FILE NAME: CERAMICS (CER)

DATE: 1949 July

DOC#: CER042

DOCUMENT DESCRIPTION: Journal Article - Respiratory Disease: An Industrial Hazard

MIOLOGICAL NOTES.

28th May, 1949.

gistered and Deaths.*

K ENDING

May 20 †		May 28	
—	—	—	—
—	—	—	—
3	—	6	—
—	—	—	—
31	—	57	—
11	—	12	—
6	—	7	—
13	—	14	—
2	—	1	—
—	—	1	—
98	*1*	83	*12*
—	*1*	2	—
—	—	2	—
14	—	21	—
70	*19*	72	*25*
11	*3*	10	*5*
244	—	263	—
8	—	11	—
141	—	111	*1*
84	—	53	—
—	—	1	—
—	—	—	—
22·8		21.7	
11·8		12·8	
8		7	
1		1	
16		22	
2		—	

† Incomplete week.

noted last month still continues
four weeks ending 28th May
ere were no deaths and neither
ne disease continuing to be less

e again lower than the previous
this period a year ago.
a level although the number of
s of the disease and 14 against

measles is still prevalent to a
npared with 218 in 1948. There
comparative figures being 546

eath rate, the latter being 12.5
ly 79 deaths of children in the
period a year ago, part of this
in the deaths from diarrhoea

GLASGOW MEDICAL JOURNAL

VOLUME 30 JULY 1949 No. 7

THE JOURNAL OF THE ROYAL MEDICO-CHIRURGICAL SOCIETY OF GLASGOW

RESPIRATORY DISEASE: AN INDUSTRIAL HAZARD.*

A. T. DOIG, M.D., D.P.H.

H.M. Medical Inspector of Factories.

Until comparatively recently, the term respiratory dust disease was commonly thought of as comprising little more than the two well known diseases, silicosis and asbestosis. In the last decade, however, there has been a general appreciation of the fact that the field is very considerably wider. Dusts in industry may be classed quite simply in accordance with their origin, animal, vegetable or mineral. Their actions however are much more varied and complex. Animal dusts are generally innocuous; occasionally they may cause sensitization effects, and they may also carry infection. Vegetable dusts too are usually of low pathogenicity but they are more liable than animal dusts to cause pulmonary trouble. They may produce acute sensitization effects, or acute or semi-acute lung irritation and lung infection, or chronic broncho-pulmonary disease. Mineral dusts are the most important group as regards industrial respiratory disease. Some of them are locally caustic, producing irritation of the upper respiratory tract even to ulceration and perforation of the nasal septum; others irritate the bronchi or the lung parenchyma producing atypical forms of pneumonia, and some have long term effects resulting in chronic changes in the lungs—diffuse in the case of asbestos, focal in the case of silica. Some have a carcinogenic action, some produce granulomata, and others appear to be able to lie quite inertly for long periods in the lung tissues without producing any permanent reaction by the tissues.

MINERAL DUSTS.

The Pneumoconioses. Pneumoconiosis means 'dust in the lungs' and does not necessarily imply either organic lung change or disability. A distinction might be drawn between the fibrosis-producing dusts and the

* Based on a lecture delivered to the Royal Medico-Chirurgical Society of Glasgow on 25th February, 1949.

tion produced by the latter as benign
a legal definition of pneumoconiosis
t, 1943, and the National Insurance
neumoconiosis is therein defined as
ist, asbestos dust or other dust, and
known as dust-reticulation.' This
-down of fibrous tissue in the lung,
from the inhalation of dust and so
to the inert dusts.
upational dust disease been known is
crates and Pliny and Elder mention
ifrequent in the middle ages but the
ve been attained in the early 19th
Revolution people were employed
dreadful conditions. Hygiene was
cheap. The average age at death of
rs ago was 32 years and the Sheffield
old grinder over 30 years of age was
benezer Elliott, the Rotherham poet,

his uneasy breath,
leadly trade he bends,
ars not man nor death,
- he earns he spends,
- bosom friends.'

rty, meets his doom.'
pulmonary disease have changed in
able period after 1918 attention was
tos coming into prominence alongside
ke, Merewether, Gloyne, and others.
osis is superfluous here. The disease
l has been discussed so often that its
silica dust inhalation, if the quantity
ize of the particles is small enough,
ture. Nodules formed of dense fibrous
nt of the fibres are characteristic but
ay be altered profoundly by accom-
other dusts which modify its action.
est has been raised in regard to dusts
silica. The present theory as to the
nd up with its solubility in the lung
acid. If this theory is correct sub-
lity of silica might be expected to

diminish its activity in the lungs and this has been found to be so. Kettle in 1932 found that particles of silica which were coated with fine iron particles became relatively inert, and later in Canada in 1937 Denny, Robson and Irwin showed that this property of diminishing the solubility and therefore the activity of silica was held by finely divided metallic aluminium. In their experiments they showed that even the admixture of 1% of aluminium in quartz prevented the formation of nodular fibrosis in the lungs of experimental animals. This work has been confirmed by other investigators and it is now generally accepted that in animals at least, finely powdered aluminium (or aluminium hydroxide) has a very marked action in reducing toxicity. Naturally trials have been made in men—both in normal men who are exposed to a silica risk, to try to prevent silicosis, and in sufferers from silicosis in order to try to alleviate the condition. It will be readily understood that, in regard to a disease which may take 20 years or so to develop a similar period or longer may be required to ascertain the value of any preventive measure. Controlled trials are being made in this country and abroad but it is too early to speak of results.

The introduction of aluminium dust in treatment was followed by a wave of enthusiasm—based entirely on the patient's own story of subjective improvement. At present, this wave of enthusiasm seems to be abating somewhat. It seems rather a strain on the imagination to picture the resolution of fibrotic nodules and emphysema by the inhalation of more dust. Furthermore before advocating widespread use of the inhalation of aluminium in man it is necessary to ascertain that at least it will not do any harm—and about this there is no certainty. Experiments in animals have suggested that aluminium may favour the development of tuberculosis in the lungs, and several reports of lung damage from aluminium itself have been reported, *e.g.*, from Germany. Recently a type of pneumoconiosis has been reported in certain furnace workers exposed to volatilized fumes of alumina (Shafer & Riddell, 1947). An unusual feature was a liability to spontaneous pneumothorax, although this had already been noted by some of the German workers (Goralewski, 1940 & 1941 ; Goralewski & Jager, 1941 ; Jager & Jager, 1941 ; Jotten & Eickhoff, 1942).

A further interesting observation about aluminium was made recently by Meiklejohn and Jones (1948). Pottery used to be bedded in powdered flint during its firing but this dangerous practice has now been prohibited by law. Meiklejohn and Jones report having examined 52 workmen who had used flint for many years and thereafter alumina also for several years. Although the amount of alumina that these men inhaled daily was much greater than could possibly be inhaled by existing methods of aluminium treatment, not only did known cases of silicosis advance but new cases occurred. This does not of course condemn aluminium

ne forms of alumina have very much
ier forms, and it merely shows that
undum) had little or no effect.

and Coal Trimmers. For recent work
he sociological effects the numerous
studied, although it is interesting to
spiratory disease in coal miners came
hall, 1834 ; Thomson, 1836). Towards
eginning of this century no attention
ondition and such authorities as Sir
olls (1915) stated that the disease no
obscurity and complacency—at least
.- themselves As late as 1923, Hope,
mining is not unhealthy. If it were
ents it might be reckoned amongst
nsation was payable to coal miners
and then only to workers who could
ilica rock. In 1934 the scheme was
on the coal face and other under-
lissatisfaction continued. Increasing
inty as to the nature of the disease
il to make an investigation which
e published in 1942, 1943, and 1945.
radiological abnormalities associated
f function in coal face workers, and
trimmers. This was interesting from
nereas it could be argued that the coal
ght inhale not only coal dust, but also
other workmen, this could not be so
who were exposed to dense clouds of
ot to rock dust. The Medical Research
appearance in abnormal radiographs
ip and lacelike in appearance, more
reticulation, and which was clearly
dition was subsequently made com-
mpensation Act, 1943, which defined
lungs due to silica dust, asbestos dust,
condition of the lungs known as dust

eat increase in the certification rates.
Scheme, 749 miners were certified as
silicosis in South Wales. In 1944 the
; in 1946 nearly 3,598—a total of over
reciation of these figures can only be

obtained by comparison with the rest of the country (Fletcher, 1948). The total number of cases in South Wales is approximately 7 times that of the rest of Great Britain. As there are about 6 times more miners in the rest of Great Britain, this means that the incidence is more than 40 times greater in South Wales.

Focal Emphysema. There have been considerable advances in our knowledge of the effects of coal-dust in the lungs, due mainly to the work of Gough and his co-workers in Cardiff (Gough, 1947 ; Heppleston, 1947, etc.). They have shown that the essential lesion in the pneumoconiosis of coal workers is quite different from that of silicosis. In both diseases the dust has a focal deposition, mainly in the lymphoid tissue about the bifurcation of the respiratory bronchioles. In silicosis fibrous tissue is laid down in excess of that required for simple shutting off the dust aggregation from the rest of the lung, and it has a characteristic whorled appearance. In early pneumoconiosis of miners examination shows merely deposition of dust in these lymphatic aggregations, without tissue change even in cases showing x-ray reticulation. Later a little fibrous tissue is laid down, and the nodule retracts slightly ; the fibrous tissue is not, however, obviously excessive and is not whorled. It may be said that this is merely a difference in degree depending upon the amount of silica in the dust inhaled. However, accompanying this slight retraction there occurs a well marked emphysema in the air cells immediately adjacent to the nodule thus forming a focal emphysema throughout the lung. This is a fundamental difference, for emphysema, for some reason that we do not know, is a relatively minor change in relation to the silicotic nodule while it is the most striking lesion in relation to the coal nodule. Curiously also it does not seem proportionate to the degree of fibrosis.

A further change one meets with usually in more advanced stages of both silicosis and miner's pneumoconiosis is called massive fibrosis. The x-ray picture is striking and to some extent typical. At first the shadows appear to be formed by a coalescence of the typical granular mottling of the generalized dust lesion, later they form compact homogeneous masses—usually apical or subapical but sometimes in the mid zones, and usually more or less symmetrical. The nature of this change is not understood and is the subject of considerable controversy. Is it due to increasing fibrosis drawing the collections of dust nodules closer together ; or is it due to infection? Fletcher and others appear at the moment to favour the infection theory. Their evidence appears to me no more than suggestive—briefly—increased sedimentation rate in cases of massive fibrosis, higher in the more progressive cases ; in later stages, temperature and rapid deterioration ; and the association with tuberculosis which though its presence cannot often be proved during life is not infrequently found at death. One has also to remember that some workers, notably

lways maintained that collagenous

e comparatively rarely considered
e produced by atelectasis of the
les, and in this connection it is
believes that the focal emphysema
osis of the respiratory bronchioles
If the stenosis became complete,

n iron and tin, appear to be quite
The particles are deposited in the
bronchiolar bifurcations just as
ithout leading to any reaction or
n abnormal x-ray picture because
radio-opaque by reason of their

working on iron and steel, are
work. The heat of the welding
d oxidises and the fume therefore
in a very fine state of division.
nt many years at this work may
ey have performed a lot of work
tanks. The evidence on which I
es rise to such well marked x-ray
may be considered under four

elders for 16 years and have examined
incapacity or diminished capacity for
asons. I have kept in touch with some
this period and find that they continue
equently admit to having a cough, and
se are partly due to other factors and
le particles in the lungs. They have no
of fibrosis. They have a good chest
r exercise. These opinions are amply
only as regards welders but for other
on & Walsh, 1940; Groh, 1944; Sander,
5; Barrie & Harding, 1947; etc.).

en published in the world's literature of
owed the typical x-ray changes during
strated by excellent microphotographs,
tions of iron dust. The man died of
ne, the result of an accident. The fact
literature speaks for itself and suggests
have been able to obtain post-mortem
id not show the typical x-ray changes
osits of iron, these were not associated

ne absence of fibrosis round deposits of
ve been made on other workers who
r example silver finishers. These men
successive papers, Harding, McLaughlin

and their colleagues describe the post-mortem appearances in detail in 5 cases followed up to death. In the first 4 there was absolutely no fibrosis. In the last case (Harding, 1948) there was a minimal amount of fibrosis of the 'reticulation' type—not at all like that produced by silica. Harding thinks that in this case individual susceptibility may have been of importance, but there is always the possibility that the iron dust at some period of the man's long working life—over 40 years—may not have been pure and may have contained silica or other fibrosis producing constituent.

3. *Statistical.* The Registrar-General in his Occupational Mortality Supplement shows that in 1931 in the group of welders and burners, numbering 11,542 in England and Wales, there were only 123 deaths compared with 161 expected on the basis of age-rates of all males. Unfortunately no more recent figures are available. I have, however, collected from various factories information regarding the sickness absence of welders in relation to other workers and find that welders are favourably placed compared with other groups, not only for total sickness, but also for respiratory illnesses. I have been asked to consider these figures as confidential and therefore unfortunately they cannot be reproduced here.

Collen *et al* (1944) give reliable evidence about pneumonia in welders in an analysis of the sickness rates of the 90,000 workers in the Kaiser Richmond shipyards in America. They found that there was no increased incidence in welders. Collen, in a later article (1947) confirms his earlier findings saying that the annual incidence, death rates and case fatality rates for welders were similar to all other shipyard workers, and that the cases were similar in severity, required the same number of days for treatment and showed no difference in the incidence of complications.

4. *Radiological.* The presence of pulmonary fibrosis in and around dust aggregations cannot be diagnosed from an examination of single x-ray films; the film of a welder may be indistinguishable from that of a sandblaster with silicosis or a miner with pneumoconiosis. However, the x-ray changes in silicosis, asbestosis, and pneumoconiosis are permanent; if they change at all it is in the direction of progression, and this is common. McLaughlin and I, however, recently described two of our original welders whose x-ray appearances have changed for the better (Doig & McLaughlin, 1948). We showed that one man, who showed definite x-ray changes of the dust inhalation type in 1934, and who ceased welding on being told of these changes, now has an x-ray picture within normal limits. The pneumoconiosis has resolved, and no one could guess at its previous existence. The other man who had marked changes in 1933, and who continued to work as a welder, became a welding instructor in 1940, with consequent great diminution in his exposure to fume; his x-ray film now shows partial clearing of the abnormal shadows.

Other dusts which are considered on present evidence to be inert include tin, calcium, and barium, but much more knowledge is required before we can be sure that this is so. Tin is much more radio-opaque than iron, therefore deposits of tin in the lungs cause particularly dense shadows. Pendergrass & Pryde (1948) show an x-ray film of this type, discovered on routine examination, the subject being a man aged 45 years who had bagged tin oxide for 15 years. There was no disability.

Pneumonitis. Some dusts, both inorganic and organic, produce inflammatory changes. Chronic manganese poisoning is not a respiratory disease and is outwith the scope of this review. Since 1921, however, a relation between manganese and pneumonia has been suspected and numerous reports supporting this have appeared in the literature especially from the continent. For instance the installation of a manganese smelting factory at the village of Sauda in Norway was followed by a tenfold increase in the mortality rate for pneumonia, and similar reports have been made in regard to workers in German factories, and also miners of the ore. In this country there are few reports in regard to smelting but Lloyd Davies in 1946 showed that a group of men in a chemical works who were exposed to oxides of manganese existing as

fine particles in the air of the workroom had, between 1938 and 1945 a pneumonia rate 20 to 90 times that of other workers of the factory. The cases usually resolved without leaving any residual disability but compared with non-industrial pneumonia resolution was slower and less influenced by sulphonamides.

Some of the newer metals are also capable of causing pneumonitis. One of the most important of these is beryllium. Beryllium has recently assumed a place of considerable importance in industry. Some of its alloys have particularly valuable properties. In most cases the onset of the respiratory trouble is insidious and may be preceded by upper respiratory tract irritation, thereafter there is cough, sputum sometimes blood streaked, substernal pain and fever. X-ray shows diffuse haziness of both lungs with irregular fluffy areas of infiltration and necropsies in fatal cases have revealed a diffuse pulmonary oedema with haemorrhagic extravasation and many plasma cells but a relative absence of polymorphonuclears.

Delayed Pneumonitis. Cadmium, osmium, and vanadium may also produce chemical pneumonia when these compounds are inhaled as fine dust or fume, but before leaving beryllium mention should be made of another and most interesting condition which is caused by it. The pneumonitis already referred to is acute, coming on during the patient's employment. A delayed pulmonary condition has also been described coming on up to 6 years after exposure. The use of beryllium in Britain has lagged behind its use in America owing to the war and so far only one case of this type of beryllium reaction has been described in this country (Agate, 1948) but there have been several in America since 1936. Symptoms are mainly loss of weight, marked dyspnoea, and cough. X-ray changes are variable but usually consist of granularity, reticulation and sometimes nodulation. The most interesting point is that these cases are indistinguishable from sarcoidosis, and many are diagnosed as such. It cannot be said that beryllium is a cause of sarcoidosis but the two diseases are certainly very similar and to distinguish them may be impossible, especially as the pulmonary condition may be associated with skin changes. The late Leroy Gardner reported in 1946 that he had in his files some 100 cases of sarcoidosis of which 60 were known to have some exposure to beryllium. Certain it is that this lesion produced by beryllium is not a true pneumoconiosis, for the lung changes consist of granulomata, and must be regarded as only part of a general pathological condition with granulomata in the liver, the spleen and sometimes in the lymph glands.

Lung Cancer. The best known example of the relationship of carcinoma of the lung to occupational dust exposure is perhaps that of the Schneeberg miners where cobalt in the form of the arsenide is mined. Nowadays the mining of bismuth [illegible] cases of carcinoma of the lungs [illegible] cobalt, and possibly bismuth [illegible] be included as possibly responsible [illegible] radio-activity in the Schneeberg [illegible]

In this country there are [illegible] of lung cancer in the Cornish [illegible] arsenic. Several cases however are [illegible] in which arsenic is handled, [illegible] 1947 the Factory Department [illegible] relationship of arsenic [illegible] detailed investigation was [illegible] workers exposed to arsenic [illegible] cancer over other occupations [illegible] their figures as to site of [illegible] lungs. Bradford Hill & [illegible]

Amor (1938) reported [illegible] respiratory tract in nickel smelters [illegible] arsenic which existed as [illegible]

Chromium is another [illegible] capable of causing lung cancer. [illegible] from Germany, and related [illegible] Gregorius (1948) have [illegible] workers in the chromate industry [illegible] population at large. They showed [illegible] and that 23% of all deaths in the [illegible] the group of workers under [illegible] varied in the different plants from [illegible] population in the same age-group.

Asthma. Asthma is not [illegible] dusts but certain platinum [illegible] Milton and Perry in 1945 examined [illegible] refineries. Of 91 of them in contact [illegible] during the processes 52 suffered [illegible] sneezing was followed by profuse [illegible] ness of the chest, shortness of breath [illegible] persisted during the time the men [illegible] hour after they left. During the [illegible] quently had a severe bout of cough [illegible] divided metallic platinum were also [illegible] was quite conclusive that the cough [illegible] of the syndrome.

VEGETABLE DUSTS.

Generally speaking vegetable dusts are of far less importance than mineral dusts. Nevertheless the subject has so far been very largely neglected and their effects underrated. We know that the vegetable dusts cause several fairly well-defined types of respiratory disease, but how they are caused is obscure. There are several possibilities The first and most obvious one is that the noxious agent is the vegetable matter itself—some protein, alkaloid, or other constituent which is irritant or otherwise toxic to the bronchial or pulmonary tissue. Impurities must also be considered, for example silica Every sample of vegetable dust—whether it is of cotton, flax, grass seed, grain, etc , contains some silica derived from the soil on which the crop was grown—but generally it is not held that the small amount of silica present—usually about 2% or less, is likely to be harmful, particularly as much of it exists as fairly coarse grains. Then biological contaminants must receive much attention, for almost every sample of vegetable dust of any kind contains a very high bacterial and mycelial flora Some of these are known to be pathogenic and there is certainly evidence that some of the diseases due to vegetable dusts are due to bacteria or fungi It is also possible that the bacteria or fungi, though non-pathogenic in themselves, may by their digestive action on the cellulose or other constituent of the vegetable matter produce toxic substances Lastly some of the conditions are almost certainly produced by allergens

Mill Fever This is an acute, transient and usually slight illness occurring in new workers in dusty processes It usually comes on after 1 or 2 days in the mill, lasts a few days and does not reappear ; one attack confers immunity. Often the symptoms are not sufficiently severe to occasion absence from work They usually take the form of a chill, with pyrexia, headache, malaise and tiredness, and [illegible] there may be respiratory symptoms, such as sneezing, cough and dryness of the throat This curious little acute illness occurs in new workers exposed to a wide variety of vegetable dusts and goes under a variety of names—*e.g.*, 'combers fever' in hemp mills, 'cotton cold' in cotton mills, 'malt fever' in malt workers Under present day conditions it is likely that mill fever is less pronounced than formerly, nevertheless in flax mills the condition is still quite well known. It is sometimes said that all new employees get it but this seems an exaggeration ; probably the symptoms are experienced by 25 - 50% of new workers.

The cause is quite unknown Various suggestions made include the action of a foreign protein, histamine, bacterial endotoxin, allergy, but there is no proof.

Illnesses due to Fungi and Bacteria The definition of the role played by fungi and bacteria or their toxins is a very difficult matter. Obviously the finding of particular fungi [illegible] of little value in defining the role [illegible] the illness. However, there is [illegible] because they occur in small epide[illegible] vegetable matter and run atypica[illegible] fungi or bacteria

Outbreaks of an acute illness [illegible] amongst cotton weavers or [illegible] been associated with the humidi[illegible] thread which have been sized [illegible] irritation of the nose and throat paroxysmal cough and scanty [illegible] malaise The symptoms begin [illegible] weaving shed but sometimes [illegible] some weeks (Collis, 1915 [illegible] illness with the handling of [illegible] in the same factory handling [illegible] mildew as the cause Various [illegible] mucor, and aspergillus have been [illegible] the yarn

A similar condition has been [illegible] farmer's lung or thresher's lung [illegible] The illness is associated with [illegible] occurs most often after a wet [illegible] more serious and more prolonged [illegible] dyspnoea, haemoptysis, [illegible] seat of a broncho-pneumonia whi[illegible] Potassium iodide is said to be useful [illegible] or, after a prolonged convalescence [illegible] and emphysema X-ray films show [illegible] through snowflake mottling to co[illegible]

Also in this group should be [illegible] after the sugar has been extracted [illegible] for a great variety of bacteria, fu[illegible] described in America, and in 1[illegible] described 13 cases with necropsy [illegible] are again those of an acute or[illegible] pneumonia, arising after an expo[illegible] although I have personal knowledge [illegible] week's work in the factory Ag[illegible] disease is due to fungi, but these [illegible] include the suggestion that the [illegible] swells with the bronchial secretion [illegible] which become infected. Another

Reinhart and Caminita (1948) in America is that the disease is due to the endotoxin of *Aerobacter cloacae*.

Similar illnesses occur in connection with the handling of other vegetable materials known to be contaminated with moulds or bacteria, and *A. cloacae* is strongly suspected of being the cause of at least one of these, that occurring in workers handling low-grade stained cotton. Some hundreds of cases were studied in America by Neal and his co-workers. In most of their cases the illness appeared to be an acute infection of a general nature, with headache, fever, pains and prostration, but there were respiratory symptoms. They reproduced the symptoms in humans (all their experimental animals were immune) by inhalation of the dust of this low-grade cotton and by inhalation of a mist from cell-free filtrates of *A. cloacae* cultures.

Monday Fever. This term was introduced by Prausnitz but numerous observers before him had described an aggravation of respiratory symptoms on Mondays. It seems to be quite different from mill fever in that (*a*) it occurs in workers after many years' exposure, not in beginners, and (*b*) no acclimatization takes place; rather the condition tends to get progressively worse.

The symptoms, which occur on Mondays after the weekend break, or on return to work after an absence, usually consist of a sudden aggravation of cough, with chest tightness or actual breathlessness. At first the symptoms are confined to Mondays with the rest of the week fairly free. Gradually, however, the condition tends to persist until Tuesday and spread over the ensuing days, until ultimately the workers may be forced to give up work with constant bronchitis, dyspnoea and asthmatic wheezing. Improvement may be expected in the early days on giving up work but ultimately the condition becomes permanent, and in cotton workers the condition is spoken of as byssinosis.

Chronic Respiratory Disease. Chronic bronchitis has been reported in workers exposed to practically every kind of vegetable dust—from those used in enormous quantities like grain, cotton, flour, flax, hemp and malt to those used in comparatively small quantities as tobacco, and even tea. The proving of an etiological relationship between the disease and the dust is again often a matter of difficulty, as the disease produced differs very little from the chronic bronchitis, often with superimposed asthma, which may occur in the general population. Unlike the pneumoconiosis produced by most of the mineral dusts, the condition has neither characteristic x-ray findings nor typical post-mortem changes. The proving of a relationship is largely a statistical matter of proving that a certain occupation is associated with a mortality significantly in excess of the standardized mortality rates for the disease, and unless the occupation is precisely defined, the excess mortality may not be evident.

For example, many occupations in the [illegible] placed regarding standardized mortal[illegible] Cotton weavers in the Registrar General[illegible] of 94, and for 1921 - 22, 88. The rate [illegible] and for blow room workers are [illegible] It appears that chronic respiratory d[illegible] occupations is less than it was in [illegible] introduction of improved methods of [illegible] of appliances for intercepting and [illegible] also because of alterations in the [illegible] flax mills, which used to be com[illegible] performed by hand, seems to have [illegible] be remembered only by the older [illegible]

The best known of the chronic [illegible] dusts is byssinosis, [illegible] Although the symptoms and [illegible] examination do not differ from the [illegible] —frequently with a tendency to asth[illegible] lation, the condition taken in conj[illegible] histories is deemed to be sufficient[illegible] constituted Medical Board to [illegible] origin. Compensation was made [illegible] to workers with 20 years' experi[illegible] byssinosis reveal the changes of [illegible] do not show any feature which [illegible] occurs in the general population. No [illegible] tissue. Radiographic appearances are [illegible]

Asthma. Asthma, [illegible] and obviously due to allergy has been [illegible] vegetable dusts, for example [illegible] kinds of wood. In most inst[illegible]

I have personally known in only two [illegible] hypersensitive asthma (as opposed to the [illegible] occurring simultaneously in a factory [illegible] outbreaks were due to the same wood [illegible] have not found any references in the lit[illegible] firm engaged in shipbuilding used some [illegible] mine came across two cases in the firm [illegible] I saw one of these men—the other having [illegible] was quite conclusive as to this wood being [illegible] He mentioned that even the smell of the [illegible] upset him without there necessarily being [illegible] from slight attacks and more or less conti[illegible] wood being stacked in his work shed. [illegible] immediately disappeared. I also examined [illegible] of 3 more cases in former employees the pres[illegible]

At another firm of woodworkers [illegible] kilns I found six cases of asthma due to [illegible] a former employee, at his home. The latter [illegible] 1922 when this wood was first used in the [illegible]

with the use of this wood, usually by one of his fellow-workers for he refused to handle it himself. Since his retirement 2 years ago he has had no further attacks.

I have known of other types of allergic reaction due to this wood. One man suffered from such intense feeling of nausea when it was worked that he was prepared to give up his employment rather than be exposed again. At another factory an office employee who frequently had to pass through the woodworking shop suffered when Western red cedar was used, from sneezing attacks so violent and prolonged as to result in exhaustion. This man has remained fit and well during and after the war years since leaving the factory.

Such reactions raise many questions and there is as yet insufficient information to answer them although further enquiries are proceeding. One interesting feature, however, is that although Lehman and other workers refer to wood dust as sharp pointed and of average size 10 microns, several samples collected in the sawmill where the asthma cases occurred showed a very high count of small round particles down to something like $\frac{1}{10}$ micron, and of average size about 1 - 2 microns.

ANIMAL DUSTS

Animal dusts are much less injurious than either vegetable or mineral dusts. Occasionally they may carry infections; the sorting of wool containing anthrax spores may give rise to a pneumonic form of anthrax, the so-called wool-sorters disease. Sensitization effects have also been described, for instance "cough, tightness of the chest and feverish attacks" have been recorded in workers exposed to the dust from feathers (Bridge, 1934).

SUMMARY

Dusts met with in industry may produce a wide variety of reactions in the respiratory tract. Mineral dusts may give rise to pneumoconiosis which exists in three main forms, distinguished, from the pathological viewpoint, by fibrosis, or by focal emphysema, or by an absence of any tissue reaction. The fibrotic pneumoconioses exist in two main forms, one in which the fibrosis is dense and nodular, as in silicosis, the other in which the fibrosis is diffuse as in asbestosis. Other conditions resulting from the inhalation of mineral dusts include pneumonitis, immediate or delayed, lung cancer, and, rarely, asthma.

Vegetable dusts are in general less irritant than mineral dusts, but considerable further research is required to elucidate their toxic potentialities. Similar conditions, which may be grouped under five main headings, mill fever, acute and subacute infections, Monday fever, chronic bronchitis and emphysema, and acute (allergic) asthma, are caused by a large number of the vegetable dusts. Some of these, especially those in the chronic bronchitis and emphysema group, have no features which distinguish them clinically or pathologically from similar non-occupational illness.

Animal dusts comparatively rarely produce diseases of the respiratory tract, although asthma or hay fever may occur in sensitive individuals and infections of the respiratory tract, for example pulmonary anthrax, may be produced.

RE...

Agate, J N (1948) *Lancet* 2 ...
Amor, A J (1938) *Rep VIII ...* *Leipzig*, 1939 2 248
Barrie, H J & Harding H E (1947) ...
Bradford Hill, A & Faning, E L (194...
Bridge, J C (1934) *Annu Rep Chief* ...
Britton, J A & Walsh E L (1940) ...
Campbell J M (1932) *Brit med J* 2 ...
Collen, M F, Dybdahl, C L & O ...
Collen M F (1947) *Ibid* 29 2 ...
Collis, E L (1915) *Publ Hlth* ...
Cooke W E (1927) *Brit med J* 2 ...
Dennys J J Robson W D & ...
Dennys J J Robson W D & ...
Doig, A T & McLaughlin A I G ...
Doig A T & McLaughlin A I G ...
Frazer N & Snyder O A (1938) ...
Fletcher, C M (1948) *Brit med J* 1 ...
Gardner L U (1946) ...
... from *J* ...
Gloyne S R (1932) *Lancet* 1 1351
Gloyne S R (1933) *Tubercle* ...
Gorzlewski, G (1940) ...
Gorzlewski G (1941) *Ibid* 11 1 ...
Gorzlewski G & Jager R 1941 ...
Gough J (1947) *Occup Med* 4 ...
Gregory, J C (1831) ...
Groh J A (1944) ... 13 ...
Harding H E 1948 *Brit J* ...
Harding H E, Grout J L A & ...
Heppleston, A G (1947) *J Path* ...
Hope E W, Hanna W & St ...
Hunter, D, Milton R & Perry K ...
Hunter, D & Perry K M A (1946) ...
Jager, R & Jager F 1942 ...
Jotten K W, & Eickhoff, ... 1942
Kettle, E J (1932) *J Path Bact* 35 ...
Lanza A J (1945) *J Missouri* ... 1 ...3)
Lloyd Davies, T A (1946) *Brit J* ...
Machle, W & Gregorius F (1948) ...
McLaughlin, A I G, Grout, J L A ... 1, 337
Marshall, W (1834) *Lancet* 2 271
Medical Research Council (1942) *Sp...*
Medical Research Council (1943) ...
Medical Research Council (1945) ...
Meiklejohn, A & Jones, W W (1948) ...
Merewether, E R A (1930) *J ind Hyg* ...
Merewether, E R A (1933) *Tubercle* ...
Middleton, E L (1920) *J ind Hyg* ...

one of his fellow-workers for he refused to
st 2 years ago he has had no further attacks.
Bergic reaction due to this wood. One man
ised when it was worked that he was prepared
n be exposed again. At another factory an
pass through the woodworking shop suffered
m sneezing attacks so violent and prolonged
as remained fit and well during and after the

estions and there is as yet insufficient
ough further enquiries are proceeding.
. is that although Lehman and other
sharp pointed and of average size 10
in the sawmill where the asthma cases
unt of small round particles down to
average size about 1 - 2 microns.

AL DUSTS

urious than either vegetable or mineral
ry infections; the sorting of wool con-
ise to a pneumonic form of anthrax, the
tization effects have also been described,
e chest and feverish attacks' have been
e dust from feathers (Bridge, 1934).

MMARY.

ay produce a wide variety of reactions
dusts may give rise to pneumoconiosis
s, distinguished, from the pathological
l emphysema, or by an absence of any
eumoconioses exist in two main forms,
nd nodular, as in silicosis, the other in
asbestosis. Other conditions resulting
sts include pneumonitis, immediate or
asthma.

l less irritant than mineral dusts, but
equired to elucidate their toxic poten-
hich may be grouped under five main
subacute infections, Monday fever,
ma, and acute (allergic) asthma, are
getable dusts. Some of these, especially
l emphysema group, have no features
or pathologically from similar non-

nely produce diseases of the respiratory
r may occur in sensitive individuals and
. for example pulmonary anthrax, may

REFERENCES.

Agate, J. N. (1948). *Lancet.* **2** : 530

Amor, A. J. (1938). *Rep. VIIIth int. Congress industr. Accidents & occup. Dis., Leipzig.* 1939. **2** : 248

Barrie, H. J. & Harding, H. E. (1947). *Brit. J. industr. Med.* **4** : 225

Bradford Hill, A. & Faning, E. L. (1948). *Brit. J. industr. Med.* **5** : 1, 2

Bridge, J. C. (1934). *Annu. Rep. Chief Insp. of Factories*, p. 66

Britton, J. A. & Walsh E. L. (1940) *J. industr. Hyg & Tox* **22** : 125

Campbell, J. M (1932). *Brit. med J.* **2** : 1143

Collen, M. F., Dybdahl, G. L. & O'Brien, G. F. (1944). *J. industr. Hyg & Tox* **26** : 1

Collen, M. F. (1947). *Ibid.* **29** : 2, 113

Collis, E. L. (1915). *Publ. Hlth. Lond.* **28** : 252, 292

Cooke, W. E. (1927). *Brit. med J.* **2** : 1024

Denny, J. J., Robson, W. D. & Irwin, D. A. (1937) *Canad. med. Ass J.* **37** 1

Denny, J. J., Robson, W. D & Irwin, D. A (1939) *Industr. Med* **8** 136

Doig, A. T. & McLaughlin, A. I. G. (1936). *Lancet* **1** : 771

Doig, A. T. & McLaughlin, A. I. G. (1948) *Lancet* **1** : 789

Enzer, N. & Sander, O. A (1938) *J. industr. Hyg. & Tox.* **20** 5, 333

Fletcher, C. M. (1948). *Brit. med. J.* **1** : 1015, 1065

Gardner, L. U. (1946) *Trans. XIth Ann. Meeting industr. Hyg. Foundation (November)*, p. 88 (from *J. industr. Hyg & Tox.* **29** 4 abs. p. 7)

Gloyne, S. R. (1932). *Lancet.* **1** : 1351

Gloyne, S. R. (1933). *Tubercle, Lond.* **14** : 445

Goralewski, G. (1940) *Arch. Gewerbepath. Gewerbehyg.* **10** . 4, 384

Goralewski, G. (1941). *Ibid.* **11** : 1 106

Goralewski, G. & Jager R. (1941) *Ibid.* **11** : 1 102

Gough, J. (1947). *Occup. Med.* **4** : 86

Gregory, J. C. (1831). *Edinb. med. surg. J.* **36** . 389

Groh, J. A. (1944). *Industr. Med.* **13** : 8, 598

Harding, H. E. (1948) *Brit. J. industr. Med.* **5** : 70

Harding, H. E., Grout, J. L. A. & Lloyd Davies, T. A (1947) *Ibid.* **4** : 223

Heppleston, A. G. (1947). *J. Path. Bact.* **59** : 453

Hope, E. W., Hanna, W. & Stallybrass, C. O. (1923). *Industr. Hyg. Med.*

Hunter, D., Milton, R. & Perry, K. M. A (1945). *Brit. J. industr. Med.* **2** : 92

Hunter, D. & Perry, K. M. A (1946). *Ibid.* **3** : 64

Jager, R. & Jager, F. (1941). *Arch. Gewerbepath. Gewerbehyg.* **11** : 1, 117

Jotten, K. W. & Eickhoff, W. (1942). *Arch. Hyg., Berl.* **127** : 5, 344

Kettle, E. J. (1932). *J. Path. Bact.* **35** : 395

Lanza A. J. (1945). *J. Missouri med. Ass.* **42** : 765 (from *Occup. Med.* (1946). **1** : 193)

Lloyd Davies, T. A. (1946). *Brit. J. industr. Med.* **3** : 111

Machle, W. & Gregorius F. (1948). *IXth int. Congress on industr. Med., London*, 1948

McLaughlin, A. I. G., Grout, J. L. A., Barrie, H. J. & Harding, H. E. (1945). *Lancet.* **1** : 337

Marshall, W. (1834). *Lancet.* **2** : 271

Medical Research Council (1942). *Spec. Rep. Ser. No.* 243

Medical Research Council (1943). *Ibid. No.* 244

Medical Research Council (1945). *Ibid. No.* 250

Meiklejohn, A. & Jones, W. W. (1948). *J. industr. Hyg. & Tox.* **30** : 160

Merewether, E. R. A. (1930) *J. industr. Hyg.* **12** : 198

Merewether, E. R. A. (1933). *Tubercle, Lond.* **15** : 69

Middleton, E. L. (1926). *J. industr. Hyg.* **8** : 428

Neal, P. A., Schneiter, R. & Caminita, B. H. (1942). *J. Amer. med. Ass.*, 119, 1074.

Oliver, T. (1908). *Diseases of Occupation.* London: Methuen.

Pendergrass, E. P. & Pryde, A. W. (1948). *J. industr. Hyg. & Tox.*, 30, 119.

Prausnitz, C. (1936). *Spec. Rep. Ser. med. Res. Coun. London*, No. 212.

Sander, O. A. (1944). *J. industr. Hyg. & Tox.*, 26, 3, 79.

Schneiter, R., Reinhart, W. H. & Caminita, B. H. (1948). *J. industr. Hyg. & Tox.*, 30, 238.

Sh[illegible]er, C. G. & Riddell, A. R. (1947). *Ibid.*, 29, 145.

Thomson, W. (1836). *Med. Chir. Trans.*, 20, 230.

Williams, E. (1944). *Min. Fuel & Power. Rep. of [illegible] Tr[illegible] Pel Palitat[illegible] Miners in the Wales [illegible]* [illegible] H.M.S.O.

[illegible] (1938). [illegible], 2, [illegible]4.

PSORIASIS VULGARIS [illegible] SKIN, JOINT AND EYE LESIO[illegible]

S. HOROWITZ [illegible]

From the Department of Dermat[illegible]

Psoriasis vulgaris represe[illegible] disease [illegible] the British Isles [illegible] [illegible] quality [illegible] [illegible] that [illegible] per cent [illegible] cases are on record [illegible] corneal disease [illegible] [illegible] on record in which [illegible] patient. The p[illegible] lesions and thus estab[illegible] eye syndromes.

The eye lesions [illegible] non-specific chronic conjunctivitis [illegible] secret[illegible] [illegible] spread to the conjunct[illegible] may involve [illegible] the cornea consists [illegible] infiltrated zone under Bowm[illegible] zation and a hom[illegible] cences of this nature may appear and usually bilateral [illegible] chronic ulcers [illegible] been reported. [illegible] with the cutaneous erupt[illegible]

Hutchinson in 1887 [illegible] in connection with psoriasis [illegible] sixteen years, and the eye lesion [illegible] of the case and no correlation [illegible]

Soueix in 1896 described a case [illegible] At twenty-five he developed conj[illegible] the skin lesions. Later he devel[illegible] which impaired his visual acuity [illegible] conjunct[illegible], and an ulcer on the [illegible]

Pergens in 1931 described [illegible] vulgaris for twenty years and de[illegible] photophobia and an excessive [illegible] a corneal ulcer which perforated [illegible] no relationship between the eye [illegible]