

Asthma in goldminers

Robert L. Cowie, Salmon K. Mabena

Objectives. To determine whether asthma in goldminers is caused by or contributed to by their working environment.

Design. A case-control study in which men with asthma working underground in goldmines were compared with underground goldminers without asthma in relation to their age, duration of exposure to the underground environment, atopy and family history of asthma.

Setting. An in- and outpatient facility providing for the medical needs of approximately 90 000 miners employed on goldmines in the Free State.

Outcome measures. Occupational history, atopy and family history of asthma were compared in the two groups. The age of onset of asthma and duration of occupational exposure were examined in the men with asthma.

Results. The study sample included 78 underground miners with asthma and 46 without asthma. The men in the two groups were of similar age, but those with asthma had worked underground for a longer period than the men without asthma. Twenty of the asthmatic and none of the control group had been exposed to paint and cement in the course of their work. Fifty of the asthmatic and only 3 of the control group were atopic. The mean age of onset of asthma (\pm SD) was 30.6 ± 10.73 years. Six of the men had developed asthma before starting to work in the mines, and the disease had developed 13.4 ± 8.22 years after starting to work underground in the remaining 72.

Conclusion. The late age of onset and the onset after exposure to the underground environment suggest that the disease was work-related.

S Afr Med J 1996; **86**: 804-807.

Department of Medicine, Ernest Oppenheimer Hospital, Welkom, Free State

Robert L. Cowie, M.D., M.Sc., F.C.P. (S.A.) (Present address: Department of Medicine, University of Calgary, Alberta, Canada)

Salmon K. Mabena, R.N.

Although asthma is reported to be uncommon in rural southern Africa,¹ it is a common chronic disease among men from those areas who work on the goldmines in the Free State province of South Africa.

Occupational asthma may be the commonest of the occupational lung diseases.² It is important and sometimes difficult to distinguish between the asthmatic in the workplace and the individual who has acquired asthma through exposure in the workplace.

In this case-control study of black South African goldminers, a group with asthma was examined and compared with a control group without asthma. Comparisons between the two groups included workplace exposures, atopic status and family history of asthma.

Methods

The study group comprised a consecutive sample of men with asthma attending the routine follow-up clinic at Ernest Oppenheimer Hospital. The hospital provided all in- and out-patient medical care for a population of approximately 90 000 goldminers. During a 3-month period, 84 men with asthma were seen at the clinic and invited to participate in the study. All agreed.

The control group comprised 50 men who attended the hospital for minor surgical disorders. They, too, were goldminers employed in the same mines as the study group. The control group would have been seen at the same medical facility had they suffered from or developed asthma.

The goldminers with asthma had that disease as their major disorder. All had previously attended the outpatient clinic for evaluation and management. Their clinical diagnosis, made by one of the physicians at the clinic, had been confirmed, in every instance, by demonstrating instability on spirometry with a difference of at least 15% between their best and their worst forced expiratory volumes in 1 second. The control group was distinguished by having never attended the hospital for asthma and by their negative response to questions relating to respiratory symptoms.

All the subjects in the study were interviewed by one of the authors (S.K.M.), using a questionnaire-based interview. The interviews were conducted in the subject's own language. The questionnaire addressed their occupation in the goldmine, whether they had changed their occupation in the mine, and if so, what their previous occupation(s) had been. Each man was asked whether he had ever been exposed to chemicals, including cleaning agents, gases, fumes or any strong-smelling or irritant substances in the course of his work on the mines. Men were asked whether they recalled any childhood chest illnesses, and whether they had a family history of chest disease in general and of asthma in particular. Men with asthma were asked their age at the onset of their disease and their occupation at that time. Each man was then tested using the standard prick test method for reaction to house-dust mite, South African grass pollens, cat dander and *Aspergillus fumigatus*.

A negative control and histamine were used with each test.

Results

On reviewing the data it was found that 6 of the 84 men in the asthma group and 4 of the 50 men in the control group had never worked underground in a goldmine. These men were excluded from further analysis. Details concerning the two groups are provided in Table I. In summary, the group of men with asthma were not significantly older than the control group. They had, however, worked significantly longer underground than had the control group. Atopy, defined by a positive skin-prick test to at least one of the common aero-allergens,³ was present in 64% of the asthmatic group and in only 7% of the control group. None of the men in the control group reported any exposure to chemicals or other irritants, while 20 (26%) of the men with asthma made specific mention of being exposed to paint and/or cement (Fisher's exact test, $P < 0.001$). These two substances tend to be used in the same area by men working in the haulage areas of the mine. Eleven of the men with a history of paint/cement exposure were atopic and 9 were non-atopic (Fisher's exact test, $P > 0.05$). Only 1 of the men in the control group had changed his job in the mine during his career as a goldminer. In contrast, 41 (53%) of the asthmatic group had changed their jobs (Fisher's exact test, $P < 0.001$). The mean age of onset of asthma (\pm SD) was 30.6 ± 10.73 years. Fig. 1 shows the ages of onset for the men with asthma. The onset of asthma occurred 13.4 ± 8.22 years after starting to work as a miner in the 72 men whose disease had not developed at the time of their first recruitment to the mines.

Table I. Details on the asthmatic goldminers and the controls

	Asthmatic (N = 78)	Controls (N = 46)	Significance
Age (yrs) (mean \pm SD)	37.2 (\pm 9.09)	34.5 (\pm 9.37)	NS
Age at onset of asthma (yrs) (mean \pm SD)	30.6 (\pm 10.73)	—	
Years worked underground (mean \pm SD)	20.1 (\pm 9.27)	14.8 (\pm 10.07)	$t = 3.0$ $P = 0.003$
Atopy (skin-prick test positive)	50 (64%)	3 (7%)	$P = 0.001$
Job changed	41 (53%)	1 (2%)	$P = 0.001$
Paint/cement exposure	20 (26%)	0	$P = 0.001$
Family asthma	14 (18%)	0	$P = 0.002$
Childhood chest disease	6 (8%)	0	NS

Job change refers to any change in occupation within the mine. Paint/cement exposure identifies men who specifically mentioned exposure at work to paint or to cement.

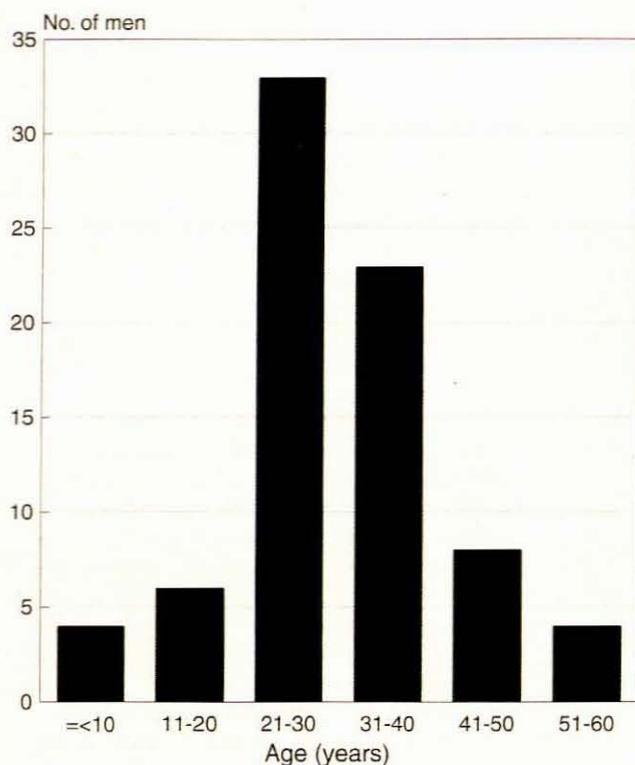


Fig. 1. Age at which subjects with asthma reported having first experienced symptoms. The mean age of onset (\pm SD) was 33.5 ± 8.79 years for the 72 men with onset of asthma after starting to work as a miner. The median age of onset of asthma for that group was 31 years, with a range of 21 - 59 years.

Discussion

This study shows distinct differences between the men with asthma and the control group. The choice of the control group was determined in part for the convenience of including men who attended the medical facility rather than attempting to obtain a random sample of working men on the mines. It was considered appropriate to choose men whose attendance at the medical facility had no connection with asthma and whose disorders had no features which might be associated with or confused with asthma. On the other hand, the men in the control group would have been seen in the same facility had they developed asthma, and in that sense were representative of the non-asthmatic portion of the working population.⁴

The study has not clearly determined whether asthma in these goldminers is attributable to their occupation. In favour of that hypothesis is the unusually late age of onset of the disease and the fact that, in all but 6 of the 78 men, asthma had developed after starting work on the mines. This pattern of onset would ordinarily suggest occupational asthma, but is possibly explained by self-selection of men with early-onset asthma for occupations other than mining. It might also be attributable to the previously noted late onset of asthma in black Africans.^{5,6} The men in the present study group could therefore have been asthmatics who at the time of their first enrolment as goldminers had not yet expressed their disease.

The prevalence of atopy in the men with asthma is the strongest evidence to suggest that these men had a pre-existing predisposition to develop asthma. It is recognised, however, that atopy can predispose to occupational asthma caused by exposure to experimental animals⁷ and, possibly, to other high-molecular-weight compounds.⁸ Atopy can predispose to sensitisation to, and probably also to asthma from exposure to, platinum salts.⁹ There is currently no evidence that high-molecular-weight asthma-inducing compounds are to be found in the mine environment. Nevertheless, the pre-existence of atopy does not exclude the possibility that asthma in these goldminers was caused by occupational exposure.

No attempt was made to determine the prevalence of smoking in the two groups in the study. Smoking is highly prevalent in this working population, but the asthmatic subjects had been extensively advised not to smoke and it was therefore felt that their uniform insistence that they did not smoke was not necessarily reliable. On the basis of an earlier study in this working population, 70% of the control group would have been expected to smoke.¹⁰ It is likely that the asthmatic group smoked less than the control group, but unlikely that the excess of atopy in the asthmatic group was due to differences in smoking.¹¹

The significant relationship between exposure to paint and cement in the men with asthma is difficult to interpret. The paints to which they were exposed included lime wash, latex and oil paints but not polyurethane, twin-pack paints. Paints and cement are used together by men working largely in the mine haulage areas. Work in these areas is generally the least physically demanding of the underground occupations, and it is possible that work there would attract men with respiratory disease. Men with asthma were more likely to change their occupations in the mine. In general men will find the most demanding occupation they can manage, and since the pay scales are related directly to the physical difficulty of the occupations, men will tend to remain in their chosen positions; the pattern of changing of jobs would be likely to reflect a progressive scaling down of occupation by the men with asthma. It is therefore possible that exposure to paint or cement followed the development of respiratory symptoms that caused the miner to move to a less demanding occupation. Exposure to cement has been associated with bronchitic disease and with chronic airflow limitation, but not with asthma.¹²⁻¹⁴ The paint to which these men were exposed has similarly been associated with chronic bronchitis and chronic airflow limitation, especially in smokers, but not with asthma.^{15,16} Nevertheless, the exposure to these substances was specifically and spontaneously mentioned by the men, who also stated their belief that it was the cause of their disease, or at least that it had contributed to their disease.

One method that has been used to distinguish occupational asthma from asthma in a working population has been to follow airway reactivity and peak expiratory flow rates after cessation of exposure. While this might be feasible, failure to improve away from the workplace would not necessarily exclude an occupational cause in our subjects, who had had asthma for several years. Studies have shown that it is not unusual for occupational asthma to persist after withdrawal from the working environment, and the likelihood of persistence increases directly with the

duration of the disorder.¹⁷⁻²⁰ Nevertheless, future studies of asthma in this working population should include evaluation of men during the periods between working contracts. A remission from their asthma would support an occupational association and justify further investigation.

This study group epitomises the problem of definition of occupational asthma.^{8,21} Most definitions include the requirement that the disease commenced after a period of symptom-free exposure to an agent known to induce asthma. In the mine environment the atmosphere contains dusts, remnants of fumes from blasting, and chlorine from the water used to control the dust. Contamination of the water used for humidification and dust control may well occur and may be a cause of occupational asthma.²²

In conclusion, although the higher prevalence of atopy and greater prevalence of a family history of asthma in the study group suggests that their occupation and their disease were coincidental, an occupational cause for their asthma is suggested by the onset of the disease after several years of exposure to the mine environment; the fact that the men with asthma had worked underground longer than had the control group; and the late age of onset of their asthma. Additional studies of the mine environment and peak flow monitoring of miners with asthma at and away from the workplace might help to clarify the nature and the cause of their asthma.

REFERENCES

1. Weinberg EG, Van Niekerk CH, Shore SC, Heese HdeV, Van Schalkwyk DJ. Prevalence of asthma. *Lancet* 1977; **2**: 500.
2. Chan-Yeung M. Occupational asthma update. *Chest* 1988; **93**: 407-411.
3. Pepys J, Davies RJ. Allergy. In: Clark TJH, Godfrey S, eds. *Asthma*. London: Chapman & Hall, 1977.
4. Miettinen OS. *Theoretical Epidemiology: Principles of Occurrence Research in Medicine*. New York: John Wiley, 1985: 54-55.
5. Warrell DA, Fawcett IW, Harrison BDW, et al. Bronchial asthma in the Nigerian Savannah region. *Q J Med* 1975; **44**: 325-347.
6. Cookson JB. Prevalence rates of asthma in developing countries and their comparison with those in Europe and North America. *Chest* 1987; **91**: 97S-103S.
7. Newman-Taylor AJ. Laboratory animal allergy. *Eur J Respir Dis* 1982; **63**: suppl 123, 60-64.
8. Chan-Yeung M, Lam S. Occupational asthma. *Am Rev Respir Dis* 1986; **133**: 686-703.
9. Dally MB, Hunter JV, Hughes EG, Stewart M, Newman-Taylor AJ. Hypersensitivity to platinum salts: a population study (Abstract). *Am Rev Respir Dis* 1980; **121**: 230.
10. Cowie RL, Mabena SK. Silicosis, chronic airflow limitation and chronic bronchitis in South African goldminers. *Am Rev Respir Dis* 1991; **143**: 80-84.
11. Burrows B, Halonen M, Barbee RA, Lebowitz MD. The relationship of serum immunoglobulin E to cigarette smoking. *Am Rev Respir Dis* 1981; **124**: 523-525.
12. Kessel R, Redl M, Mauermayer R, Praml GJ. Changes in lung function after working with the shotcrete lining method under compressed air conditions. *Br J Ind Med* 1989; **46**: 128-132.
13. Abrons HL, Petersen MR, Sanderson WT, Engleberg AL, Harber P. Symptoms, ventilatory function, and environmental exposures in Portland cement workers. *Br J Ind Med* 1988; **45**: 368-375.
14. Shamssain MH, Thompson J. Effect of cement dust on lung function in Libyans. *Ergonomics* 1988; **31**: 1299-1303.
15. White MC, Baker EL. Measurements of respiratory illness among construction painters. *Br J Ind Med* 1988; **45**: 523-531.
16. Schwartz DA, Baker EL. Respiratory illness in the construction industry: airflow obstruction among painters. *Chest* 1988; **92**: 134-137.
17. Hudson P, Cartier A, Pineau L, et al. Follow up of occupational asthma caused by crab and various agents. *J Allergy Clin Immunol* 1985; **76**: 662-688.
18. Moller DR, Brooks SM, McKay RT, Cassidy KM, Kopp S, Bernstein IL. Chronic asthma due to toluene diisocyanate. *Chest* 1986; **90**: 494-499.
19. Lozewicz S, Assoufi BK, Hawkins R, Newman-Taylor AJ. Outcome of asthma induced by isocyanates. *Br J Ind Med* 1987; **81**: 14-22.
20. Chan-Yeung M, Lam S, Koener S. Clinical features and natural history of occupational asthma due to Western red cedar. *Am J Med* 1982; **72**: 411-415.
21. Hendriks DI. Editorial: Occupational asthma — problems of definition. *J Occup Med* 1983; **25**: 488-489.
22. Burge PS, Finnegan M, Horsfield N, et al. Occupational asthma in a factory with a contaminated humidifier. *Thorax* 1985; **40**: 248-254.