EPIDEMIOLOGICAL EVIDENCE ON SMOKING, DUST EXPOSURE, AND LUNG DISEASE IN COAL MINERS

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INTRODUCTION

Coal miners are known to experience various adverse effects from breathing coal mine dust, including pneumoconiosis,[1,2] reduced ventilatory function,[3,4] and increased respiratory symptoms. (5,6). They as do all other groups of workers, suffer from the deleterious effects of smoking. Thus, those who experience high levels of dust and who also smoke are in double jeopardy from the combined insults to their respiratory systems.

A question that occasionally gets asked relates to the possible interactive nature of these two respiratory hazards. In particular, Bundy[7] asked whether the chance of developing pneumoconiosis was affected by the smoking status of the miner. Similar questions can be posed about the decline in FEV_1 with dust exposure in smokers and non-smokers, and trends in symptom prevalences.

In essence, these questions relate to the potentially synergistic nature of the combined effects of dust exposure and smoking.

Some answers to these questions do exist, but they are scattered in the literature, or have not been published. The intention of this report is to bring together information on these topics from various sources, so as to explore the question of the mutual effects of coal mine work and smoking, with the primary intention of determining whether miners who smoke are at additional risk of lung disease, due to the simple additive effects of occupation and smoking.

The results presented here are from two large studies: the Pneumoconiosis Field Research,[8] which deals with British miners; and the National Study of Coalworkers' Pneumoconiosis, which is a study of U.S. coal miners. Three main topics will be covered, including evidence on small and large opacities, respiratory symptoms, and ventilatory function as measured by the forced expiratory volume in 1 second (FEV_1).

The section on radiological abnormalities will deal first with small rounded opacities. In this, the role of phlegm production in the deposition of dust in the airways will be touched upon. As interest in the relationship between small, irregular opacities and dust exposure has been shown recently,[9] this topic is also reviewed. Lastly in this section, the limited evidence on smoking and PMF development is discussed.

Apart from radiological abnormalities, there are two other measures of respiratory health for which evidence of relationships with dust exposure exist. These are respiratory symptoms and ventilatory function (most commonly, FEV_1). The second section in this report deals with symptoms of chronic bronchitis, as defined by persistent cough and phlegm. Finally, attention is given to the problem of the association between FEV_1 and dust exposure in the presence or absence of smoking.

Sources of information

Since its beginnings in 1952, The Pneumoconiosis Field Research (PFR) has published a long series of articles relating to the various diseases and abnormalities that can arise through exposure to coal mine dust. It is clearly not possible to describe the methods and approaches used in those investigations here. Instead, those interested should read Fay and Rae[8] for a history of the PFR, while consulting the papers referenced here for further details of the methods of the individual studies.

The data presented here on U.S. miners, much of it for the first time, are drawn from the first round of the National Study of Coalworkers' Pneumoconiosis (NSCWP). The x-ray abnormalities analyzed are those of one of the three interpreters used during that round. Morring and Attfield[10] have shown that that reader obtained prevalences of pneumoconiosis similar to those of PFR readers, and, as a result prevalences based on his readings are comparable to British readings. Since PFR data are also being reviewed here, it is appropriate to use this interpreter's readings.

Information on chronic bronchitis in the NSCWP was obtained by interview, using a modified British Medical Research Council questionnaire.[11] In the results presented here, a miner was classified as having chronic bronchitis if he or she admitted to coughing, and to producing phlegm, first thing in the morning or during the day for most days or nights, for as much as three months in the year. The FEV_1 data used were derived from the maximum of three valid repeat blows for each person, subsequent to two trial blows.

Dust exposure information for the period prior to the first round of surveys was computed from records of time spent in various jobs, as reported by each miner, coupled with concentration data drawn from the survey reported on by Jacobson [12] and supplemented by data collected by the Mine Safety and Health Administration (MSHA, previously MESA) in the early 1970's. A description of the derivation of these exposures, and a discussion of their validity, is given by Attfield and Morring.[13] Details of the results from their correlation with radiological data and ventilatory function are currently being prepared for separate publication.

The number of miners included in all of the NSCWP analyses, except that for those with chest symptoms consisted of over 1500 non-smokers, and more than 4000 current smokers. The symptom analysis was based on 1335 miners aged 35 - 44, of whom 311 were non-smokers.

In this section, as in all sections, information from the PFR is given first, as results from that study were analyzed and published prior to those for U.S. miners.

RESULTS

Small rounded opacities

Figure 1 gives prevalences of category 0/1 or greater small rounded opacities, by age, for smokers and non-smokers. These data were extracted from Table 1 of Jacobsen et al.,[14] and include information for both those admitting and not admitting to phlegm production. Later in the same article, Jacobsen et al. presented the data plotted against dust exposure, and again the slopes were similar for smokers and non-smokers, indicating lack of interaction between smoking

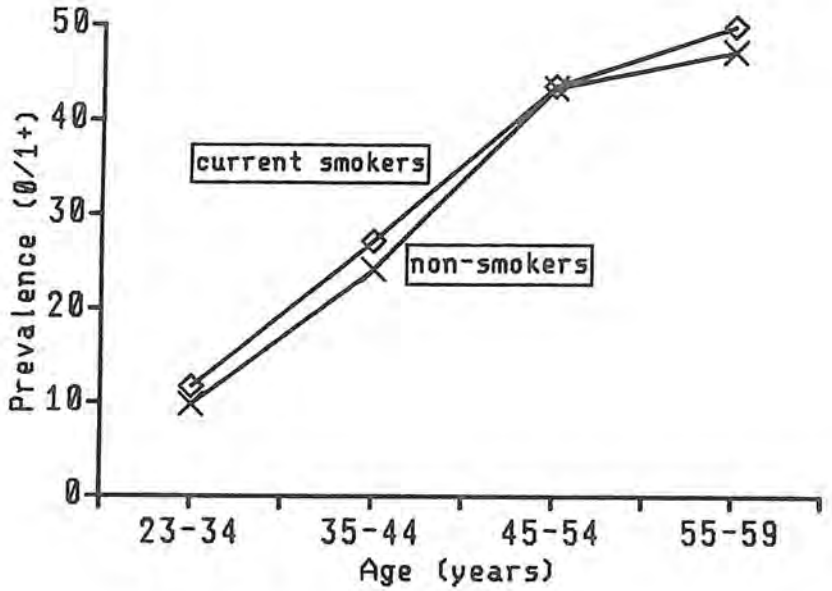

Figure 1. Prevalence of small rounded opacities (category 0/1 or greater) against age for two smoking groups. British data. (Data from Jacobsen et al.[14])

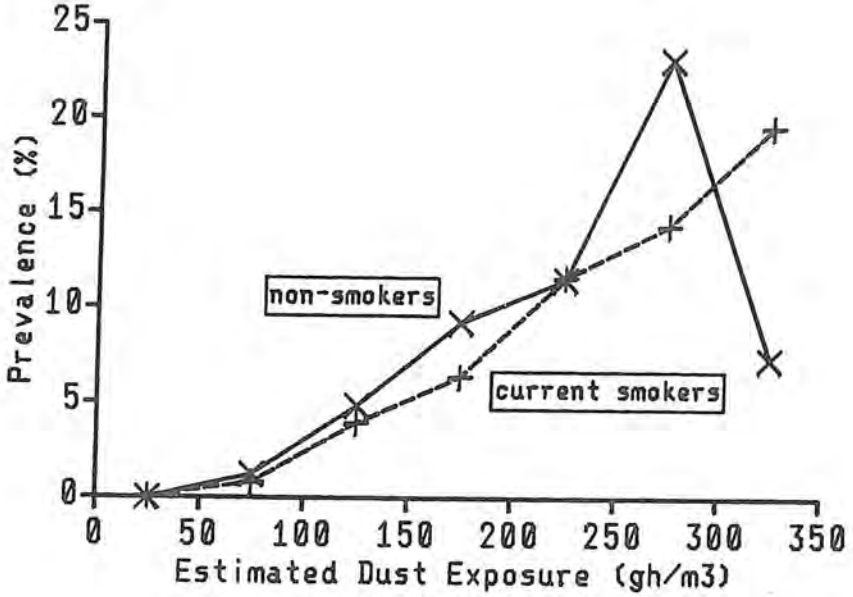

Figure 2. Prevalence of small rounded opacities (category 0/1 or greater) against dust exposure for two smoking groups. U.S. data.

status and dust exposure.

The above analysis was undertaken primarily in response to the question of Bundy[7], who questioned whether smoking history influenced the slope of the probability curve (linking risk of pneumoconiosis against dust concentration). In the summary, Jacobsen et al. wrote, "It is concluded that the main variable determining the development of simple pneumoconiosis is exposure to airborne dust, and that this effect is not modified appreciably by whether or not coal miners smoke".

Soutar et al.[15] later analyzed data in a follow-up study of miners and ex-miners. Although smoking was not of prime importance in their study, they did note that, whereas ex-smokers had a greater response to coal mine dust in terms of category 0/1 or greater, when category 2/1 or greater was studied, no difference between the smoking groups was discernable.

Figure 2 shows the prevalences of category 1/0 or greater as reported by the single reader at round 1 of the NSCWP, plotted against estimated dust exposure for smokers and non-smokers. Note that the trends for the two smoking groups are all similar, and that there is no evidence of a potentiating effect between smoking and dust exposure. This observation was confirmed in a logistic analysis based on presence or absence of category 0/1 or greater small rounded opacities, and using exposure, age, and smoking as predictor variables. No association between smoking and prevalence was seen (P=0.49). These results therefore support those reported by the PFR.

Smoking, Phlegm Production and Small Rounded Opacities

Jacobsen et al.[14] not only examined the influence of smoking on CWP development, but were also concerned with the role of phlegm production, one suggestion being that smoking caused phlegm production, which then either aided the removal of dust, or had the opposite effect of increasing its deposition. They devised four hypotheses and then went on to test them using various analyses of their data.

They found that, in two of the three analyses undertaken (both on the same subset), the prevalence and attack rate of pneumoconiosis was elevated in those miners reporting bplegm production. However, this elevation was minor and was seen in both smokers and non-smokers equally. Hence the authors' conclusion that dust exposure remains the prime determinant of CWP occurrence remained valid.

Irregular Opacities

As noted earlier in this report, nearly all of the research on radiological abnormalities and dust exposure (excepting massive fibrosis (PMF)) in the PFR has concentrated on small round opacities. This is in contrast to the U.S. situation, where most reports on prevalence have dealt with determinations of combined opacities which include both rounded and irregular opacity readings. However, as noted by Dick et al.,[9] the PFR researchers have taken increasing interest in the past few years in the causation of irregular opacities on chest radiographs of workers exposed to various dusts, including coal dust.

In order to explore the relationship between dust exposure and prevalence of small irregular opacities, Dick and colleagues obtained readings on 900 films of miners. These films were divided into groups depending upon whether predominantly small rounded, predominantly small irregular, or no opacities were seen. The readings from the second group were then correlated with dust exposure, smoking habits, and age, using a logistic model. The results showed that increasing exposure to coal mine dust was related to prevalence of irregular opacities. However, in addition to dust exposure, age had a very strong relationship to prevalence ($p < .001$). While there was a trend in prevalence with smoking status (non-smokers to ex-smokers to

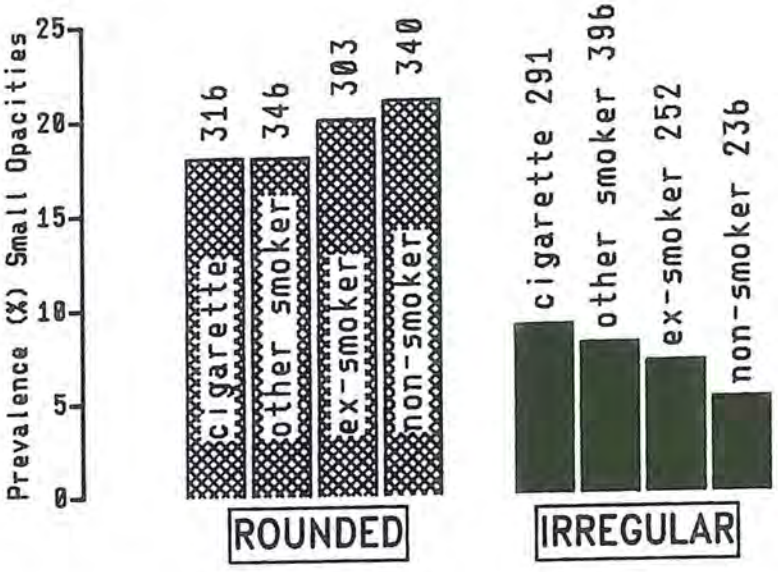

Figure 3. Prevalence of small rounded and small irregular opacities for four smoking groups. British data. (Data from Dick et al.[9])

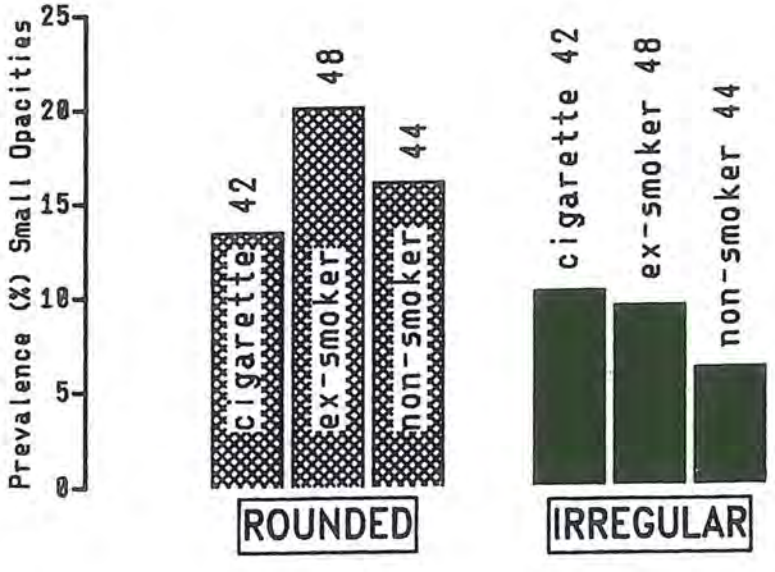

Figure 4. Prevalence of small rounded and small irregular opacities for three smoking groups. U.S. data.

other smokers to cigarette smokers), Dick et al. found that the apparently more frequent occurrences of these signs in cigarette smokers could have arisen by chance ($p < 0.22$).

Figure 3 of this report summarizes some of the information from Dick et al.[9] Based on Table 5 of that report, it shows the prevalence of small rounded and small irregular opacities by smoking group. The data confirm the view that cigarette smokers are not at increased risk of developing small rounded opacities.

A slight trend of increased prevalence of irregular opacities with smoking status is seen, but at least part of this could be explained by dust exposure (mean values shown above bars). An analysis of irregular opacities for the U.S. miner group showed a similar pattern (Figure 4). The results for small rounded opacities are shown for comparison. Again, there is no indication that smokers suffer from greater prevalences of small rounded opacities due to their smoking. In contrast, smokers are seen to have a greater prevalence of irregular opacities than Do non-smokers, with ex-smokers lying in between.

A logistic analysis based on category 0/1 or greater small irregular opacities indicated a significant contribution by smoking to prevalence, after allowance for age and exposure ($P < 0.0001$). This observation is therefore in agreement with the trend seen for the PFR data, even though that association was not statistically significant.

Progressive Massive Fibrosis

Little published information exists on this topic, but that which does indicates no influence of smoking on the development of PMF. These results are reported in Hurley et al.[16] They are drawn from a case-control study in which the cases were miners who experienced an attack of PMF over a certain year period, while controls were selected from those who did not demonstrate such an attack, matching being done on mine worked and prior dust exposure. Matching was not done on age and simple CWP status. Smoking was one of a number of variables studied for possible influence on PMF development.

The study demonstrated the importance of a number of factors on PMF attacks, such as simple CWP, age, and weight/height index, but smoking was not one of them. The authors concluded that the effect of smoking was generally negligible, although there was the suggestion that South Wales cases had relatively fewer smokers than did their respective controls.

Respiratory symptoms

The relationship between increasing prevalence of symptoms of chronic bronchitits and measured dust exposure was first demonstrated in coal miners by Rae et al.[5] These researchers derived indices of chronic bronchitis, using answers to questions on chronic cough and phlegm. These indicators of chronic bronchitis were then tabulated by smoking group and dust exposure category, and were given in Table IV of that report. Statistically significant associations between prevalence and dust exposure were found for non-smokers aged 25-34, and both smokers and non-smokers aged 35-44. No associations were detected in the older age groups.

Figure 5 of this report shows the Rae et al. relationships for the 35-44 age group. While a trend of prevalence with exposure is evident in both smoking groups, there is no sign of a potentiating effect of smoking on dust exposure, as the lines are mostly parallel.

Figure 6 presents the results of a similar analysis undertaken on miners aged 35 - 44 in the NSCWP. Chronic bronchitis was defined similarly through use of answers on cough and phlegm, the prevalences thus obtained being tabulated against estimated exposures. Again it is clear that the lines for the two smoking groups are

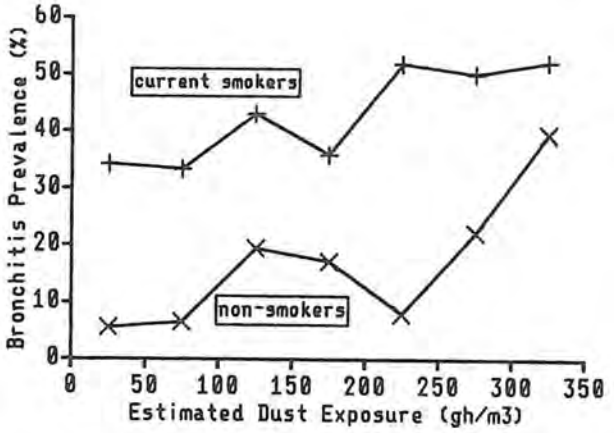

Figure 5. Relationship of bronchitic symptoms to dust exposure for two smoking groups. British data. (Data from Rae et al.[5])

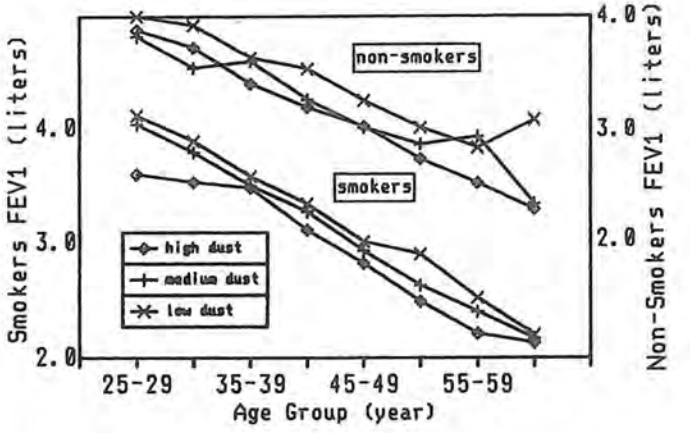

Figure 7. Relationship of FEV_1 to dust exposure for two smoking groups. British data. (non-smokers data displaced vertically one liter for clarity) (Data from Rogan et al.[3])

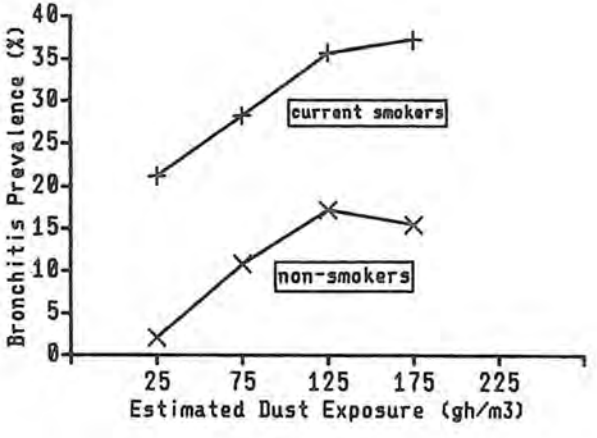

Figure 6. Relationship of bronchitic symptoms to dust exposure for two smoking groups. U.S. data.

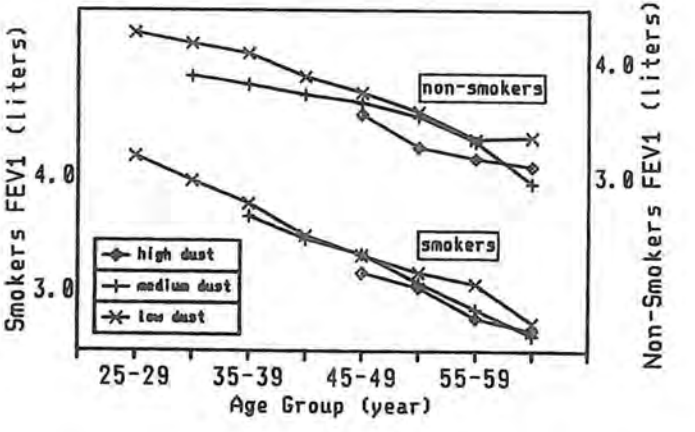

Figure 8. Relationship of FEV_1 to dust exposure for two smoking groups. U.S. data. (non-smokers data displaced vertically one liter for clarity)

almost parallel. As there were no confounding factors involved in this analysis no formal analysis was necessary. The results confirm those of the PFR, and indicate that smoking does not appear to aggravate the effects of dust exposure in giving rise to reports of respiratory symptoms.

Ventilatory Function

The first paper to demonstrate a clear relationship directly between dust exposure and FEV_1 was that of Rogan et al.[3] Data from Table I of their report have been extracted and are presented in Figure 7. Note the obvious effects of age and smoking. Note also the clear relationship of FEV_1 to dust exposure, and particularly the lack of potentiating effect between smoking and dust exposure. If anything, the opposite situation seems to exist,as the effect of dust appears greater in the non-smokers.

Rogan et al. estimated that dust exposure was related to a reduction in FEV_1 of about 0.0006 liters per gram hour/cubic meter (gh/m^3), when averaged over both smoking groups. Subsequently, Jacobsen reported on estimates of the dust effect for each smoking group separately,[17] these being 0.00069 and 0.00055 liters reduction per gh/m^3 for non-smokers and smokers respectively. More recently, in their study of miners and ex-miners, Soutar and Hurley[18] have provided confirmatory evidence on the lack of potentiation caused by smoking. In this analysis, one estimate of the dust exposure effect was .00090 liters reduction in FEV per gh/m^3 for non-smokers, and 0.00065 for smokers (Table 3 of their report). Thus, again,no excess dec line with dust exposure is seen for smokers over that for non-smokers.

A similar analysis to that of Rogan et al.[3] was undertaken using estimated dust exposures derived as described by Attfield and Morring[13] for U.S. miners. The results are shown in Figure 8. Again note the expected age and smoking effects, but particularly the trend with dust exposure and its lack of potentiation with smoking status.

To test for a dust/smoking interaction, a simple regression model on FEV_1 was used which contained terms for age, smoking, age x smoking, height, exposure, and, of course, a dust x smoking interaction. (Mine and regional effects were also taken into account, but their presence did not affect the overall conclusions).

The model revealed an estimated overall effect of dust exposure on reduction in FEV_1 of 0.00036 ml per gh/m^3 ($p<0.05$). For smokers,the effect of dust exposure on reduction in FEV_1 was 0.00029 ml per gh/m^3, while for non-smokers it was 0.00052, the probability being large that the difference in the two coefficients arose by chance (P=.45). A similar result was obtained using years underground as a surrogate for exposure. The results confirm those from the PFR, indicating that smoking does not exacerbate the effect of dust exposure on ventilatory function.

COMMENT AND CONCLUSION

Three different measures of respiratory impairment have been considered here, and in each case no obvious sign was seen that smoking exacerbated the effects of dust exposure. In fact, the opposite tendency was apparent, for there were some indications that non-smokers evidenced more severe effects of dust than did smokers. The main finding, then, from this report is that reduction in lung disease must be sought through parallel reductions in exposures to both coal mine dust and tobacco smoke. Reduction in one without the other can only achieve partial success.

Note that the effects reported here for the NSCWP cohort refer to miners examined in the early 1970s. These effects developed during a time when dust exposures were considerably greater than those mandated today. Evidence on this is given by Jacob-

son,[12] where dust concentrations for face jobs were often 6 mg/m^3 or higher. In contrast, the current federal limit on dust is 2 mg/m^3, while the provision for excess quartz often requires even lower levels.

As a result of this drop in dust levels, the amount of respiratory disease suffered by U.S. coal miners should be considerably less in the future than that seen in the past. As a consequence, the question of interactions between dust exposure and smoking will become of progressively less importance in this country, although the minimization of exposure to both hazards must remain a desirable objective. However, while this is true for U.S. miners, there are many other miners in other parts of the world who receive higher dust exposures. For these, both dust control and reduction in smoking must remain high priority aims.

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