national population, and then age-standardised to the world standard population published in 1966.[23] These death rates were then averaged for the same period.

The amount of historical asbestos consumption was defined as the yearly average asbestos consumption per head (kg per head per year) for 1960–69 (ie, the average of data for 1960 and 1970, the 2 years for which data are available). For every country, the volume of asbestos production, import, and export (in metric tonnes) was extracted from the US Geological Survey report.[24] The definition of consumption was production plus import minus export,[24] and the resulting number was divided by the national population for that period. The period of asbestos consumption (1960–69) was chosen a priori, to allow for a disease latency of about 30–40 years between exposure and the average yearly deaths from asbestos-related diseases in 2000–04. Although variable latency periods have been reported for asbestosis morbidity, we postulated the same latency period in asbestosis patients to account for the additional period from onset of symptoms to death.

33 countries had both consumption and mortality data available for analyses. These countries accounted for 63% of all asbestos consumed worldwide in 1960–69 and for 22% of the world's population in 2000–04—84%, 74%, and 48% of the population of the Americas, Oceania, and Europe, respectively, but only 8% in Africa and 5% in Asia.

For individual asbestos-related diseases, separate linear regression analyses were done for each sex, with age-adjusted mortality rates of each asbestos-related disease as the dependent variable and historical asbestos consumption as the independent variable. National mortality rates were log-transformed to comply with the assumptions underlying the random errors in the regression model. Parameters in the regression model were estimated by the least-square method, and weighted by the size of sex-specific national populations in 2000–04. All statistical procedures were done with SAS version 8·02. Graphs were drawn with SigmaPlot version 9·01. A p value less than 0·05 was deemed statistically significant.

Role of the funding source
The sponsor of the study had no role in study design, data collection, data analysis, data interpretation, or writing of the report. The corresponding author had full access to the data in the study and had final responsibility for the decision to submit for publication.

Results
The figure shows scatter plots of national data for the diseases associated with asbestos (male sex had the highest adjusted R^2 value and was therefore chosen for presentation in the figure). A positive linear relation between historical asbestos consumption and log-transformed death rates can clearly be seen. The sex-specific association of individual asbestos-related diseases was expressed as a linear regression model in the table, with the parameters intercept B_0 and slope B_1, and adjusted R^2 values. Strong linear relations are apparent for all mesothelioma in both sexes and asbestosis and peritoneal mesothelioma in men.

The relation for all mesothelioma in men (see figure A) shows high asbestos consumption corresponding to a high log-transformed mortality rate. Historical asbestos consumption was a highly significant positive predictor of all mesothelioma mortality (see table), with an adjusted R^2 value of 0·74 in men (p<0·0001). In women, asbestos consumption was a significant positive predictor of all mesothelioma mortality, with an adjusted R^2 value of 0·58 (p<0·0001). The slope (B_1) of the regression lines suggested that for an increment in asbestos consumption of 1 kg per head in a population, men had a 2·4-fold (95% CI 2·0 to 2·9) $(10^{0·382 \times 1}$

	n	Regression parameters						Adjusted R^2	p value
		B_0 (95% CI)	SE	p value	B_1 (95% CI)	SE	p value		
All mesothelioma									
Male	32	–0·135 (–0·325 to 0·055)	0·093	0·1567	0·382 (0·299 to 0·465)	0·041	<0·0001	0·738	<0·0001
Female	31	–0·326 (–0·477 to –0·175)	0·074	0·0001	0·208 (0·143 to 0·274)	0·032	<0·0001	0·578	<0·0001
Pleural mesothelioma									
Male	29	–0·408 (–0·756 to –0·059)	0·170	0·0237	0·257 (0·108 to 0·406)	0·073	0·0015	0·293	0·0015
Female	25	–0·748 (–1·108 to –0·388)	0·174	0·0003	0·123 (–0·029 to 0·275)	0·073	0·1066	0·071	0·1066
Peritoneal mesothelioma									
Male	25	–1·475 (–1·779 to –1·171)	0·147	<0·0001	0·333 (0·205 to 0·461)	0·062	<0·0001	0·539	<0·0001
Female	27	–1·190 (–1·359 to –1·021)	0·082	<0·0001	0·132 (0·061 to 0·204)	0·035	0·0008	0·345	0·0008
Asbestosis									
Male	27	–1·255 (–1·462 to –1·048)	0·100	<0·0001	0·439 (0·348 to 0·530)	0·044	<0·0001	0·789	<0·0001
Female	19	–1·513 (–1·718 to –1·307)	0·098	<0·0001	0·050 (–0·036 to 0·135)	0·041	0·2375	0·027	0·2375

n=number of countries. B_0=intercept of regression line. B_1=slope of regression line. *Regression model: $\log_{10}$(age-adjusted mortality rates of asbestos-related diseases) (deaths per million population per year)=$B_0+B_1 \times$historical asbestos consumption (kg per head per year) . †2000–04. ‡1960–69.

Table: Regression analyses* for age-adjusted mortality rates† of asbestos-related diseases versus historical asbestos consumption‡ weighted by the size of sex-specific national populations

−81−

[95% CI $10^{0.299\times1}$ to $10^{0.465\times1}$]) increase and women had a 1·6-fold (1·4 to 1·9) ($10^{0.208\times1}$ [$10^{0.143\times1}$ to $10^{0.274\times1}$]) increase in deaths from mesothelioma. The intercepts (B_0) were small at $10^{-0.135}=0.73$ (0·47 to 1·14) for men and $10^{-0.326}=0.47$ (0·33 to 0·67) for women. Death rate predicted for the mean consumption value (1·67 kg per head) was 3·18 (2·55 to 3·82) in men and 1·05 (0·56 to 1·54) (per million people per year) in women. Thus the male-to-female death rate ratio was 3·0.

Historical asbestos consumption was a significant positive predictor of pleural mesothelioma rate in men(see figure B; $R^2=0.29$, p=0·0015, $B_0=10^{-0.408}=0.39$ [0·18 to 0·87]). The slope suggested a 1·8-fold (1·3 to 2·5) ($10^{0.257\times1}$ [$10^{0.108\times1}$ to $10^{0.406\times1}$]) increase in pleural mesothelioma deaths in men per 1 kg rise in asbestos consumption in a population. However, the relation was not statistically significant (p=0·107) in women.

The relations for peritoneal mesothelioma mortality rate were positive and significant for both sexes (see figure C; $R^2=0.54$, p<0·0001, $B_0=10^{-1.475}=0.03$ [0·02 to 0·07] for men and $R^2=0.35$, p=0·0008, $B_0=10^{-1.190}=0.06$ [0·04 to 0·10] for women). The slope suggested a 2·2-fold (1·6 to 2·9) ($10^{0.333\times1}$ [$10^{0.205\times1}$ to $10^{0.461\times1}$]) and a 1·4-fold (1·2 to 1·6) ($10^{0.132\times1}$ [$10^{0.061\times1}$ to $10^{0.204\times1}$]) increase in deaths from peritoneal mesothelioma in men and women, respectively, per 1 kg incremental rise in asbestos consumption in the population. The mortality rate predicted for the mean consumption value (1·67 kg per head) was 0·12 (−0·72 to 0·96) in men and 0·11 (−0·40 to 0·61) in women (per million people per year). Thus the male-to-female death rate ratio was 1·1.

The association for asbestosis mortality rate was positive and statistically significant in men and yielded the highest adjusted R^2 in all studied diseases (see figure D; $R^2=0.79$, p<0·0001, $B_0=10^{-1.255}=0.06$ [0·03 to 0·09]). The slope showed a 2·7-fold (2·2 to 3·4) ($10^{0.439\times1}$ [$10^{0.348\times1}$ to $10^{0.530\times1}$]) increase in deaths from asbestosis in men per 1 kg incremental rise in asbestos consumption in the population. Again, the association was not significant in women.

Discussion

We recorded that recent national death rates from diseases associated with asbestos were closely related to historical asbestos consumption rates in 1960–69. The ecological association was consistently positive for all studied diseases in men. In women, significantly positive ecological relations between asbestos consumption and deaths from associated diseases were recorded for peritoneal and all mesothelioma, but not for pleural mesothelioma or asbestosis. The associations were especially strong for asbestosis in men, all mesothelioma in men, all mesothelioma in women, and peritoneal mesothelioma in men. For these categories, mortality rates increased from between 1·6-fold and 2·7-fold per 1 kg incremental rise in asbestos consumption.

For all studied asbestos-related diseases, the increase in deaths per incremental increase of asbestos consumption was higher in men than in women, with the highest increase in men for asbestosis and lowest for pleural mesothelioma. However, in women, the highest increase was for all mesothelioma and lowest was for asbestosis. The positive correlation in women was more apparent for all mesothelioma than pleural mesothelioma, which could be because there were fewer overall cases of pleural mesothelioma, and some countries had no data for specific subcategories of mesothelioma. These drawbacks could also have affected the data for pleural mesothelioma in men. The positive relation between asbestos and pleural mesothelioma was not significant in women (p=0·107). For all mesothelioma, the male-to-female death rate ratio of 3·0 for the predicted dependent value was within, but closer to the lower boundary, of the reported range of 2–10,[25,26] although the male-to-female distribution of exposure differs by population. The male-to-female death rate ratios for pleural and peritoneal mesothelioma were 3·7 (the regression model for women was non-significant) and 1·1 (the regression model for both sexes was significant), respectively, which were similar to previously reported values of 5 and 2.[27] The male-to-female rate ratio for asbestosis was 8·1. There are no reports with which to compare our results, but the results are within a plausible range.

Although our findings cannot be extrapolated beyond the data range, the regression lines had intercepts close to zero for all diseases associated with asbestos. Small amounts of historical asbestos consumption is therefore predictive of few deaths from such diseases.

Development of a model of the ecological relation between historical asbestos consumption and frequency of lung cancer is needed to allow estimation of the proportion of lung cancer caused by asbestos in a population. The public-health importance of asbestos-induced lung cancer has been underappreciated[28] because scientific knowledge is restricted to ratios of asbestos-induced lung cancer (ie, excess cases of lung cancer) and mesothelioma reported by epidemiological studies of occupationally exposed populations, ranging from 1–2 to 30–40, with 2 as the most frequently cited figure.[29–32] No comparable information exists for such ratios in the general population. Within the framework of an ecological study, however, the assertion of lung cancer is not straightforward since more than 80% of lung cancers in men and 45% of lung cancers in women are attributable to smoking.[33] Comparable data for historical tobacco consumption that could be feasibly analysed are few. Thus the ecological relation between national death rates from lung cancer and historical asbestos consumption, with adjustment for the concurrent effect of smoking, needs further investigation.The independent variable was defined as the national asbestos consumption volume divided by the size of the national population, and thus represented the general amount of asbestos consumed by a person for individual countries. As such, sex, age,

occupation, and other attributes of a population, or consumed fibre types (eg, amphiboles, chrysotile) were not accounted for. We also do not know whether the consumed amounts of asbestos equate to exposure amounts. However, this index has been widely used to describe the asbestos situation nationally and regionally,[11,18,34] including in ecological studies cited earlier.[12,18,19] Such previous use of this index allowed us to regard it as a reasonable surrogate for general exposure amounts in a national population.

The dependent variables were the national death rates from diseases associated with asbestos for the most recent period with available data. The most recent update to WHO mortality database included 2004 data and provided sex-specific data, which were incorporated into our analyses. Generally, risks and death rates of diseases associated with asbestos are much higher in men, probably because men have had higher exposure to asbestos, usually through their occupations.[35] On the other hand, biological responses to asbestos exposure could differ by sex. Thus regression models for all asbestos-related diseases were done separately on the basis of sex-specific mortality rates. However, the exposure variable did not account for sex difference because those data could not be apportioned between sexes.

We extracted data for consumption and mortality from a single, authoritative, and widely-used global database, which probably enhanced the comparability of data. Nevertheless, the quality of data from developing countries is probably poorer than those from developed countries, because under-recognition of asbestos-associated diseases and absence of statistics about asbestos consumption are more likely in such countries,[34,36] leading to a negative bias. Data points near the origin, representing developing countries, tended to fall below the regression line, suggesting that such a bias existed. The incorporation of data from developing countries contributed to a good representation of the situation worldwide, with inclusion of data close to the origin (low consumption and low mortality), allowing a valid interpretation of the intercept.

We used data for 1960–69, when consumption and production of asbestos is known to have increased greatly in most continents.[24] The resulting time difference until death in 2000–04 is 37·5 years on average (range 31–44 years). These timescales correspond to the higher consensus values typically reported for latency periods of diseases related to asbestos. Our choice of consumption years can be justified because our analyses assessed deaths rather than incidence (ie, additional time to death is needed after disease manifestation). Moreover, similar relations were generally maintained when data from other but close consumption periods were used, (eg, the adjusted R^2 range was 0·16–0·83 when a consumption period of 1950–79 was applied to the models).

Our study has several strengths, including that it was a comparative assessment of a wide range of diseases associated with asbestos, we used the largest possible number of countries in the analyses, allowed for a sufficient latency time, applied age-adjustment to mortality rates for valid comparisons, and weighted by the size of sex-specific national populations in the regression model. A limitation of our study, however, is that we were restricted to analyses that included only countries for which both consumption and mortality data were available, leading to the preclusion of populous countries such as China, India, and Russia. Furthermore, lung cancer, as an important contributor in the total burden of asbestos-related diseases, warrants a separate analytical framework. Thus, all findings should be cautiously interpreted within the constraints of an ecological study.

In conclusion, this ecological study incorporating country-specific data revealed clear and plausible positive relations between amounts of historical asbestos consumption and deaths from diseases associated with asbestos. These relations were most apparent in men, but were also apparent in women. Historical asbestos consumption alone explained the bulk of the variance in subsequent death rates from such diseases. Our results lend support to the notion that all countries should move towards eliminating the use of asbestos.

References
1   Selikoff IJ, Churg J, Hammond EC. Asbestos exposure and neoplasia. *JAMA* 1964; 188: 22–26.
2   Selikoff IJ. Asbestos disease-1990–2020: the risks of asbestos risk assessment. *Toxicol Ind Health* 1991; 7: 117–27.
3   Peto J, Hodgson JT, Matthews FE, Jones JR. Continuing increase in mesothelioma mortality in Britain. *Lancet* 1995; 345: 535–39.
4   Peto J, Decarli A, La Vecchia C, Levi F, Negri E. The European mesothelioma epidemic. *Br J Cancer* 1999; 79: 666–72.
5   Pelucchi C, Malvezzi M, La Vecchia C, Levi F, Decarli A, Negri E. The Mesothelioma epidemic in Western Europe: an update. *Br J Cancer* 2004; 90: 1022–24.
6   Montanaro F, Bray F, Gennaro V, et al. Pleural mesothelioma incidence in Europe: evidence of some deceleration in the increasing trends. *Cancer Causes Control* 2003; 14: 791–803.
7   Banaei A, Auvert B, Goldberg M, Gueguen A, Luce D, Goldberg S. Future trends in mortality of French men from mesothelioma. *Occup Environ Med* 2000; 57: 488–94.
8   Hodgson JT, McElvenny DM, Darnton AJ, Price MJ, Peto J. The expected burden of mesothelioma mortality in Great Britain from 2002 to 2050. *Br J Cancer* 2005; 92: 587–93.

−83−

9   Marinaccio A, Montanaro F, Mastrantonio M, et al. Predictions of mortality from pleural mesothelioma in Italy: a model based on asbestos consumption figures supports results from age-period-cohort models. *Int J Cancer* 2005; **115:** 142–47.

10   Murayama T, Takahashi K, Natori Y, Kurumatani N. Estimation of future mortality from pleural malignant mesothelioma in Japan based on an age-cohort model. *Am J Ind Med* 2006; **49:** 1–7.

11   LaDou J. The asbestos cancer epidemic. *Environ Health Perspect* 2004; **112:** 285–90.

12   Tossavainen A. Global use of asbestos and the incidence of mesothelioma. *Int J Occup Environ Health* 2004; **10:** 22–25.

13   Anonymous. Asbestos, asbestosis, and cancer: the Helsinki criteria for diagnosis and attribution. *Scand J Work Environ Health* 1997; **23:** 311–16.

14   International Labour Organization. Asbestos: the iron grip of latency. International Labour Organization, 2006. http://www.ilo.org/public/english/bureau/inf/features/06/asbestos.htm (accessed Jan 7, 2006).

15   International Labour Organization . ILO adopts new measures on occupational safety and health, the employment relationship, asbestos. International Labour Organization, 2006. http://www.ilo.org/public/english/bureau/inf/pr/2006/34.htm (accessed Jun 16, 2006).

16   Goldberg M, Banaei A, Goldberg S, Auvert B, Luce D, Gueguen A. Past occupational exposure to asbestos among men in France. *Scand J Work Environ Health* 2000; **26:** 52–61.

17   Harington JS, McGlashan ND. South African asbestos: production, exports, and destinations, 1959–1993. *Am J Ind Med* 1998; **33:** 321–26.

18   Takahashi K, Huuskonen M, Tossavainen A, Higashi T, Okubo T, Rantanen J. Ecological relationship between mesothelioma incidence/mortality and asbestos consumption in ten Western countries and Japan. *J Occup Health* 1999; **41:** 8–11.

19   Nurminen M, Karjalainen A, Takahashi K. Estimating the induction period of pleural mesothelioma from aggregate data on asbestos consumption. *J Occup Environ Med* 2003; **45:** 1107–15.

20   World Health Organization (WHO). WHO Mortality Database: World Health Organization Statistical Information System (WHOSIS). http://www3.who.int/whosis/menu.cfm (accessed Mar 30, 2006).

21   United States Census Bureau (USCB). International Data Base: International Programs Center, Population Division, United States Census Bureau, 2005. http://www.census.gov/ipc/www/idbnew.html (accessed Nov 16, 2005).

22   UN. World Population Prospects: The 2004 Revision Population Database: Population Division, United Nations, 2004.

23   Doll R, Payne PM, Waterhouse JAH. Cancer incidence in five continents. A technical report. Springer-Verlag, Berlin: International Union Against Cancer (IUCC), 1966.

24   Virta RL. Worldwide asbestos supply and consumption trends from 1900 to 2000. Open-file report 03–83: United States Geological Survey (USGS), 2003.

25   McDonald JC, McDonald AD. The epidemiology of mesothelioma in historical context. *Eur Respir J* 1996; **9:** 1932–42.

26   Hillerdal G. Mesothelioma: cases associated with non-occupational and low dose exposures. *Occup Environ Med* 1999; **56:** 505–13.

27   Jones RD, Smith DM, Thomas PG. Mesothelioma in Great Britain in 1968–1983. *Scand J Work Environ Health* 1988; **14:** 145–52.

28   Hessel PA, Gamble JF, McDonald JC. Asbestos, asbestosis, and lung cancer: a critical assessment of the epidemiological evidence. *Thorax* 2005; **60:** 433–36.

29   McDonald JC, McDonald AD. Epidemiology of mesothelioma. Boca Raton, FL: CRC Press, Inc, 1991.

30   Liddell FD, McDonald AD, McDonald JC. The 1891–1920 birth cohort of Quebec chrysotile miners and millers: development from 1904 and mortality to 1992. *Ann Occup Hyg* 1997; **41:** 13–36.

31   Albin M, Magnani C, Krstev S, Rapiti E, Shefer I. Asbestos and cancer: An overview of current trends in Europe. *Environ Health Perspect* 1999; **107** (suppl 2): 289–98.

32   Darnton AJ, McElvenny DM, Hodgson JT. Estimating the number of asbestos-related lung cancer deaths in Great Britain from 1980 to 2000. *Ann Occup Hyg* 2006; **50:** 29–38.

33   Anonymous. The World Cancer Report–the major findings. *Cent Eur J Public Health* 2003; **11:** 177–79.

34   Takahashi K, Karjalainen A. A cross-country comparative overview of the asbestos situation in ten Asian countries. *Int J Occup Environ Health* 2003; **9:** 244–48.

35   Price B, Ware A. Mesothelioma trends in the United States: an update based on Surveillance, Epidemiology, and End Results Program data for 1973 through 2003. *Am J Epidemiol* 2004; **159:** 107–12.

36   Kazan-Allen L. Asbestos and mesothelioma: worldwide trends. *Lung Cancer* 2005; **49** (suppl 1): S3–S8.

《資料》

開発中の石綿ばく露評価システムの仕様

システムの目的

主に医療現場において石綿曝露が疑われる受診者に対する医学的な問診場面を想定し、医師等の医療従事者が、受診者から得られる職歴情報を元に石綿曝露歴の評価・診断を支援するためのソフトウェア。

データベースの概念

```
┌──────────────────────────────┐        ┌──────────────────────────────────┐
│  ╭────────────╮              │     ┌─辞書情報─┐                          │
│  ( 個人属性情報 )             │─────│          │                          │
│  ╰────────────╯              │     │ ┌───────┬───────┐    産業          │
│      │                        │     │ │用語コード│ 用語名 │    職業          │
│   ╭─問診─────────╮            │     │ └───────┴───────┘    石綿作業内容     │
│   (              )            │     │        ┌───────┐    商品（製品）      │
│   ( 職歴情報等*   )            │     │        │ 説明文  │                    │
│   (╭──────────╮ )            │     │        └───────┘                    │
│   (( 石綿情報   ))  ┌─────────┐│     │        ┌───────┐                    │
│   (╰──────────╯ )  │ 問診履歴  ││     │        │ 出典情報 │                    │
│   ╰──────────────╯  └─────────┘│     │        └───────┘                    │
│                     ┌─────────┐│     │        ┌───────┐                    │
│                     │リスクスコア計算││     │        │ 関連用語 │                    │
│  *職歴情報、居住歴情報、生活歴└─────────┘│     │        └───────┘                    │
│                              │     │ ┌──────────────┐                  │
│                              │     │ │ リスクスコア情報  │                  │
│                              │     │ └──────────────┘                  │
└──────────────────────────────┘        └──────────────────────────────────┘
```
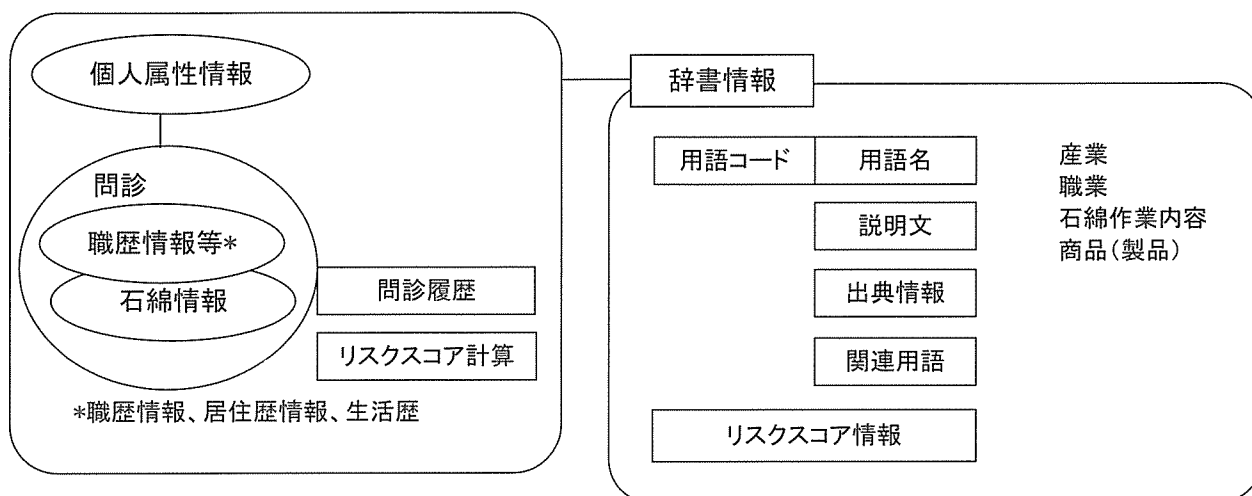
システムの特徴

・個人の問診履歴の時系列化

・リスクスコア算出

・辞書内関連用語のリレーショナル機能

システム機能構成

```
メイン
メニュー
```

個人情報入力 ─ □ 個人属性情報を登録するための画面

問診入力

- リスク計算 ─ 条件入力 ─ スコア表示

スコアの計算方法（ルール）
パーセンテージ
段階（例；10段階）
点数
……

ある日の情報でのリスク計算

- 従事期間、年代
  - 産業
  - 職業
  - 石綿作業内容
  - 商品（製品）

個人年表 問診の登録・修正機能と時系列グラフ表示。

検索 用語の検索機能。

- 生活歴 ─ □
- 居住歴 ─ □

辞書登録

- 用語登録 ─ □

用語コード	説明文
用語コード	説明文
用語コード	説明文

- 用語照会・修正 ─ □

用語コード	出典情報
用語コード	出典情報
用語コード	出典情報

用語コード	関連用語
用語コード	関連用語
用語コード	関連用語

例 バルブ
バルブ製造

→
ガスケット
シール剤
発電所
修理
メンテナンス

時期（従事時期）
2004年以降であれば
殆ど使用されていないのでリスク

用語コードで説明文、出典情報、関連用語などのリレーションを行う。
1個の用語で複数の出典情報や複数の関連用語のリレーションが可能

- 用語削除 ─ □

- リスクスコア設定 ─ □

リスク情報（スコア）

スコア	用語コード	用語コード	用語コード
スコア	用語コード		
スコア	用語コード	用語コード	

例；胸膜プラーク
⇒将来、中皮腫になるリスクが極めて高い。

辞書検索 ─ 条件入力 → 一覧表示 → 情報表示

DBのデータ構造の説明

下記各データに共通でつけられる情報 ； 登録日、登録効力、登録者、時代（年度 From-To）、更新前コード、更新日

用語コード

```
┌──┬──┬──┬──────────────┐
│  │  │  │   用語名      │      120byte（全角60文字）　全角半角混在OK
└──┴──┴──┴──────────────┘
        └─── 小分類コード　　3桁                    1byte文字テキスト（ANK）
      └───── 中分類コード　　3桁                    入力は左詰（桁の位置に意味があるため
    └─────── 大分類コード　　2桁                    システム上で補完や右詰は行わない。）
  └───────── 分野コード（産業分類、職業分類、石綿作業内容分類*、製品分類、99（定義なし）……）2桁
```

分野コード"99"はリンク用語とする。（用語そのものは分類に直接属さないが、
　　　　　　　　　　　一般表現などを吸収できるようにするためのもの。）
　　　　　　　　　例；漁船、網投げ、漁師、etc.
　　　　　　　　　　職業に関する書籍を読み込んで、使用してはどうか。
　　　　　　　　　　職業分類解説文
　　＊ 石綿作業内容分類の例；吹きつけ、現場監督（『石綿曝露のリスク評価表』の作業別リスク分類 参照）

説明文

```
┌────┬──┬──┬──┬──────┬────────────┬────┐
│分野│大│中│小│      │   説明文     │ KW │      キーワードのフィールドコードを設けて
└────┴──┴──┴──┴──────┴────────────┴────┘      キーワードを登録する？
              └── 説明文の分類（プライオリティ等）
```
　　　説明文は、同一用語に複数の説明文の設定が可能。　説明文の分類は後日検討。暫定で2桁としておく。
　　　　　　　　　　　　　　　　　　　　　　　　　　　　　説明文のデータ長は制限なし

出典情報
　　　　　　　　　　　　　　　　　　　　　　　　　　　　　キーワードのデータ長は30Byte、個数はMax10

```
┌────┬──┬──┬──┬──┬──────────────────────────────┐
│分野│大│中│小│  │ 著者名、書名、研究タイトル、出版社名、号数・巻、ページ数 │    フィールドコードを設ける？
└────┴──┴──┴──┴──┴──────────────────────────────┘
              └── 文献の種類（著書、論文、法律、省令、条例、通達、学会のガイドライン、学会発表、研究報告書、
                            商業誌、新聞、インターネット、……）　清本先生
          └───── 説明文の分類　　　文献の種類：2桁　　ENDNOTE参照
```
　　　出典が複数ある場合の登録が可能。

用語のリンク（関連用語）

```
        主コード                      リンクされる用語コード
  ┌──────────────┐          ┌──────────────┐
┌────┬──┬──┬──┬────┬────┬──┬──┬────┬──┬──┬──┐
│分野│大│中│小│同義│確実性│頻度│濃度│分野│大│中│小│
└────┴──┴──┴──┴────┴────┴──┴──┴────┴──┴──┴──┘
                └─ 関係分類
                   それぞれが3段階必要
```

　　　一つの用語に対し、複数のリンク設定が可能。
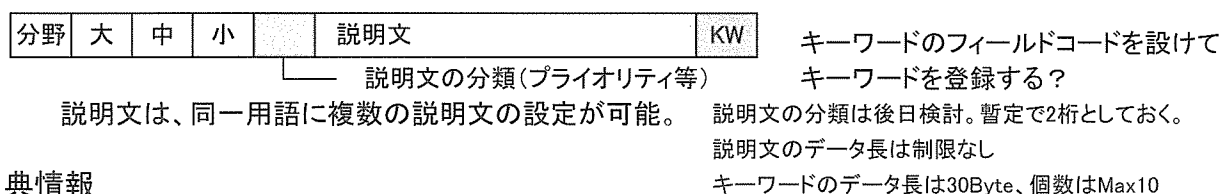　　　関連付けは追加登録により変わることがあるので、再付与を可能とする。

リスク情報（スコア）

```
                          ウェイト
┌────┬────┬────┬──────┬──────┬────┬──┬──┬────┬──┬──┬──┐
│スコア│作者│疾病│時代    │従事  │（量）│  │  │分野│大│中│小│    単独の場合のスコア
│    │    │分類│（年-年）│時間  │    │  │  │    │  │  │  │
└────┴────┴────┴──────┴──────┴────┴──┴──┴────┴──┴──┴──┘
                                    │         └──────┘
                                  予備          用語コード

┌────┬────┬────┬──────┬──────┬────┬──┬──┬────┬──┬──┬──┐        ┌──────┬──────┬────┬────┬──┬──┬──┐
│スコア│作者│疾病│時代    │従事  │（量）│  │  │分野│大│中│小│………  │時代    │従事  │（量）│分野│大│中│小│
│    │    │分類│（年-年）│時間  │    │  │  │    │  │  │  │        │（年-年）│時間  │    │    │  │  │  │
└────┴────┴────┴──────┴──────┴────┴──┴──┴────┴──┴──┴──┘        └──────┴──────┴────┴────┴──┴──┴──┘
                                                                          組み合わせによるスコア
```
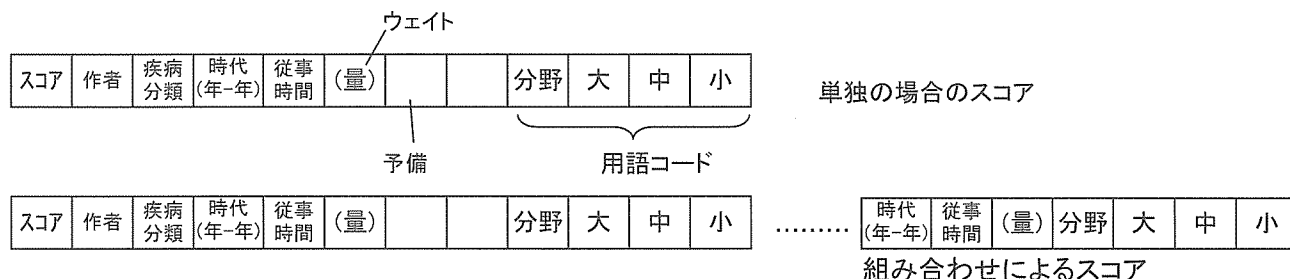
例　現場監督　　　1945-1970　　　1年
　　現場監督　　　1945-1970　　　5年
　　現場監督　　　1945-1970　　　10年
　　現場監督　　　1980-1999　　　5年

石綿曝露のリスク評価表

1. 作業別リスク分類

グループ	D	特高	①石綿の採掘、搬出、粉砕、生成作業
グループ	D	特高	②石綿原料の袋詰め、船積み、荷下ろし、石綿製品の梱包・運搬作業
グループ	B	中	③石綿製品の製造工程における作業
グループ	D	特高	④石綿吹付け作業
グループ	C	高	⑤耐熱性の石綿製品を用いた断熱・被覆・補修作業
グループ	C	高	⑥石綿製品の切断等の加工作業
グループ	B	中	⑦石綿製品が用いられた建物、その附属施設などの補修又は解体作業
グループ	D	特高	⑧石綿製品が用いられた船舶・車両(汽車・電車)の補修または解体作業
グループ	A	低	⑨石綿を不純物として含有する鉱物(タルク等)の取扱い作業
グループ	B	中	⑩石綿製品(フィルター、布)を直接取り扱う作業
グループ	B	中	⑪その他(自動車整備工等)

※2つ以上職業があった場合、リスクが高いグループを基準とする

2. 時代別リスク分類

時代	C	高	①～1940年
時代	B	中	②1941年～1945年
時代	D	特高	③1946年～1975年
時代	C	高	④1976年～1987年
時代	B	中	⑤1988年～2004年
時代	A	低	⑥2004年～

※時代がまたがる場合は、リスクが高いグループを基準とする

3. 従事期間別リスク分類

従事期	A	低	①1年未満
従事期	B	中	②1年～10年未満
従事期	C	高	③10年～

※2つ以上職業があった場合、従事期間を合計する

4. リスクレベルの判定表

年度が跨った時、按分して係数を算出する。

作業グループ	従事期間		時代A 2004年～	時代B 1988年～2004年	時代C 1976年～1987年	時代D 1946年～1975年	時代C ～1940年	時代B 1941年～1945年
A	短期間	1年未満	1	1	1	1	1	1
	中期間	1年～10年未満	1	1	1	2	1	1
	長期間	10年～	1	1	2	2	2	1
B	短期間	1年未満	1	1	1	1	1	1
	中期間	1年～10年未満	1	2	2	2	2	2
	長期間	10年～	2	2	3	3	3	2
C	短期間	1年未満	1	1	2	2	2	1
	中期間	1年～10年未満	2	2	3	3	3	2
	長期間	10年～	2	3	4	4	4	3
D	短期間	1年未満	2	2	3	3	3	2
	中期間	1年～10年未満	3	3	4	4	4	3
	長期間	10年～	3	4	4	4	4	4

5. リスク評価に基づく対策表

レベル	総合評価	有所見がない場合の次回フォローアップ	
		初回曝露から10年～20年	初回曝露から20年～
1	曝露レベルはほぼ問題とならない。	不要	不要
2	曝露レベルは軽度である。	不要	5年後
3	曝露レベルは中等度である。	5年後	3年後
4	曝露レベルは高度である。	3年後	1年後