1.1
	1961—1969	98	22	25.3	0.9
1931—1948 ^a	1931—1940	18	3	0.4	7.5 *
Stage I	1941—1950	63	4	6.5	0.6
TB+	1951—1960	63	28	14.8	1.9 **
	1961—1969	63	16	15.7	1.0
1931—1948 ^a	1931—1940	24	8	1.3	6.2 **
Stage II—III	1941—1950	58	28	8.1	3.5 **
TB+	1951—1960	58	14	15.4	0.9
	1961—1969	58	4	15.8	0.3 **
1949—1969	1949—1950	108	0	1.2	0
Stage I	1951—1960	416	47	37.5	1.3
TB—	1961—1969	697	99	99.7	1.0

^a One postmortem notification, cause of death tuberculosis.

* $p < 0.05$, ** $p < 0.01$.

Table 56. Observed and expected mortality according to stage of silicosis and presence (TB+) or absence (TB-) of suspected or verified tuberculosis — Ceramic industry.

Subcategory	Years	Sample size at end of period	Deaths		Ratio observed/ expected
			Observed (N)	Expected (N)	
1931—1948	1931—1940	177	14	5.5	2.5 **
Stages I—III	1941—1950	139	41	19.8	2.1 **
TB—	1951—1960	139	29	30.8	0.9
	1961—1969	139	23	30.5	0.8
1931—1948	1931—1940	16	6	1.1	5.5 **
Stages I—III	1941—1950	41	16	4.6	3.5 **
TB+	1951—1960	41	7	8.5	0.8
	1961—1969	41	4	9.5	0.4
1949—1969	1949—1950	34	0	0.4	0
Stages I—III	1951—1960	133	17	11.9	1.4
TB+ and TB—	1961—1969	204	47	28.4	1.7 **

* $p < 0.05$, ** $p < 0.01$.

Table 58. Median reduction (a) of life expectancy (Estimated confidence intervals within parentheses) — Iron and steel industries.

Cases reported	Stage of silicosis	Tuberculosis a		
		+	—	+ and —
1931—1948	I	4.3 (2.5— 8.0)	3.2 (2.0— 5.0)	
	II—III	15.1 (14.0—16.5)	9.2 (6.5—11.0)	
1949—1969	I		1.0 (—1.3— 2.7) ^b	3.2 (2.5—4.5)
	II—III		2.7 (0.7— 5.4) ^b	5.2 (2.5—7.0)

a + = with tuberculosis, — = without tuberculosis.

b 75 % survival intercept.

MINING QUARRYING AND TUNNELING

SIL > 2 TBC+ 1931-1948 N=93

Fig 15. Mining, quarrying, and tunneling — Cumulated mortality in the part of the study population admitted to the study in 1931—1948: Silicosis (SIL) stage II or III and suspected or verified tuberculosis (TBC). [Number (N) = 93]

STEEL AND IRON INDUSTRY

SIL < 2 TBC- 1931-1948 N=177

Fig 16. Steel and iron industry — Cumulated mortality in the part of the study population admitted to the study in 1931—1948: Silicosis (SIL) stage I and no tuberculosis (TBC). [Number (N) = 177]

STEEL AND IRON INDUSTRY

CASES ADMITTED 1939-1948

Fig 18. Steel and iron industries — Cumulative overall mortality rate (SMR) and time after admission in the part of the study population admitted in 1931–1948: Smoothed curves (3-point moving average) for the stages of silicosis (SIL) in combination with or without suspected or verified tuberculosis (TBC).

CASES ADMITTED 1931-1948

A: STEEL AND IRON B: MINING ETC

Fig 19. Mining, quarrying, and tunneling versus steel and iron industries — Cumulative overall mortality rate (SMR) and time after admission for the part of the study population admitted in 1931–1948: Smoothed curves (3-point moving average) for stages of silicosis (SIL) with suspected or verified tuberculosis (TBC).

CASES ADMITTED 1931-1948

A: STEEL AND IRON B: MINING ETC

Fig 20. Mining, quarrying, and tunneling versus steel and iron industries — Cumulative overall mortality rate (SMR) and time after admission for the part of the study population admitted in 1931–1948: Smoothed curves (3-point moving averages) for the stages of silicosis (SIL) without tuberculosis (TBC).

Section IX

Table 68. Silicosis in persons less than 40 a of age, reported in 1971—1977 — All occupational categories.

Case number	Year of birth	Silicosis notified	Silicosis manifest	Exposure began	Exposure ended	Years of exposure ^a	Conditions of exposure
<i>Mining, quarrying, tunneling, etc</i>							
1	1935	1972	1972	1956	1976	16	Engine driver below ground, 1956; driller below ground, 1956—1964; loading, etc, below ground, 1964—1976
2	1936	1977	1975	1965	1975	10	Loader, stone-crushing plant, 1965—1968; driller in quarry; 1965—1975
<i>Steel and iron industries</i>							
3	1932	1976	1971	1954 1974	1958 —	about 2.5	Crushing-plant worker, 1955—1957; fettler in iron foundry 6 months; steel molding-shop worker since 1974
4	1939	1973	1972	1955	1972	17	Furnace mason, steel works, 1955—1972
5	1934	1975	1972	1957	1974	15	Crane operator in steel molding shop, 1957—1969; molder, 1969—1974
6	1934	1971	1971	1948	1971	23	Iron molder, 1948—1971
<i>Other industries</i>							
7	1937	1975	1974	1954	1969	15	Packaging of scouring powder (high content of fine-particled quartz)
8	1935	1974	1973	1957 1968	1958 —	8	Truck-driver in foundry molding shop, 1957—1958; castings repairer (near fettling processes), 1968—1969; driver of excavator and caterpillar tractor

^a Preceding manifestation of silicosis.

Table 69. Silicosis after 10 a of exposure to dust; cases reported in 1971—1977 — All occupational categories.

Case number	Year of birth	Silicosis notified	Silicosis manifest	Exposure began	Exposure ended	Years of exposure ^a	Conditions of exposure
<i>Mining, quarrying, tunneling, etc</i>							
9	1919	1974	1971	1963	1974	8	Tunneling, drilling, mainly in quarries, 1963—1974
10	1908	1976	1976	1953	1957	4	Quarrying, near crushing plant, 1953—1957
<i>Steel and iron industries</i>							
11	1931	1974	1974	1965	1973	8	Furnace mason, steel foundry, 1965—1973
12	1915	1974	1971	1956	1962	6	Steel fettler, 1956—1962
13	1919	1976	1976	1959	1963	4	Steel molder, occasional furnace work, 1959—1963
14	1922	1971	1971	1947	1955	8	Iron foundry ladler, 1947—1955
15	1920	1971	1971	1945 1956	1949 1957	5	Iron foundry ladler, 1945—1948, 1956—1957; Quarrying, drilling, 1949
16	1913	1971	1971	1965	1971	6	Box annealing furnace operator, 1965—1971
<i>Ceramic industry</i>							
17	1914	1974	1974	1935	1941	6	Pottery forming-shop worker, 1935—1941
<i>Other industries</i>							
18	1910	1972	1972	1954	1961	7	Sandblaster (bathtub making), 1950—1954
19	1921	1975	1971	1950	1954	4	Sandblaster (metal building components), 1950—1954

^a Preceding manifestation of silicosis.