Relation between exposure to respirable silica dust
and silicosis in a tungsten mine in China

D Pang, S C Fu, G C Yang

Abstract
To estimate the quantitative relation between exposure to respirable silica dust and risk of an
attack of silicosis, 1151 workers exposed to silica dust and employed from 1958 to 1987 in a
tungsten mine in China were investigated. The
results showed that the ratio of respirable silica dust concentration to total silica dust
concentration was 0·529. Then, the total silica dust concentration in historical surveillance
and monitoring data was converted to respirable silica dust concentration. The free silica
content in respirable dust determined by x ray
diffraction averaged 24·7%. Multiple logistic
regression was used for the dichotomous
dependent variables (presence or absence of
silicosis). The independent variables in the
multiple logistic regression with presence of
silicosis as the dependent variable were age
when first exposed, tuberculosis (presence or
absence), and cumulative exposure to respirable
silica dust. The partial regression co-
efficient of individual cumulative exposure
was estimated as 0·079. It implied a positive
association between exposure to respirable
silica dust and risk of an attack of silicosis. The
exposure limit for respirable silica dust was
estimated as 0·24 mg/m³ under given
conditions.

Respirable dust is defined as that reaching the alveoli
and is assumed to be associated more with
pneumoconiosis than is total dust. As a result, health
standards for respirable dust have been introduced
by many countries—for example, America, Japan,
Australia—in the context of prevention of
pneumoconiosis. We are presently conducting a
retrospective epidemiological study of selected work-
ers from a tungsten mine in China to establish the
quantitative relation between exposure to respirable
dust and silicosis. The aim of the present study is to
provide epidemiological evidence for setting and
revision of the exposure limit for respirable silica
dust.

Subjects and methods
SUBJECTS
The study took advantage of both historical
surveillance and monitoring data collected in the
period from 1958 to 1987.

Men eligible for inclusion in the study were
underground miners hired from 1958 to 1987
inclusive, without exposure to dust before 1958.
Those who were previously exposed to other kinds of
dusts, such as tungsten, were excluded. The criteria
for inclusion were met by 1151 men.

EXPOSURE AND ESTIMATION
Information about the mine and the estimation of
individual exposures by extrapolation based on fixed
point membrane filter measurement has been
published elsewhere. The total and respirable silica
dust concentrations at workplaces of major job titles
were measured. The free silica content in respirable
dust was determined by x ray diffraction.

STATISTICAL METHODS
The relation between respirable dust concentration
and total dust concentration was assessed by a linear
regression model. The relation between silicosis and
cumulative respirable silica dust concentration was
determined by multiple logistic linear regression
with the logist procedure of the SAS package.¹

QUALITY CONTROL MEASURES
Quality control measures were instituted to ensure
the quality of information collected. All data were
checked when completed to establish that all items
had been attempted. When completed all dat: were
checked by computer procedures to ensure no logis-
tic mistakes. Then all data were checked manually to
ensure no entry errors. An airflow calibrating meter
was used to calibrate the flow of samplers.

Results
RESPIRABLE AND TOTAL DUST CONCENTRATIONS
The figure shows the correlation between respirable
and total dust concentrations at workplaces in the
underground mine. A linear trend was found. Table
Relation between exposure to respirable silica dust and silicosis in a tungsten mine in China

Regression of respirable and total dust concentrations.

1 shows the $t$ test statistics of regression. The regression coefficient was $0.529$ ($p < 0.001$). It implied that the respirable dust concentration correlated with the total dust concentration. According to the regression coefficient, the ratio of respirable dust concentration to total dust concentration was $0.529$. Therefore the respirable dust concentration was estimated based on total dust concentration in historical surveillance and monitoring data.

FREE SILICA CONTENT OF RESPIRABLE DUST
The arithmetic mean of six samples was $24.7\%$.

RESPIRABLE SILICA DUST CONCENTRATION AND SILICOSIS
Table 2 presents the results of the multiple logistic regression with presence of silicosis as the outcome. The partial regression coefficient between the cumulative exposure to respirable silica dust and silicosis was statistically significant ($\beta = 0.079$, $p < 0.05$) after allowing for the age of first exposure and tuberculosis (TB). The probability of developing silicosis correlated positively with cumulative exposure to respirable silica dust. The multiple logistic regression model can be expressed as follows:

$$p = 1/(1 + \exp(-((-7.331) + 0.079 \cdot \text{CUMCON} + 0.108 \cdot \text{AGE1} + 1.59 \cdot \text{TB})))$$  \hspace{1cm} (1)

$$\text{CUMCON} = C \times U$$  \hspace{1cm} (2)

where:
- $p$ = The probability level of developing silicosis.
- CUMCON = Cumulative exposure to respirable silica dust in units of mg years/m$^3$.
- AGE1 = Age of first exposure (y).
- TB = Tuberculosis before silicosis developed.
- C = Respirable concentration (mg/m$^3$).
- U = Duration of exposure (y).

If the sensitivity of the model was $95\%$, a probability level for classification of silicosis of $0.8\%$ was derived. An exposure limit of $0.24$ mg/m$^3$ was estimated according to equations (1) and (2) when given a probability level for classification of silicosis as $0.8\%$, a duration of exposure of 30 years, and TB as 0 (those without TB).

**Discussion**
Silicosis is a chronic occupational disease with a long period of latency and it is important to establish its relation to respirable dust. To determine respirable dust concentrations it is necessary to convert total dust concentration in historical surveillance and monitoring data to respirable dust concentration by making experiments on respirable and total dust concentrations. Our results showed that the ratio of respirable to total dust concentrations was $0.529$. It was assumed that changes in the ratio were small over various periods because working conditions and dust protective measures were stable. Estimates of respirable dust concentration were therefore derived from the total dust concentrations by applying a conversion factor of $0.529$.

The probability of developing silicosis was positively correlated with cumulative exposure to respirable silica dust (table 2). This would be consistent with published evidence. The less the sensitivity of the regression model, the better the protection of workers from silicosis. It was accepted that the sensitivity was $95\%$. The age of first exposure was assumed to be 18 years according to the 12th clause of the Act of Silicosis of the People's Republic of China, which stated that workers were “... not eligible to participate in dust work without being more than 18 years old.” The duration of exposure was assumed as 30 years taking the duration of employment in a worker's whole life as 30-40 years. The free silica content in respirable silica dust was $24.7\%$. A respirable dust concentration of $0.24$ mg/m$^3$ was estimated based on these conditions. We conclude that the exposure limit of $0.24$ mg/m$^3$ would be safe allowing for epidemiological evidence.

**Table 1** Regression analysis of respirable and total dust concentrations

<table>
<thead>
<tr>
<th>Independent variable</th>
<th>Dependent variable</th>
<th>No of samples</th>
<th>t Value</th>
<th>p Value (one sided)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Respirable dust</td>
<td>Total dust</td>
<td>25</td>
<td>15.247</td>
<td>0.000</td>
</tr>
</tbody>
</table>

**Table 2** Linear multiple logistic regression with presence of silicosis as outcome and with age of first exposure, TB, and cumulative exposure to respirable silica dust as independent variables

<table>
<thead>
<tr>
<th>Variable</th>
<th>$\beta$ (SD)</th>
<th>$\chi^2$</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intercept</td>
<td>-7.331 (0.877)</td>
<td>69.95</td>
<td>0.0001</td>
</tr>
<tr>
<td>Cumulative concentration</td>
<td>0.079 (0.031)</td>
<td>6.38</td>
<td>0.0115</td>
</tr>
<tr>
<td>Age of first exposure</td>
<td>0.108 (0.029)</td>
<td>13.71</td>
<td>0.0002</td>
</tr>
<tr>
<td>Tuberculosis</td>
<td>1.59 (0.609)</td>
<td>8.83</td>
<td>0.0089</td>
</tr>
</tbody>
</table>

Regression of respirable and total dust concentrations.
We thank Dr M Jacobsen from the Institute of Occupational Medicine, Edinburgh, UK, Dr Mike Phillips, and Dr Xin Kai Lu, Centre for Advanced Studies, Australia for design and data processing. The cost was covered by a grant from the Ministry of Public Health of China.

Inhalation Fever: A proposed unifying term for febrile reactions to inhalation of noxious substances

Over the past several years there have been many publications that consider respiratory illnesses of farmers.1–3 Much of the interest has centred around the description of a febrile illness caused by exposure to organic dust that has been called several names including: grain fever, pulmonary mycotoxicosis, silo unloader’s syndrome, and organic dust toxic syndrome (ODTS). There is now sufficient evidence to suggest that these febrile attacks are separate from allergic alveolitis. The physiological impact of the febrile illness on the lung is minimal whereas in allergic alveolitis one sees abnormal blood gases, chest radiographs, and pulmonary function tests.4

In the modern industrial era there has been extensive writing about febrile responses to the inhalation of metal fumes and dusts such as metal fume fever among welders and foundry workers.5–6 As plastics emerged papers appeared describing febrile illnesses in those who work with complex polymers.7

The clinical picture of these inhalation ailments is similar. All have a latent period of several hours between the exposure and the onset of fevers, chills, malaise, myalgias, and chest tightness. Patients often have a leucocytosis with a left shift.8–11 There are reports of chemical pneumonitis and pulmonary oedema in some metal and plastic exposures and a single report suggesting more severe lung involvement after exposure to organic dust in an agricultural setting, but most reports suggest that little lung damage occurs.12–15

We are struck by the described similarities and believe that a generic term is best for this family of respiratory ailments. In the absence of a single aetiological agent for these very different exposures we think that the fevers and symptoms represent a common pathway in which the lung reacts to noxious substances. We suggest that they all be called inhalation fever, a term originating from 1978.16 This avoids the colourful but confusing litany of names currently in use. Henceforth we propose that organic dust toxic syndrome, all the fume fevers, spelter shakes, mill fever, Monday fever, card room fever, and brass founders ague etc, be called simply Inhalation fever.

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doi: 10.1136/oem.49.1.38

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