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Incidence of the Pneumoconioses in the United Kingdom General Population between 1997 and 2008

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For editorial comment see p. 191

Key Words

Asbestosis · Epidemiology · Incidence · Interstitial lung disease · Pneumoconioses

Abstract

Background: The incidence of the pneumoconioses in the UK is primarily estimated using occupational-based registries and disability pension schemes. These sources indicate a downward trend in the incidence of the pneumoconioses from 1995 onwards. There are no previously published general population-based observational studies quantifying the incidence of the pneumoconioses in the UK. ***Objectives:*** The aim of this study was to investigate the incidence of the pneumoconioses in the UK general population between 1997 and 2008 using data from the General Practice Research Database (GPRD). ***Methods:*** Data from the UK-based GPRD were used to estimate the incidence of pneumoconioses over a 12-year period (1997–2008). Crude incidence rates for asbestosis and non-asbestos-related pneumoconioses were stratified by gender, age group and calendar period, and rate ratios were adjusted using Poisson regression. ***Results:*** The majority of cases was diagnosed with asbestosis, and the overall, crude incidence density for this pneumoconiosis during the 12-year study period was 2.7 (95% confidence interval 2.5–2.9) per 100,000 person-years. The incidence increased progressively during the period 1997–2005 and then decreased slightly during the period 2006–2008, even after controlling for the strong effect of an ageing UK population. The non-asbestos-related pneumoconioses, in contrast to asbestosis, showed a progressive reduction in incidence from 2003 onwards. ***Conclusions:*** This study demonstrates that the pneumoconioses remain an important public health issue and, furthermore, documents an overall increase in asbestosis incidence in the UK between 1997 and 2008.

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Introduction

The pneumoconioses are a group of interstitial lung diseases caused by inhalation and retention of inorganic dusts. These diseases generally evolve over decades of occupational exposure to mineral dusts and are characterized by the formation of nodular, fibrotic changes to the

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lung parenchyma [1]. In the absence of extremely high exposure to such particles, there is a relatively long time lag between first-time occupational exposure and clinically detectable disease. This is reflected in the fact that the majority of incident cases is detected in retired workers [2].

The primary data sources used to investigate the incidence and prevalence of the pneumoconioses in the UK are the Department for Work and Pensions Industrial Injuries and Disablement Benefit scheme and The Health and Occupation Reporting (THOR) network. During the period 1998–2008, the number of new cases of pneumoconiosis reported by chest and occupational physicians within the THOR network fluctuated between 108 and 321 cases per year [3]. Trends in incidence of the pneumoconioses from both sources are difficult to interpret due to varying definitions of disease states and changing eligibility criteria for compensation benefits over time. Additionally, it is known that case numbers have been affected by fluctuations in the numbers and reporting habits of participating health care practitioners over time [3]. Data from THOR and the Industrial Injuries and Disablement Benefit scheme suggest a downward trend in the annual number of new pneumoconiosis cases from 2005 onward [3, 4]. However, as is the case when using data from disease registries, measures of disease frequency from both of these sources may be underestimated due to case ascertainment and selection biases.

The UK General Practice Research Database (GPRD) contains anonymized electronic patient records collected from routine general practice. The geographic, age and gender distributions of the GPRD population have been shown to be representative of the UK general population [5]. The aim of this study was to investigate the incidence of the pneumoconioses in the UK general population between 1997 and 2008 using data from the GPRD.

Materials and Methods

Data Source

The GPRD has been described elsewhere in detail [5], but briefly, it is a large and well-validated UK-based database that was established in June 1987. The GPRD encompasses nearly 5 million patients who are or were enrolled with selected general practitioners throughout the UK, covering approximately 50 million patient-years of follow-up. The participating general practitioners have been trained to record medical information in a standard manner and to provide anonymized records to the GPRD Group within the Medicines and Healthcare products Regulatory Agency (MHRA), the UK's medicines and devices regulator. The recorded data include demographic information, medical diagnoses and drug prescriptions. The Boston Collaborative Drug Surveillance Program and its associated researchers at the Basel Pharmacoepidemiology Unit (University of Basel) use a large subset of data from the GPRD; data from general practices that have failed to report consistent numbers of drug prescriptions and diagnostic codes over time have been removed from the Boston Collaborative Drug Surveillance Program GPRD [6]. Data from the GPRD have been used in previous studies involving interstitial lung disease, and use of the data for respiratory epidemiology has been validated [7, 8]. The GPRD is managed by the MHRA, and the present study protocol was approved by the Independent Scientific Advisory Committee for MHRA database research.

Study Population

We identified in the GPRD all patients with a first-time diagnosis of a pneumoconiosis between 1 January 1997 and 31 December 2008. Diagnoses in the GPRD are coded using the Read coding system, and our list of pneumoconiosis diagnoses included any Read code explicitly mentioning pneumoconiosis, silicosis, silica/silicate pneumoconiosis, silicotic fibrosis, talc pneumoconiosis, siderosis, berylliosis, stannosis, asbestosis and coal worker's pneumoconiosis. Read codes without explicit mention of a pneumoconiosis, such as 'Pneumopathy due to inhalation of other dust NOS', were not used. In order to increase the likelihood of capturing incident rather than prevalent cases, we excluded patients with less than 3 years of active recording history prior to the date of the first pneumoconiosis diagnosis.

Data Analysis

Person-time denominators for the calculations of incidence density were derived from the GPRD. For each calendar period, only those patients in the GPRD who were alive, actively registered and had at least 3 years of recorded history contributed person-time to the denominators. Incident cases were grouped into four age groups ($<$60, 60–69, 70–79 and $\geq$80 years), and calendar time was divided into four 3-year periods (1997–1999, 2000–2002, 2003–2005 and 2006–2008). Crude incidence rates were calculated for asbestos-related pneumoconiosis (i.e. asbestosis) and for non-asbestos-related pneumoconioses separately. The crude rates were stratified by gender, age group and calendar period, with exact 95% confidence intervals (CIs) being calculated from the Poisson distribution. In order to model disease incidence and estimate incidence rate ratios, fixed-effects Poisson multivariate regression was used. We accounted for overdispersion in all regression models using the quasi-likelihood approach, that is, by fixing the scaled deviance at unity and introducing a dispersion parameter into the model which adjusted the standard errors of the parameter estimates. All analyses were performed with the statistical software SAS (release 9.2, SAS Institute, Inc., Cary, N.C., USA).

Results

A total of 1,070 patients with an incident diagnosis of any type of pneumoconiosis were identified during the period 1997–2008, which included 840 cases of asbestosis (87.5%) and 230 cases of non-asbestos-related pneumoconioses. The frequency distribution of incident diagnoses is shown in table 1.

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Table 1. Frequency of incident pneumoconiosis diagnoses (n = 1,070)

Diagnosis code	Frequency	Percentage
Asbestos-related (total)	840	100.00%
Asbestosis	826	98.33%
Asbestosis NOS	14	1.67%
Non-asbestos-related (total)	230	100.00%
Pneumoconioses NOS	173	75.22%
Coal worker's pneumoconiosis	17	7.39%
Silica and silicate pneumoconiosis	7	3.04%
Talc pneumoconiosis	1	0.43%
Simple silicosis	5	2.17%
Massive silicotic fibrosis	1	0.43%
Silica pneumoconiosis NOS	6	2.61%
Pneumoconiosis due to other inorganic dust	1	0.43%
Chronic beryllium disease	1	0.43%
Siderosis	8	3.48%
Stannosis	1	0.43%
Pneumoconiosis due to inorganic dust NOS	9	3.91%

NOS = Not otherwise specified.

Table 2. Crude incidence rates for non-asbestos-related pneumoconiosis diagnoses (n = 230)

	Cases n	Total person-years	IR	95% CI (exact Poisson)
Overall	230	31,165,672.41	0.74	0.65–0.84
Gender				
Female	13	16,421,082	0.08	0.42–1.35
Male	217	14,744,590	1.47	1.28–1.68
Age group				
<60 years	39	23,309,579.76	0.17	0.12–0.23
60–69 years	50	3,474,347.44	1.44	1.07–1.90
70–79 years	78	2,689,162.52	2.90	2.29–3.62
>80 years	63	1,692,582.69	3.72	2.86–4.76
Calendar period				
1997–1999	55	6,696,773.29	0.82	0.62–1.07
2000–2002	83	7,646,978.15	1.09	0.87–1.35
2003–2005	57	8,232,635.23	0.69	0.52–0.90
2006–2008	35	8,589,285.74	0.41	0.28–0.57

IR = Incidence rate (density) per 100,000 person-years.

Table 3. Poisson regression modelling of incidence for the non-asbestos-related pneumoconioses (n = 230)

	IRR	95% CI	LRT p value
Gender			<0.0001
Female (ref.)	1.00	–	
Male	16.15	7.26–35.96	
Age group			0.2197[1]
<60 years (ref.)	1.00	–	
60–69 years	1.43	0.79–2.61	
70–79 years	2.29	1.32–3.97	
>80 years	2.12	1.20–3.76	
Calendar period			0.1017[1]
1997–1999 (ref.)	1.00	–	
2000–2002	1.30	0.80–2.12	
2003–2005	0.82	0.48–1.39	
2006–2008	0.49	0.27–0.90	

IRR = Incidence rate ratio; LRT = likelihood ratio test.
[1] p for trend.

Non-Asbestos-Related Pneumoconiosis

The mean age (SD) at diagnosis was 70 years (13.9), and 217 cases (94%) were male. Females were younger than males at the time of first diagnosis [57.8 years (21.8) vs. 71.1 years (13.0), respectively], and the majority of diagnosed cases was 70 years of age and over.

Between 1997 and 2008, the overall incidence density rate of the non-asbestos-related pneumonconioses was 0.74 (95% CI 0.65–0.84) per 100,000 person-years, and the rate in males was approximately 16 times the rate in females. The incidence density increased during the period 1997–2002 [from 0.82 (95% CI 0.62–1.07) during 1997–1999 to 1.09 (95% CI 0.87–1.35) during 2000–2002 per 100,000 person-years] and decreased progressively thereafter from 2003 to 2008. Crude incidence rates by calendar period for the non-asbestos-related pneumoconioses are shown in table 2. Poisson regression modelling of incidence rates for the non-asbestos-related pneumoconioses yielded no statistically significant heterogeneity of rates over time (p for trend = 0.1017; table 3).

Asbestosis

The mean age (SD) at diagnosis was 69 years (11.4), and 784 cases (93%) were male. Females were younger than

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Table 4. Crude incidence rates for asbestosis (n = 840)

	Cases n	Total person-years	IR	95% CI (exact Poisson)
Overall	840	31,165,672.41	2.70	2.52–2.88
Gender				
Female	56	16,421,082	0.34	0.26–0.44
Male	784	14,744,590	5.32	4.95–5.70
Age group				
<60 years	108	23,309,579.76	0.46	0.38–0.56
60–69 years	280	3,474,347.44	8.06	7.14–9.06
70–79 years	321	2,689,162.52	11.94	10.67–13.32
>80 years	131	1,692,582.69	7.74	6.47–9.18
Calendar period				
1997–1999	110	6,696,773.29	1.64	1.35–1.98
2000–2002	195	7,646,978.15	2.55	2.20–2.93
2003–2005	266	8,232,635.23	3.23	2.85–3.64
2006–2008	269	8,589,285.74	3.13	2.77–3.53

IR = Incidence rate (density) per 100,000 person-years.

Table 5. Poisson regression modelling of incidence for asbestosis (n = 840)

	IRR	95% CI	LRT p value
Gender			<0.0001
Female (ref.)	1.00	–	
Male	17.87	11.36–28.09	
Age groups			<0.0001[1]
<60 years (ref.)	1.00	–	
60–69 years	17.56	12.13–25.42	
70–79 years	28.14	19.57–40.48	
>80 years	22.58	14.77–34.55	
Calendar period			0.0238[1]
1997–1999 (ref.)	1.00	–	
2000–2002	1.54	1.04–2.27	
2003–2005	1.91	1.32–2.76	
2006–2008	1.76	1.22–2.55	

IRR = Incidence rate ratio; LRT = likelihood ratio test.
[1] p for trend.

males at the time of first diagnosis [61.8 years (22.1) vs. 70.0 years (10.0), respectively], and the majority of cases were diagnosed in the age group 60–79 years.

Between 1997 and 2008, the overall incidence density rate of asbestosis was 2.7 (95% CI 2.5–2.9) per 100,000 person-years, and the rate in males was approximately 18 times the rate in females. The analysis of crude incidence density for asbestosis yielded progressively increasing rates during the period 1997–2005 [from 1.6 (95% CI 1.4–2.0) during the period 1997–1999 to 3.2 (95% CI 2.9–3.6) per 100,000 person-years], with a slight downward blip during the period 2006–2008. Crude incidence rates by calendar period for asbestosis are shown in table 4.

In the asbestosis cohort, there was evidence of statistically significant heterogeneity of incidence rate ratios over time (p = 0.024), after adjusting for the effects of age and gender. The incidence density increased from the period 1997–1999 to 2000–2002 by a factor of 1.5. Using the same baseline period, the incidence nearly doubled during the period 2003–2005 (incidence rate ratio 1.9). Incidence rate ratios for asbestosis are shown in table 5.

To provide an additional estimate of the average annual increase in the incidence of asbestosis, we repeated our analyses fitting year as a continuous variable. After controlling for the effects of age and gender, we estimated the average annual increase in the incidence of asbestosis to be 6% during the 12-year study period (incidence rate ratio 1.064, 95% CI 1.04–1.09; $p < 0.0001$).

Discussion

In this large, general population-based cohort of pneumoconiosis patients, the overall incidence rate for asbestos-related and non-asbestos-related pneumoconioses was approximately 3 and 1 per 100,000 UK population per year, respectively, suggesting that each year during the period 1997–2008 there were approximately 1,800 new cases of asbestosis and 600 new cases of non-asbestos-related pneumoconiosis in the UK. The incidence of asbestosis increased progressively during the period 1997–2005 and then decreased slightly during the period 2006–2008, while the incidence of non-asbestos-related pneumoconiosis showed progressive decline from 2003 to 2008. Incidence rates were substantially higher in men than in women, likely reflecting the industrial-occupational nature of the disease etiology.

The majority of cases in our cohort (approx. 80%) was diagnosed with asbestosis, and the observed increase in incidence during the study period would seem to be in line with the estimation that the peak global incidence

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of asbestos-related disease is expected to occur 30–40 years after the period of peak asbestos usage (i.e. the 1960s and 1970s) [9]. This is further reflected in a report by the UK Health and Safety Executive which predicts increasing rates of mesothelioma-related mortality until around 2015, after which rates are expected to decline [10, 11]. Therefore, the projected, overall annual estimate of approximately 1,800 new cases of asbestosis will probably not continue far into the future. From the analysis of non-asbestos-related pneumoconiosis, it can be seen that the incidence of pneumoconiosis due to causes other than asbestos has declined over time, except for a relative rise in incidence noted during the period 2000–2002.

A potential weakness of the present study could be the validity of pneumoconiosis diagnoses, as we have not tested these directly. However, the validity of many disease diagnoses has been assessed in the UK GPRD and consistently found to be high [6, 12, 13]. Furthermore, other researchers have reported a high validity of the diagnosis of idiopathic pulmonary fibrosis, another interstitial lung disease, in the GPRD [14]. Moreover, it is unlikely that general practitioners would record a pneumoconiosis diagnosis without confirmation by specialist referral. Therefore, we expect the diagnoses used for the present study to have high specificity.

On the other hand, the authors are aware that the sensitivity of the diagnoses used may not be as high as the specificity. Two main reasons for this are the potential for misclassification of true pneumoconiosis cases into less specific disease diagnosis categories and the possibility of having missed less severe cases. For instance, we did not use less specific respiratory-related diagnosis codes such as 'lung disease due to other external agents NOS' or 'other external agent causing respiratory condition' in our case selection algorithm. Overall, there were 649 cases with such an unspecific diagnosis which we subsequently reviewed in more detail. Only 10 of them (1.5%) had some evidence of a pneumoconiosis diagnosis at a later point in time in their patient record, which makes substantial misclassification unlikely. It is also possible that under-diagnosis on the part of general practitioners (and hence non-referral to specialists) is present in this study or that less severe cases have simply escaped detection by general practitioners and/or specialists. Unfortunately, the degree to which both possibilities might have occurred cannot be readily quantified in retrospective, observational database research. Thus, it is possible that we did not have available for analysis all true pneumoconiosis cases from the given sampling frame, which means that we may have underestimated the true incidence density of the pneumoconioses in the UK. Furthermore, we cannot be certain that all newly identified cases were indeed incident cases. However, we excluded cases with less than 3 years of recorded medical history in order to minimize the possibility of including prevalent cases in our incidence numerators, so we expect the occurrence of such error to be minimal. To the extent that this error occurred, it will have resulted in a small overestimation of the incidence density rates. Moreover, the percentage of cases disregarded due to the above exclusion criterion was 11% of the total number of cases identified during the defined study window (2,071 vs. 1,843), and the percentages of cases excluded across the study time periods remained relatively stable at 10–12%. This would support the conclusion that the results presented here are not influenced by fluctuations in the frequency of recorded diagnoses of pneumoconiosis.

Despite the potential limitations mentioned above, this study determined the best current estimates of incidence density for inorganic dust pneumoconioses in the UK general population. We have been unable to find any other general population-based cohort analyses in the UK with which to compare our results. Also, comparison of our incidence estimates to those published from the Surveillance of Work-Related and Occupational Respiratory Disease – as part of the THOR network – is difficult. Recent analyses of Surveillance of Work-Related and Occupational Respiratory Disease data yielded an average annual decline in pneumoconiosis incidence of –0.8% (95% CI –4.8 to +3.3) over the period 1999–2006 [15]. However, with respect to age and gender demography, some similarities can be noted; nearly all cases were males (>97%) with a mean age between 66 and 70 years, and approximately 55% of all cases were observed in the 60–75 years age range. Nevertheless, we acknowledge that employed adults generally seek medical care less often than the unemployed, which may partially explain the relatively higher frequency of first-time pneumoconiosis diagnosis during retirement age. Finally, it is acknowledged by the Health Safety Executive – the national independent watchdog for work-related health, safety and illness in Great Britain – that incidence figures may be substantially underestimated by THOR 'since the scheme will only include those cases that are serious enough to be seen by a chest consultant, or that occur in individuals with access to occupational physicians' [3]. By the same token, we acknowledge the potential for under- or overrecognition of the pneumoconioses in our

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study, should the catchment area of contributing practices not be representative of the industrial mix in the UK. However, we expect the potential for such bias to be minimal, since the geographical distribution of the participating practices in the GPRD is representative of the UK population [5].

The observed upward trend in asbestosis incidence in the present study should be interpreted with caution. We cannot tell whether this finding reflects a real increase or whether it is, at least to a certain extent, attributable to secular trends. Firstly, during the study window, major political changes in the NHS occurred, including more than a doubling in funding under the auspices of the New Deal [16]. This investment in national health care was in part used to boost the capacity of NHS services and to modernize facilities, thus potentially increasing the likelihood of detecting new cases of pneumoconioses. Secondly, a certain proportion of incident cases in this time period may have been due to increased recognition of interstitial lung diseases in general via the increased use of high-resolution computed tomography. Thirdly, following publication of both the British Thoracic Society's guideline on diagnosis and care of diffuse parenchymal lung disease in 1999 [17] and the American Thoracic Society/European Respiratory Society consensus statements on idiopathic interstitial pneumonia in 2002 [18], a greater appreciation of the value of precise diagnosis according to defined criteria will likely have occurred, not to mention increased patient expectations. Finally, the observed increase in non-asbestos-related pneumoconiosis from 1997–1999 to 2000–2002 could be partly attributable to a publicity campaign by the Department for Work and Pensions around 2002 inviting people whose claims had been wrongly disallowed between 1994 and 1999 to re-claim [3]. For the above reasons, an increase in case ascertainment may have played a role in the observed increase in the incidence density of asbestosis. However, the observed numerical decline in the incidence of the non-asbestos-related pneumoconioses from 2003 onwards would argue against increased case ascertainment being responsible for the progressive upward trend in the incidence of asbestosis until the end of 2005.

Conclusions

This study makes use of data from a well-validated database which has consistently been found to be representative of the UK general population. To our knowledge, this is the first general population-based observational study quantifying incidence rates of pneumoconioses in the UK. Further research using data sources such as the UK GPRD is needed to assess future trends in pneumoconiosis incidence and to confirm the assumption that asbestos-related diseases are truly beginning to decline in incidence as a consequence of health and safety measures enacted at the end of the last century.

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Financial Disclosure and Conflicts of Interest

Dr. Rosenberg is an employee of Actelion Ltd. Dr. Amar is an employee of Novartis Pharma AG.

The remaining authors have no conflicts of interest to disclose.

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