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Respiratory effects of borax dust

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ABSTRACT The relation of respiratory symptoms, pulmonary function, and abnormalities of chest radiographs to estimated exposures of borax dust has been investigated in a cross sectional study of 629 actively employed borax workers. Ninety three per cent of the eligible workers participated in the study and exposures ranged from 1·1 mg/m^3 to 14·6 mg/m^3. Symptoms of acute respiratory irritation such as dryness of the mouth, nose, or throat, dry cough, nose bleeds, sore throat, productive cough, shortness of breath, and chest tightness were related to exposures of 4·0 mg/m^3 or more, and were infrequent at exposures of 1·1 mg/m^3. Symptoms of persistent respiratory irritation meeting the definition of chronic simple bronchitis were related to exposure among non-smokers. Decrements in the FEV_1 as a percentage of predicted were seen among smokers who had heavy cumulative borax exposures ($\geqslant$80 mg/m^3 years) but were not seen among less exposed smokers or among non-smokers. Radiographic abnormalities were uncommon and were not related to dust exposure. Borax dust appears to act as a simple respiratory irritant and perhaps causes small changes in the FEV_1 among smokers who are heavily exposed.

Borax is an alkaline salt which causes irritation of the respiratory tract among exposed workers.[1] The intensity and duration of exposure necessary to cause irritation have not been documented, although nose bleeds, sore throats, and eye irritation occur under the working conditions common in the borax mining and refining industry. There are an estimated 420 000 workers in the United States in various segments of industry who have potential occupational exposure to borax.[2] This investigation was undertaken to determine the frequency and severity of respiratory disease that develops as a result of breathing borax dust and to define the dose-response relation.

This investigation was performed in a large borax mining and refining plant in which borax ore is recovered from a large open pit mine. The ore is crushed and dissolved with steam, insolubles are removed by sedimentation, and the resulting borax solution is crystallised, washed, and kiln dried. The borax produced in this process contains either five ($Na_2B_4O_7 \times 5H_2O$) or 10 moles of water of hydration ($Na_2B_4O_7 \times 10H_2O$), depending on the temperature at which the crystallisation process is carried out. Anhydrous borax ($Na_2B_4O_7$) is also produced by heating either of the hydrated forms to a molten glass, and then cooling and pulverising the glass to a fine powder.

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Methods

STUDY POPULATION

The study population consisted of all employees actively employed for five years or more and those workers presently employed in the areas where borax is crystallised and dried or made into anhydrous borax regardless of the duration of employment. Of those eligible, 629 (93%) participated in the study. The participation was 95·4% among hourly workers, 93·6% among salaried workers, and 63·5% among workers on long term disability.

INTERVIEW

The American Thoracic Society Epidemiology Standardisation Project questionnaire on respiratory disease[3] was administered by interview to the 629 study participants to obtain information on persistent respiratory symptoms and the history of smoking. Additional questions about acute irritation of the eyes and respiratory tract were asked, and a history of past exposure to respiratory hazards was also obtained. Subjects were instructed to take a deep breath and cough hard; the interviewer judged the cough to be loose (productive) or dry (unproductive) by its sound, using the method of Gandevia.[4]

EXPOSURE EVALUATION

Occupational histories were obtained by reviewing each participant's personnel file. The job title, plant assignment, and starting and stopping dates to the nearest month were recorded for each job held and the employment history before entering the borax industry was abstracted.

The results of industrial hygiene surveys made by the company and by the Mine Safety and Health Administration (MSHA) between 1977 and 1981 were used to estimate the exposures in the mine and refinery. Most of the data came from the company. Sampling data were available on 37 jobs from six areas of the facility. Total particulate was collected in closed face filter cassettes attached to the worker's collar, using PVC membrane filters with a 5 micron diameter pore size. A total of 103 full shift total dust samples were collected and weighed. In most areas of the plant the dust was almost entirely borax, except for trace quantities of other soluble minerals. In the mine other mineral compounds derived from shale were present in addition to the borate ores. There were no asbestiform minerals and silica did not exceed 1% of any sample.

The dust samples were used to derive two separate measures of exposure, the mean particulate exposure in each plant area and the particulate exposure in each job. The mean exposure in each plant area was estimated by a weighted average of the dust levels measured in all jobs in the area. The weight given to each job was proportional to the number of subjects who had ever worked in that job. The areas and their weighted mean dust levels are presented in table 1. The particulate exposure in each job was determined by making qualitative estimates of the exposures in each job and then grouping the jobs with others that had similar estimates. The mean dust level for each of these groups was next calculated from the dust measurements which had been made on jobs within the group, and this dust level was then assigned to each job. Three groups were defined: low exposure (0·9 mg/m^3 total particulate), medium exposure (4·5 mg/m^3 total particulate), and high exposure (14·6 mg/m^3 total particulate).

Table 1 *Mean dust exposures in plant areas*

	Weighted Mean total particulate (mg/m^3)
Anhydrous borax production	14·6
Shipping department	8·4
Mine, maintenance shops, other production areas	4·0
Accounting, laboratory, safety department, other non-production areas	1·1

PULMONARY FUNCTION TESTS

Spirometry was performed on an Ohio 840 rolling seal spirometer wired to an analogue processor and tape recorder. Six hundred and twenty subjects participated in spirometry (nine refused) and each completed a minimum of five blows, yielding at least three blows of acceptable quality. Ninety eight per cent of the participants met the ATS criteria for acceptable spirometry.[3] For data analysis the subject's largest FVC, FEV_1, peak flow rate ($\dot{V}max$), and FEF_{75} were used after correction to standard temperature and pressure.

CHEST RADIOGRAPHS

A single standard 72 inch posteroanterior chest radiograph was taken for each subject (621 participated and eight refused or were excused because of pregnancy). Each radiograph was classified according to the ILO International Classification of Radiographs of Pneumoconioses (1980)[5] by three NIOSH certified "B" readers. The three readers classified the radiographs independently and the results were combined. Opacities were judged to be present if the average was 1/0 or greater. Pleural thickening was judged to be present if it was identified by at least two of the three readers. All radiographs were of acceptable quality for interpretation.

STATISTICAL ANALYSIS

Each acute respiratory tract symptom was analysed by determining the proportion of participants ever having worked in a given area who had experienced the symptom in that work area. In order to assess whether the proportion of participants noting symptoms increased with exposure level, a test for trend in proportions was performed.[6]

For each persistent respiratory symptom we calculated age standardised risk estimates by the indirect method in each of six smoking exposure categories. The risk for each exposure smoking level was obtained by dividing the observed number of subjects with the symptom (or condition) by the sum over all age groups of the expected numbers in each age group. These age specific expected numbers were derived by multiplying the age specific rate in the total study population by the number of subjects at risk in the age smoking exposure category. Five age categories (< 25, 25–34, 35–44, 45–54, ≥ 55) and two smoking categories (ever smokers and never smokers) were used. For persistent respiratory symptoms, exposure was defined on the basis of the exposure level (low, medium, or high) of the job held at the time of the interview. Relative risks for each smoking exposure category were estimated using non-smokers at low exposure as the referent group. Tests for trend across exposure levels in each smoking category were performed by adapting the method of Mantel[7] to accommodate adjustment for age. All analyses were performed on the DEC PDP-11-45 computer.

Stepwise multiple regression techniques were used to analyse the relation between the pulmonary function measurements (FEV_1, FVC, FEV_1/FVC, $\dot{V}$max, and FEF_{75}) and age, height, smoking status (ever/never), years smoked, and various indicators of exposure (years actively employed, cumulative total particulate, and years in various job groups). In addition, the ratios of observed to predicted FEV_1 and FVC were calculated using the equations of Knudson *et al*[8] to obtain predicted values. These values, as percentages of predicted (PPFVC and $PPFEV_1$), were analysed in relation to categories of exposure by comparing means between the categories. The analyses of the pulmonary function measurements were restricted to white men because the number of women and non-white men was not large enough to permit us to derive separate regression equations for them.

t Tests were performed to determine whether participants with radiographic abnormalities differed from those with normal chest radiographs in terms of age, years smoked, and particulate exposure in the borax industry. Odds ratios, relating the presence or absence of an abnormality to previous hazardous exposures, were evaluated using the Mantel-Haenszel test.[7] All analyses were performed on the DEC PDP-11-45 computer. Multiple regression and *t* test procedures were implemented using the SPSS batch system.[9]

Results

The study participants were predominantly white men (92%). They had an average age of 40·2 years and had worked in the borax industry for an average of 11·4 years (table 2). Roughly a quarter of the participants had never smoked, half were present smokers, and a quarter were ex-smokers.

ACUTE SYMPTOMS

Acute respiratory tract symptoms were analysed in relation to the total particulate exposure in each of the four exposure categories shown in table 1. The proportions of workers who had ever worked in each borax exposure category who noted symptoms at that exposure level are presented in the figure. Thirty three per cent of the workers noted dryness of the mouth, nose, or throat and 28% noted eye irritation at exposures of 14·6 mg/m^3. Nose bleeds and dry cough were noted by 15% and sore throats and productive cough were noted by 8% at this exposure. Shortness of breath and chest tightness were noted by 5%, whereas chest pain and haemoptysis were noted by less than 2%. Based on a test for linear trend, the proportion of workers noting each symptom increased as exposure increased, with the exception of chest pain and haemoptysis. At an exposure level of 4·0 mg/m^3 no symptom except eye irritation was noted by more than 5% of the exposed participants. At an exposure level of 1·1 mg/m^3, no symptom was noted by more than 3% of the exposed participants.

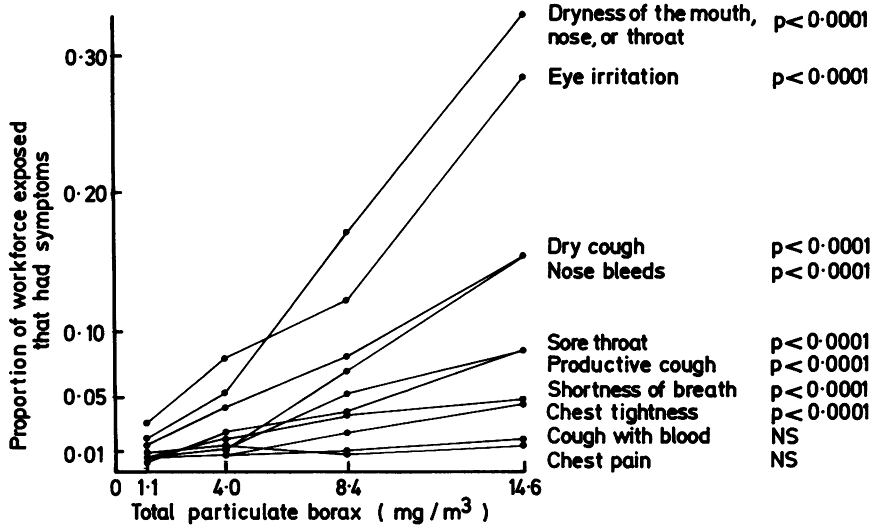
PERSISTENT SYMPTOMS

For each persistent symptom, the risk of developing the symptom was estimated separately for ever and never smokers in relation to the exposure level of the job at the time of interview by indirect standardisation to the age specific rates of the total study population. Individuals on disability were excluded from this analysis because they were not presently working. The results are presented in table 3 for never smokers and in table 4 for past and present smokers. For each exposure level and smoking status, the risk of developing a symptom is presented relative to that of never smokers in low exposure jobs.

Never smokers—The risks of cough, phlegm production, and chronic bronchitis (defined as a productive cough present on most days at least three months of the year for at least two consecutive years) increased with increasing dust exposure. At exposures of 14·6 mg/m^3, the risk of cough was 1·4 times that at 0·9 mg/m^3; however, this increase was not statistically significant. At exposures of 14·6 mg/m^3 the risk of phlegm production was 1·85 and of chronic bronchitis 5·9 times the risk at 0·9 mg/m^3. There was a statistically significant dose-response relation for both of these symptoms. At exposures of 14·6 mg/m^3 the risk of objective cough, judged to be loose (productive) by

Table 2 *Characteristics of study participants*

	No	*%*
White men	576	92
Other men	27	4
Women	26	4
Total	629	100
	Mean	*SD*
Age (years)	40·2	11·5
Height (cm), white men only	176·8	6·6
Education (years)	12·5	5·6
Years employed in borax industry	11·4	8·1
Distribution of duration of employment in borax industry (years)	*No*	*%*
<5	112	18
5–9	238	38
10–14	122	19
15–19	42	7
20–24	67	11
25–29	26	4
≥30	22	3



Relation of acute respiratory symptoms to particulate borax concentration.

the interviewer, was 1·7 times the risk at exposures of 0·9 mg/m^3, and the trend was statistically significant. Breathlessness did not show a clear relation to exposure. The risk of wheezing decreased as exposure increased but not significantly.

Ever smokers—The risk of breathlessness increased significantly with exposure, but the risks of other symptoms did not. All the symptoms were more common among the ever smokers than among the never smokers.

PULMONARY FUNCTION

The regression analyses of the FVC, FEV$_1$, FEV$_1$/FVC, $\dot{V}$max, and FEF$_{75}$ showed that none of these measures was statistically significantly related to borax exposure.

The PPFVC and PPFEV$_1$ (FVC and FEV$_1$ as percentages of predicted) were analysed by grouping subjects into two exposure categories based on cumulative particulate exposure (<80 mg/m^3 years or ≥80 mg/m^3 years). Among never smokers there was no significant difference in the mean PPFVC or mean PPFEV$_1$ between exposure categories. Among ever smokers the mean PPFEV$_1$ was statistically significantly lower in the high exposure group than in the low exposure group for those subjects who had smoked less than 25 years (table 5). Among individuals who had smoked for at least 25 years the mean

Table 3 *Prevalence of persistent symptoms according to exposure level of job at time of interview: never smokers*

Symptom	*Exposure level (total particulate in mg/m³)* Low (0·9)	Medium (4·5)	High (14·6)	*Test for trend 1 sided p value*
	No of workers affected/No of workers in group			
Cough	7/57	6/65	6/37	
Phlegm	5/57	6/65	5/37	
Chronic bronchitis*	1/57	3/65	3/37	
Wheezing	14/57	10/65	4/37	
Breathlessness†	8/57	6/61	5/36	
Loose objective cough	3/57	2/65	2/37	
		Risk ratio‡		
Cough	1·0	0·76	1·40	NS§
Phlegm	1·0	1·09	1·85	0·01
Chronic bronchitis*	1·0	2·82	5·91	0·006
Wheezing	1·0	0·63	0·46	NS
Breathlessness†	1·0	0·75	1·27	NS
Loose objective cough	1·0	0·67	1·71	0·03

*Defined as a productive cough on most days at least three months of the year for at least two consecutive years.
†Excluding subjects who were disabled from walking by conditions other than heart or lung disease.
‡Indirectly standardised to never smokers at low exposure.
§Not significant, p > 0·10.

Table 4 *Prevalence of persistent symptoms according to exposure level of job at time of interview: ever smokers*

Symptom	*Exposure level (total particulate in mg/m³)*			*Test for trend 1 sided p value*
	Low (0·9)	*Medium (4·5)*	*High (14·6)*	
	No of workers affected/No of workers in group			
Cough	39/158	70/211	16/67	
Phlegm	46/158	74/211	21/67	
Chronic bronchitis*	25/158	46/211	11/67	
Wheezing	40/158	56/211	16/67	
Breathlessness†	40/154	59/204	23/65	
Loose objective cough	18/158	32/211	11/67	
		Risk ratio‡		
Cough	1·98	2·78	2·00	NS§
Phlegm	3·24	3·97	3·67	NS
Chronic bronchitis*	9·00	13·45	10·27	NS
Wheezing	1·02	1·12	0·99	NS
Breathlessness†	1·82	2·15	2·78	0·04
Loose objective cough	2·10	3·21	3·64	0·09

*Defined as a productive cough on most days at least three months of the year for at least two consecutive years.
†Excluding subjects who were disabled from walking by conditions other than heart or lung disease.
‡Indirectly standardised to never smokers at low exposure.
§Not significant, $p > 0.10$.

$PPFEV_1$ was lower in the more heavily exposed, but the difference in means did not achieve statistical significance. The mean PPFVC did not show a significant relation to exposure among ever smokers.

CHEST RADIOGRAPHS

Small linear opacities of profusion 1/0 or greater were present in 17 participants' radiographs (2·7%). Sixteen of these individuals were smokers. The duration of cigarette smoking was significantly associated with linear opacities ($p < 0.001$), as was previous employment in cotton mills (odds ratio = 9·9, p = 0·001). There were no significant relations between the measures of borax exposure and linear opacities. Previous employment in other dusty occupations, including exposure to asbestos, was not significantly related to linear opacities.

Table 5 *Analysis of FEV_1 as a percentage of predicted by cumulative total particulate exposure; 422 white male ever smokers*

Cumulative total particulate	*Years smoked*		
	<10	*10–25*	*≥25*
<80 mg/m³ years			
Mean FEV_1 as a percentage of predicted	105·3	99·8	88·7
No	80	137	71
Mean years smoked	5·6	16·1	34·3
Mean age	29·6	36·4	51·5
Mean height (cm)	178	177	175
≥80 mg/m³ years			
Mean FEV_1 as a percentage of predicted	90·8	96·4	86·7
No	9	66	59
Mean years smoked	4·3	17·5	33·4
Mean age	44·0	42·8	52·0
Mean height (cm)	174	177	176
t test for difference between means of FEV_1 as a percentage of predicted	$p < 0.025$	$p < 0.05$	$p < 0.30$

Small round opacities of profusion 1/0 or greater were present in four of the participants' radiographs (0·6%), all of whom were smokers. These subjects had worked more than twice as long at medium exposure as subjects without opacities. A review of their work histories, however, did not show any consistent pattern of exposure. Two had been employed predominantly in the truck repair shop, one in the electrical shop, and one in the mine and shipping department. None of the subjects who had round opacities had been employed previously in other dusty occupations.

Pleural thickening was present in 16 radiographs (2·6%) and was not associated with any exposure variable except years employed at medium exposure, with which it was negatively associated. Thirteen of these subjects had positive smoking histories and pleural thickening was significantly associated with the duration of cigarette smoking ($p < 0.001$). It was also significantly associated with previous asbestos exposure (odds ratio = 4·2, p = 0·036).

Discussion

The results indicate that borax dust is an acute respiratory irritant at exposures of 4·4 mg/m³ or more. Persistent respiratory irritation, evidenced by chronic bronchitis and objectively measured productive cough, is also related to exposures above this level. The measures of pulmonary function and chest radiographs do not show a clear pattern of abnormalities, although a small decrement in FEV_1 was detected among smokers.

This pattern of respiratory irritation, with no clear physiological impairment, indicates that borax probably acts as a simple respiratory irritant which produces chronic simple bronchitis but probably no significant reductions in ventilatory function, at least over the range of exposures we have studied. Negative conclusions about respiratory impairment cannot be made with complete confidence in this case, however, because a cross sectional study such as this would be unlikely to include workers who were impaired by exposure and who would have left employment.

The dose response relation we have presented depends on the accuracy of the dust measurements and on unbiased reporting of symptoms by the participants. Although we did not measure the dust ourselves, we reviewed the written descriptions of the workers' activities that were given by the industrial hygienist at the time of the measurements, and we selected only those that reflected routine circumstances of exposure. We believe that these measurements are an unbiased estimate of the true exposures of the workers.

We took considerable precautions against biased reporting of symptoms. Our interviewers had no knowledge of the subjects' exposure histories at the time of the interviews. The exposure histories were later derived from the personnel files and the dust measurements, both of which were recorded before the conduct of the study. We also attempted to evaluate objectively the presence of productive cough by having the interviewer rate the cough according to its sound. This was done at the start of the interview, before the subjective report of cough and phlegm was obtained. A dose-response relation was shown.

Although the subjects could have exaggerated their symptoms, a general overreporting of symptoms would not have produced a dose-response relation. Possibly individual workers appreciated the significance of the questions asked and recognised that they had higher than average risk because of higher than average exposures. Although we cannot exclude this possibility, there is reason to believe its effect is small, if any. In tables 3 and 4 there is good agreement between the risks of phlegm (reported by the subject) and loose objective cough (reported by the interviewer) over all levels of exposure regardless of smoking status. The agreement between two measures of phlegm production indicates that the workers had no greater tendency to associate phlegm with heavy exposure than did the interviewers. The interviewers, once again, had no knowledge of the subjects' exposures.

Other reports have indicated effects of borax on the respiratory tract similar to those we report. The National Institute for Occupational Safety and Health evaluated nine workers exposed to borax dust at concentrations of 2·9 to 29·9 mg/m^3 in 1977. Complaints of eye irritation, nose bleeds, and sore throat were noted.[1] Birmingham and Key, in an evaluation in 1963 of the same plant that we studied, reported complaints of nose bleeds, nasal irritation, cough, shortness of breath, and dermatitis.[10] Air concentrations of dust were not reported, but the levels were high enough to interfere with normal visibility. The California State Department of Public Health, which performed a respiratory disease survey of 629 borax workers in 1963, showed an excess of episodes of respiratory illness among workers exposed to anhydrous borax when compared with non-exposed workers.[11] Kasparov reported that examinations of workers exposed to borax aerosol concentrations ranging up to 31 mg/m^3 showed atrophic and subatrophic changes in the respiratory mucous membranes.[12]

The American Conference of Governmental Industrial Hygienists (ACGIH) has recommended exposure limits of 1·0 mg/m^3 for anhydrous borax and borax pentahydrate and 5·0 mg/m^3 for borax decahydrate, which recognise the potential these dusts have for causing acute respiratory irritation.[13] The exposure limit is lower for anhydrous borax and borax pentahydrate than for borax decahydrate because the irritant properties of borax are thought to increase with decreasing water of hydration due to the exothermic effect of hydration.

The major value of our study derives from the use of environmental information to show the exposure levels at which respiratory irritation occurs and the level at which irritation is uncommon. Additional information is needed to define the minimum duration of exposure necessary to produce irritation.

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