Summary

A pilot study on the health effects of environmental exposure to asbestos (in particular the development of mesothelioma) is almost complete. This is a record linkage study, using birth and death records as the primary sources of data. The vital status and, if applicable, the cause of death was examined for each of the 127 members of the 'pilot' cohort (birth years 1932 - 1936). Preliminary results are presented. Eighty-seven per cent (399) of the white cohort members have been traced and the vital status of each has been determined. Sixty-six whites have died, 6 from mesothelioma. It is almost impossible to trace the black and coloured cohort members and the main study, covering the years of birth 1917 - 1936, may have to be restricted to whites.

A number of diseases have been associated with asbestos exposure, including many cancers such as lung cancer, gastrointestinal tract cancers,1 and cancers of the bladder2 and kidney.3,4 However, respiratory tract cancers (which include bronchogenic carcinoma and laryngeal carcinoma) and mesothelioma are the most important asbestos-related causes of death.5

Mesothelioma is characterised as a highly malignant cancer which originates in the lining of the chest or abdominal cavity. In almost all instances it is rapidly fatal, with median survival times ranging from 2 to 18 months from diagnosis. The disease has a long latency period with onset usually 35 years or more after exposure.6 However, cases of mesothelioma have been reported to occur after very short periods since first exposure7 and in children.8 The tumour has also been known to occur after as little as 1 month's exposure in a dusty job.9

In a recent study of the incidence of mesothelioma in South Africa,10 71 cases were referred from Prieska Hospital. This large number of cases prompted a more thorough investigation into the problem in this area.

Prieska, in the north-western Cape, is a district where environmental exposure to asbestos has occurred since the late 1800s. The town is situated on the Orange River and has been associated with the mining of crocidolite (blue asbestos) since 1893.11 Initially, mining was done in open-cast workings; inclined shaft mining became common only after 1930.12 Hand-cobbing of asbestos was the method used to extract fibres from the rock before a crushing mill was built in Prieska in 193012 in close proximity to residential and business areas; the mill was in operation until approximately 1964 (B. Swart — personal communication). During this period, all the residents were exposed to fibres, either as employees of the mining company (some women were employed as cobbers and their children accompanied them to work) or in their daily activities around the town. In the early days, the manager and labourers lived within a few yards of their place of work and their children played on the nearby dumps from the mines and the mill. In addition to this, asbestos spillage occurred during transportation (both to the mill and to the tailings dump in the town); the tailings were used to surface the roads and to make bricks for building, and were scattered over the golf tees (B. Swart — personal communication).

Many studies have shown the association between asbestos exposure and the development of mesothelioma,12-14 but these have mostly been concerned with occupational, rather than environmental, exposure. The risk posed by low-level exposure is still one of the more perplexing and technologically difficult questions to address in relation to asbestos-associated disease.5 Current knowledge of the risks associated with low-level (environmental) exposure relies on extrapolation from findings associated with high-level (occupational) exposure. The consequences of living in an environment where low-level non-occupational exposure occurs, e.g. near an asbestos mine or mill, have not been studied, although there are several well-documented reports indicating the occurrence of cases of mesothelioma in the vicinity of crocidolite mines and factories.12,13,16

We hypothesised that, owing to the close proximity of the asbestos industry to Prieska and the resulting environmental exposure of the town's residents, people born in Prieska will have a measurably higher incidence of asbestos-related causes of death than those born elsewhere in the RSA.

A secondary objective of the study was to explore the feasibility of doing record linkage studies of this kind in the RSA.

Methods

The effects of environmental exposure to asbestos are being examined by means of a birth cohort mortality study. The years 1917 - 1936 were chosen to take into account the long latency period of mesothelioma and the short survival time of patients with the tumour. Among whites most cases occur in the 51 - 70-year age group, while among blacks and coloureds most occur at a younger age, i.e. 41 - 50 years.19 The age of the cohort members will therefore range from 54 years to 73 years in 1990 and the study should be able to detect both short- and long-term effects of environmental exposure to asbestos.

To determine the feasibility of the research, a cohort for a pilot study was established for the 5-year period 1932 - 1936. This pilot study was necessary to assess the quality and accessibility of the available records. Data were collected from the birth register for the Prieska district and included date of birth, first names, surname and race of father, and surname of mother.

It was then necessary to "identify" each member of the cohort to determine his or her vital status. This involved finding the identification number of each member using the computerised population register stored in the mainframe computer of the Department of Home Affairs in Pretoria. Manual searches through personal files were also carried out.
Once the personal identification number of a person was known, it was possible to determine whether or not he or she had died and, if so, to obtain the death certificate and thus ascertain the cause of death.

The causes of death were analysed from the death certificates of all deceased members of the cohort. The crude mortality rates and 95% confidence limits were calculated for mesothelioma in the white population group.¹⁷

### Results of the pilot study

One thousand two hundred and twenty-seven births were registered in the Prieska district from 1932 to 1936, the racial distribution being as follows: white - 458 (37,3%); coloured - 468 (38,1%); black - 97 (7,9%); Asian - 2 (0,2%); unknown race - 202 (16,5%) (Table I).

Eighty-seven per cent of the whites have been identified to date (Table II). Unfortunately, very few coloureds (35%) and even fewer blacks (22%) have been identified. This is due to the way in which the death records of these population groups are archived (see 'Discussion').

<table>
<thead>
<tr>
<th>Race</th>
<th>Year of registration</th>
<th>White</th>
<th>Coloured</th>
<th>Black</th>
<th>Unknown</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1932</td>
<td>124</td>
<td>111</td>
<td>29</td>
<td>34</td>
<td>298</td>
</tr>
<tr>
<td></td>
<td>1933</td>
<td>79</td>
<td>80</td>
<td>16</td>
<td>24</td>
<td>199</td>
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<td></td>
<td>1934</td>
<td>86</td>
<td>75</td>
<td>18</td>
<td>47</td>
<td>226</td>
</tr>
<tr>
<td></td>
<td>1935</td>
<td>89</td>
<td>119</td>
<td>20</td>
<td>36</td>
<td>264</td>
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<td></td>
<td>1936</td>
<td>80</td>
<td>83</td>
<td>14</td>
<td>61</td>
<td>238</td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>458</td>
<td>468</td>
<td>97</td>
<td>202</td>
<td>1225</td>
</tr>
</tbody>
</table>

*Plus 1 Asian.

One hundred and fifty-eight of the 582 identified cohort members (27,2%) have already died. Of these, 16 died of a neoplasm of one sort or another; 10 of the 16 died of a respiratory neoplasm, of which 6 were mesotheliomas (Table III). It was established, through contact with the relevant medical practitioners, that biopsy specimens had been available on which to base the diagnoses of all 6 mesotheliomas. Eighty-five (53,8%) of the 158 deaths occurred under 1 year of age.

Three hundred and ninety-nine whites have been identified, 66 (16,5%) of whom have died. Of the 6 who died from mesothelioma, 3 were men and 3 were women.

The cumulative crude mortality rates and confidence limits for whites are shown in Table IV. The rate for women was slightly higher than that for men (16,1 and 14,1/1 000, respectively).

### Discussion

Data necessary to do a record linkage study of this kind in South Africa are obtainable for the white population group.
However, the death records for blacks and coloureds are archived according to place of death, which makes it virtually impossible to trace any one person. In addition, deaths before 1986 have not yet been computerised. The pilot study has therefore been restricted to whites until such time as these uncomputerised records can be examined. However, even in the case of whites, only those issued with 'new' identity documents can be traced on the computer.

The mortality rates reported here were obtained from the first analysis of the pilot study data and are thus only preliminary. Statistical comparison with the general population or with rates elsewhere in the world is therefore not appropriate at the moment.

Nevertheless, a crude comparison with Zwi et al.'s 10 study indicates that the mortality rates for both white males and females born in Prieska are extremely high (Table V).

<table>
<thead>
<tr>
<th>TABLE V. COMPARISON OF AGE-STANDARDISED MORTALITY RATES DUE TO MESOTHELIOMA (TOTAL SOUTH AFRICA) 10 WITH CRUDE MORTALITY RATES IN PRIESKA</th>
</tr>
</thead>
<tbody>
<tr>
<td>South Africa</td>
</tr>
<tr>
<td>(X 10^3)</td>
</tr>
<tr>
<td>annual</td>
</tr>
<tr>
<td>White males</td>
</tr>
<tr>
<td>White females</td>
</tr>
</tbody>
</table>

Three of the 6 deaths from mesothelioma occurred among white women. McDonald 18 has stated that the incidence of malignant mesothelioma among women has great potential as an indicator of environmental asbestos exposure.

White females in South Africa who die from mesothelioma should give the best indication of the risk posed by environmental exposure, since this group is least likely to have had occupational exposure to asbestos. This was borne out by a study by Sluis-Cremer 19 in 1965, when he divided a population in the north-western Cape into those who had been exposed to asbestos through their occupations and those who had not. He found no sample for exposed white women over the age of 20 years, i.e. none had worked in an asbestos mining, milling, transporting or consumer industry or capacity, for however short a period. In Prieska itself no white women were ever employed in the mill; only black and coloured workers were employed as cobbers. The relevant medical practitioners were questioned about any occupational exposure to asbestos that the 3 women who died of mesothelioma might have had. In 1 case records of the South African Asbestos Tumour Reference Panel 20 were available. None of the 3 women had histories of occupational exposure to asbestos.

It is therefore likely that none of the 3 women who died from mesothelioma in this pilot study had worked in the asbestos industry, and one may deduce that the crude mortality rate of 16.1/100 000 is due to environmental exposure alone.

In 1984/5 fibre levels in the residential area of Prieska were reported to range from 0.2 to 0.7 fibres per litre. 21 The asbestos mill in Prieska ceased operations approximately 20 years before the recording of these environmental levels, which were presumed much lower than levels in the 1940s and 1950s. Levels as high as 630 - 800 fibres per millilitre were, in fact, recorded inside the mill in Prieska in 1948 (R. S. J. du Toit - personal communication). These fibre samples were obtained by means of the Konimeter and were counted using a 125 X magnification, under dark-field illumination after acid treatment. The mill, however, was not the only source of asbestos fibres in the area. Fibres were also shed during transport of asbestos and when using the tailings.

This cohort will be followed annually for at least 10 years. The possibility of 'flagging' records in the population register for quick and easy access to records of future deaths in the cohort was examined. However, permission to do this was not granted by the relevant authorities on the grounds that the process was not feasible. Therefore, every 12 months the population register will be examined for deaths of any of the cohort members that occurred since the previous analysis of the data.

A serious limitation of the study is lack of data regarding intensity and duration of exposure. Some fibre measurements were recorded during the relevant period and these will be examined for possible estimates of the intensity of exposure of the cohort. As regards the problem of duration, data sets such as school enrolment lists and church records are available, and these may indicate which members of the cohort were still living in the area in certain years.

**Conclusion**

The high mortality rate for mesothelioma among white women is indicative of the risk posed by environmental asbestos. These preliminary results of the pilot study, which set out to determine the feasibility of such a record linkage study, indicate that many more people may die from the tumour in subsequent years of follow-up. Those who can prove occupational exposure are financially compensated but no such legislation exists for environmental exposure to asbestos. The authors are aware of the importance of assessing possible occupational exposure and are determining the feasibility of such an assessment. The final result of this study may be the first step towards motivating for compensation for people who develop mesothelioma and have had no occupational exposure to asbestos.

The authors wish to thank Mrs C. E. Compton (Department of Home Affairs) for her invaluable computer assistance.

**REFERENCES**