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Occupational disease in road and building industry

PLAINTIFF'S EXHIBIT
UC-196

Respiratory disease

Peter C Elmes

That part of the human lung where oxygen passes through into the blood stream in exchange for carbon dioxide is made up of tiny chambers called alveoli. These are lined by a layer of cells so thin that it took the electron microscope to prove that they actually existed. To preserve these cells from drying and breaking, a layer of moisture lies between them and the air in the lung, moisture whose surface tension is reduced by a surfactant to prevent the alveoli from emptying completely when you breathe out.

This delicate arrangement is protected from damage from dust by the extremely efficient filtration, moistening and warming system provided by the nose, the trachea and bronchial tubes. Coarse particles of dust are removed by the hairs at the entrance to the nose. From there on down the airways are lined by cells coated with a layer of watery jelly known as mucus. This mucus layer is kept moving by cilia, tiny hairs which wave in a coordinated manner so as to propel the mucus both from the lung and the nose into the back of the throat where it is swallowed. As air is drawn into the lungs it passes down tubes which branch repeatedly into smaller and smaller tubes. At each branching the airflow is thrown into a vortex which spins out the heavier dust particles until they touch the mucus lining in the tubes and become caught. The dust particles caught by the mucus are cleared from the tubes of the normal lung within 20 minutes of inhalation and end up in the stomach.

A 17972

Size and shape of dust particles

The system of filtration is so efficient that only a small range of particles reach that part of the lung where gas exchange occurs. If deposited there particles can do harm. The vortex clearance of the particles from the air stream is proportional to the sedimentation rate of the particles in moist air. For approximately round particles anything larger than 5μm is filtered off. Particles between 5 and 0·5μm tend to be retained in the alveolar part of the lung because they land beyond the mucus escalator. Particles below 0·5μm tend to remain suspended in air and be breathed out again without doing any harm. Elongated particles such as vegetable and mineral (asbestos) fibres sediment in air at a rate proportional to their transverse diameter. This means that fibres at 100μm or more long could get down into the lung provided they were thin enough. Furthermore, a long particle may get caught at one of the places where the tubes divide and be driven through the wall into the underlaying tissue when the person breathes out. Fortunately most vegetable fibres (cotton and so on) and wool or hair are too coarse to get very far down the airway. Almost all commercial glass fibre is also too coarse. The molecular structure of asbestos is such that it readily splits lengthways into very fine fibres. Chrysotile, the most widely used commercial asbestos readily breaks up into bundles of fibres only 200–300Å in diameter, amosite and crocidolite easily form fibres in the 600 to 1200Å range. Bundles of this thickness, and from 1 to say 150μm in length, would be retained in the lung provided they were straight. In practice dust particles of amosite and crocidolite are straight and a large proportion of inhaled particles of this size range are retained in the lung. Because they are curved the fine fibres of chrysotile are more readily caught by the filter system and are less likely to be forced through the bronchial walls into the tissue. It is probably for this reason that chrysotile dust is less likely to cause lung damage than similar dust derived from amosite or crocidolite.

In general, the size of the dust particle generated by any process depends on the speed of impact. Hand-driven tools produce very little respirable dust but high-speed drills, crushers, saws, and so on produce a high proportion of fine dust which, unless it is accompanied by larger particles, is invisible to the naked eye. In time dust particles tend to aggregate with each other and especially with particles of moisture to form larger particles which are removed by the respiratory filter. Therefore dust is most dangerous close to the point at which it is formed.

Chemical nature of dust

A dust particle, irrespective of its chemical nature, will create a disturbance when it lands on the lining of the airway and therefore most people cough and wheeze when they

Professor Elmes of Queen's University, Belfast delivered this paper at a meeting of the Road & Building Materials Group of the SCI in London on 23 November 1972.

the filter to be retained in the alveolar or gas exchange part of the lung cause no immediate disturbance. If they are inert, like the iron particles retained by an arc welder, they will be packed away in special cells in the lung and remain there for the rest of the individual's life. Silica dust is not inert and as long as it remains in the lung it dissolves to give an acid which destroys cells while the lung tries to imprison the particles in a scar. The process of cell death and scar formation makes the lung stiff so that it becomes more and more difficult to use because of the work required getting the air in and out.

Although all asbestos minerals are silicates they do not behave like pure silica particles. They cause damage to the lung apparently because of their needle-like shape rather than because of the release of a toxic chemical. Asbestos fibres become coated with brown material and may be identified in the lung 30 or more years after they were inhaled. From the occupational health point of view, asbestos differs from silica in two important ways. Firstly, particles of respirable size may be released from prepared asbestos by simple handling, high-speed impacts are not necessary. Secondly, although chemically inert, a heavy load of dust particles continues to damage the lung for many years after exposure has ceased.

History of asbestos usage

Asbestos was used by prehistoric man in Finland to reinforce clay cooking pots, and by the Greeks and Romans to make wicks for altar lamps and shrouds for emperors. Its special properties were not fully appreciated until the middle of the nineteenth century when fireproof insulation became essential in the manufacture of ships and locomotives, anywhere where high pressure steam was generated from a furnace and boiler. Old mines in Northern Italy and Finland were enlarged and then large deposits in eastern Canada, South Africa and Siberia were discovered. Very large quantities of cheap fibre became available by the end of the first world war. Until that time asbestos cloth quilts and boiler compound (a mixture of magnesia and fibrous asbestos) were the main end product, both being used for insulation.

Between the wars and following the second war, the strength, durability and cheapness of the material was appreciated increasingly. Asbestos is now mainly used to strengthen cement products such as corrugated roofing and surfacing boards, pipes and heat-resistant cladding of the steel framework of buildings. This consumes over 90 per cent of the world production of asbestos. Reinforcement of plastic tiles and so on and pure insulation comes next, and then very small quantities in certain special filters and other uses. The main technical problem at the present time is whether asbestos is really essential in the asbestos cement products or whether a cheap safe substitute can be found.

Occupational hazards of asbestos

A 17973

Asbestosis

Because of its chemically inert nature, manufacturers and workers handled asbestos like wool without fear of the consequences until 1930. Cases of lung damage in asbestos workers had been reported by doctors from 1900 onwards, but were usually attributed to a modified form of tuberculosis. It was not until a survey of asbestos factory workers was carried out by the factory inspectorate that it was realised that many workers were dying of lung fibrosis within 15 years of first exposure.[1] Death was due to a pro- [illegible] its destruction by very large numbers of asbestos fibres – perhaps a million fibres in every millilitre of lung.

Regulations were introduced in the asbestos textile and parts of the asbestos cement factories to control the dust levels. As a result asbestosis among factory workers entering the industry since 1933 has almost disappeared. Studies with chrysotile dust indicate that if the dust level is kept below 2 fibres per millilitre of air, less than 1 per cent of workers develop asbestosis before they retire after 50 years' work. This is a cumulative limit of 100 fibre years/ml, and appears to hold good irrespective of the age at which the worker is first exposed.[2] Similar standards have been applied to the use of amosite and crocidolite but are probably not satisfactory for reasons which will appear later. This standard of less than 2 fibres per millilitre in the breathing zone of workers in asbestos factories has been achieved in most factories in Britain. It was not applied to the handling of asbestos in the user industries such as shipbuilding, civil engineering and the manufacture of cars and so on.

The incidence of new cases of asbestosis among asbestos factory workers began to fall soon after the introduction of the new regulations in 1933. By the early 1950s it was already apparent that the only serious new cases were derived from the diminishing population of workers who had been employed before 1933. However, new cases of asbestosis were being reported from insulation workers in factories and shipyards and by the middle 1960s these cases exceeded the cases arising from the asbestos factories and the total number of cases certified each year continued to rise. The new regulations, if applied to all the user industries, should see a change for the better in the number of cases of asbestosis certified in the next few years.

Lung cancer

Lung cancer, especially in men who smoke, has become increasingly common during the last 30-40 years so that the first few reports of cancer in asbestos workers were regarded as chance occurrences. However, the Medical Inspector of Factories in his report for 1947 noted that 17 per cent of men dying after certification for asbestosis had lung cancers as well.[3] This was beyond the realms of chance and provoked an investigation in one particular factory population[4] which confirmed an increased risk of lung cancer in workers with asbestosis. However, this, and a follow-up study published in 1968,[5] suggested that the risk was only serious in factory workers who had been exposed before the introduction of the 1933 regulations. There remained the problem of men being exposed to asbestos in the user industries. By 1965 Buchanan had shown[6] that over 50 per cent of men certified as suffering from asbestosis in Britain died with lung cancer and this proportion may now be about 70 per cent. Reports from America indicate that this lung cancer risk may only affect cigarette smokers and that asbestos does not cause a serious increase in lung cancer in non-smokers.[7] If it proves possible to apply the current dust regulations to the construction industry then the risk of lung cancer may be avoided, but men who have already been exposed to large amounts of dust are likely to develop their lung cancer at about the age of 55 or 60 if they continue to smoke irrespective of their future dust exposure. The withdrawal of men from work because on X-ray or other medical testing they show evidence of heavy dust exposure does not reduce the risk of cancer as far as we know.

In the late 1950s a number of cases of a rare cancer of the lining of the chest wall occurred in South Africa, a number far in excess of any previously reported. These patients had asbestos dust in their lungs and had at some time or other lived near (but not necessarily worked in) the blue asbestos (crocidolite) mines in the North West Cape province.[8] Fifteen such cases of mesothelioma had already been reported from Belfast and subsequent studies there, in London, in other shipyard cities and in New York have confirmed the relationship between this tumour and asbestos exposure. The worrying aspects of this work have been the slight degree of exposure to the dust (3-6 months handling asbestos filter pads for gas masks *with* local exhaust ventilation) and the very long latent period between exposure and the development of the tumours. The average age of death from this tumour in Belfast is about 60 years and the mean interval between first exposure to asbestos and the development of the tumour is 43 years.[9]

Much work has been done on this tumour during the last 15 years. It now seems likely that crocidolite is responsible for the majority of cases but that amosite may be responsible for some. It is possible that chrysotile does not produce this tumour at levels of exposure which have existed up to the present time, although it can cause the tumour in experimental animals. It seems wise not to allow any exposure to crocidolite dust in any industry and to restrict amosite to essential uses. But restrictions applied at the present time are not likely to affect the rising incidence of this tumour for many years to come. There must be many thousands of individuals who have already received their dose of dust and who will develop this tumour when they reach the age of 60 (give or take 15 years either way). Stopping further exposure to dust and stopping smoking are both unlikely to influence the outcome.

The tumour may be present for weeks or months before the patient is aware of it. It usually starts in the chest but can start in the abdomen. The patient is usually still in good general health when he first consults his doctor because of a dull ache or heaviness in one side of his chest. Unproductive cough and breathlessness soon follow. At this stage the patient is sent to hospital and investigations reveal the presence of fluid (which may contain blood) or scarring around one lung. It can be difficult to establish the diagnosis for certain without an operation and even then the diagnosis may remain doubtful. There is no satisfactory treatment and patients usually die about a year to eighteen months after the onset of symptoms. Increasing breathlessness is the main difficulty and this can sometimes be relieved by the periodic withdrawal of fluid from the chest. Towards the end the patients may develop difficulty in swallowing and lose weight rapidly. Pain often becomes severe and is difficult to control with drugs. This may persuade doctors to try various forms of treatment including operations which are of little value. In some cases these patients may survive for two to three years after the onset of pain and one can but admire the fortitude with which many cope with this situation.

From being a rare disease this kind of cancer is now occurring at a rate of 100 to 150 new cases each year and this incidence is likely to continue to rise.

A 17974

Specific industrial risks

The general description of the hazards to health which may result from exposure to asbestos is alarming and is difficult to interpret in relation to specific occupations or industries. The studies which led to the regulation of the dust concentrations in asbestos factories were only done on textile workers. For the next 20 years attention was focused on these workers and men doing similar work in asbestos cement factories. Even in these situations dust analysis was not done regularly and it is difficult to estimate the cumulative dust exposure of older men who have been in the industry since boyhood. In most user industries, especially those like construction work which moves from site to site, information on dust levels does not exist. Changing materials, changing techniques and the use of high-speed tools all alter dust conditions so that the prediction of the health hazard in a working population from the records of past exposure is impossible. The only way to determine the hazard is to study the health of the workers. No relevant study has yet been made in the construction industry. Such studies are urgently needed and a study done on insulation workers in Belfast illustrates the way in which studies can be carried out.

The conventional way of investigating an industrial health hazard is to examine a representative sample of working men and see whether they show any signs of disease when compared with a sample of the general population. This kind of survey is effective in revealing disease which develops relatively early in a man's working career provided he is able to remain at work after the disease has become apparent. It was effective in demonstrating the presence of asbestosis in textile workers in 1930[1] only because conditions were very bad. In Belfast 50 insulation workers were selected and compared with 50 men from the municipal transport department who were matching them for age, height and smoking habits. There was no difference between the two groups of men, either from the point of view of their general health, or as a result of X-rays and detailed tests on their lungs. Both groups contained some older men and heavy smokers who had shortness of breath, a cough and the other features associated with chronic bronchitis.[10]

This preliminary study was followed by a thorough examination of all the insulation workers in Northern Ireland who were willing to spend half a day with us. 93 Per cent of the men came and they appeared normal for their age. As in the first sample the older men tended to have the symptoms of bronchitis and the X-rays showed a high proportion of abnormalities in these older men. In a very few it was possible to say for certain that they were suffering from asbestosis, but even in these it was hard to say whether their asbestosis was causing them as much trouble as their chronic bronchitis. There were no cases of lung cancer or mesothelioma picked up in this survey of men at work.[12]

The third part of the investigation consisted of going over the records of the trades union branch to which these insulation workers belong and the records of the contractors who employ them to find out who was doing insulation work in 1940.[11] One sinister piece of information which came from the union was that there were no insulation workers drawing their old age pension from their contributory pension scheme. It was possible to identify 168 men at work in 1940 and Mrs Simpson had the task of finding out what had happened to them. If they had died we then found out both from the Registrar General and the hospital records the cause of death. Wherever possible this diagnosis was confirmed by re-examination of preserved tissues removed at operations or post-mortem. This was necessary because in the early part of the period (1940-55) most pathologists were

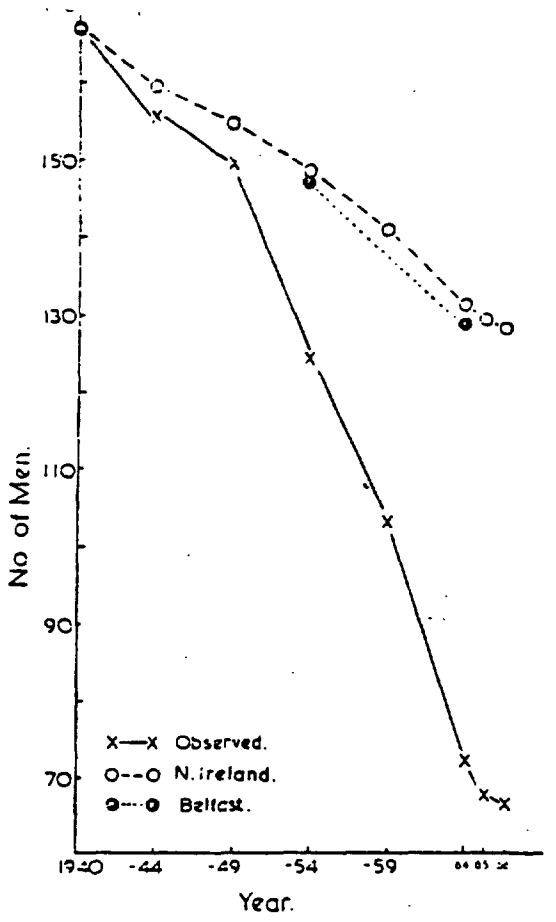

Fig 1 [illegible]

unlikely to differentiate between a mesothelioma and a lung cancer. The experience of these men was compared with the Registrar General's analysis of causes of death in Northern Ireland for men of the same age as our insulation workers over the same period (1940-66). The results of this complex investigation are shown in three graphs. In the first (Fig 1) the survival is compared with the survival of equivalent men in the general population of both Northern Ireland as a whole and more specifically of Belfast where these figures were available. The lines start to diverge in 1950 and by 1966 only 65 men survived of the 168 when about 100 should have survived. There had been 35 more deaths than expected.

In the second and third graphs (Figs 2 and 3) the number of men dying in each five-year period is compared with the number expected. In Fig 2 deaths due to all kinds of cancer are shown. In Fig 3 deaths from lung cancer and mesothelioma are shown together. Both graphs show that from 1950 onwards the number of men dying from cancer, in particular cancer of the lung and pleura, are many times the number expected. This investigation revealed an obviously unacceptable occupational hazard which was not even hinted at by the results of studies on men still at work.

The reason for different results from the two methods of study is the delay between exposure and the development of these to cancers (25 to 30 years for the lung cancer and perhaps 40 years for the mesotheliomas) so that the illness may not develop until after the man has left the industry. In any case a man does not continue to work for many weeks after a cancer has reached the stage at which it would be revealed by X-ray or clinical examination.

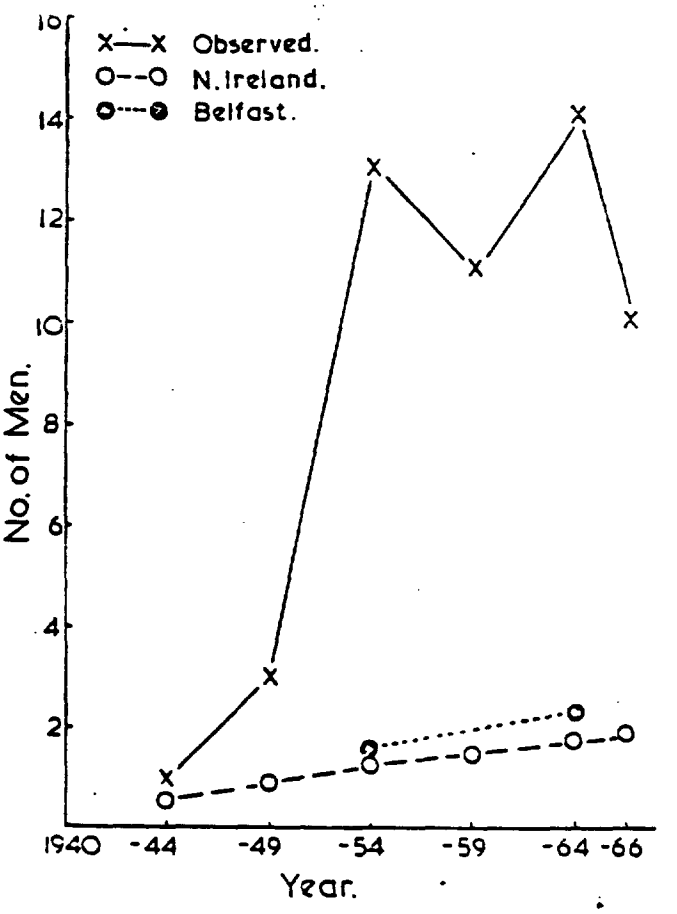

Fig 2 [illegible]

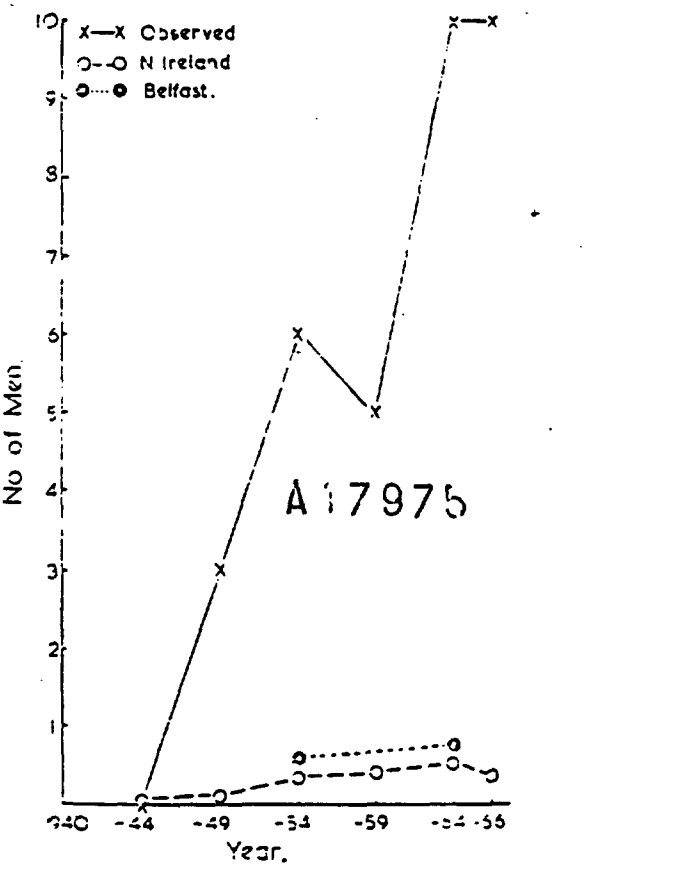

Fig 3 [illegible]

It would be a monumental task to set up this sort of investigation for all construction workers who handle asbestos at some time during their working lives. However, there is a method of finding out which workers are likely to be at hazard. The mesothelioma is a rare tumour and in Britain about 80 per cent of cases are thought to be due to exposure to asbestos at work. Therefore if an investigation is made of the work history of all people dying of mesothelioma then it is possible to show what occupations are responsible and consequently need further investigation. In Belfast the cases can be divided into two main categories, those working most of their lives in the shipyards and those who were mainly employed elsewhere. Of 62 cases recently investigated, the occupations are as shown in Tables 1 and 2. In Table 1 it is seen that the shipyard has given rise to 45 out of the 62 cases, but that only 10 of these occurred in insulation workers. The majority of the shipyard cases arose in men whose exposure was an incidental result of the work mainly because of working in confined spaces near men handling insulation material. It is understandable that pipe and engine fitters should be exposed especially during repair work because their concern is with equipment normally covered by insulation. But platers and so on and electricians were less expected and the extent and circumstances of their past and present exposure manifestly needs investigation. Outside the shipyard there were 17 cases and of obvious concern to the construction industry are the eight cases arising in builder's labourers, joiners, sawyers and timber workers. In the latter group, retrospective questioning did not reveal the source of asbestos.

This was a relatively small study in one city heavily biassed by the presence of a large shipyard. To be of real value this kind of investigation should be carried out on a nationwide basis. It has the major disadvantage of revealing the consequences of working conditions many years before. But it is the only method of pinpointing hazardous occupations with respect to asbestos at the present time. Once identified the extent of the hazard requires detailed investigation along the lines used in the Belfast insulation study. This work takes time and awareness of the possible risk should be the reason for the introduction of preventive measures rather than waiting for the results of detailed studies.

Table 1 Mesotheliomas in shipyard workers in Belfast – 45 of a group of 62 cases

Insulators:		
Full-time	5	10
Transient	5	
Other occupations (helpers and labourers included):		
Plumbers, pipefitters and boiler makers		17
Engine fitters		4
Platers, riveters and welders		4
Electricians		8
Other		2

Table 2 Mesotheliomas in non-shipyard workers in Belfast – 17 of a group of 62 cases

Known exposure:	
Heating engineers and boilermen	2
Builders' labourers and joiners	6
Dockers	1
No known exposure:	
Linen workers (women)	2
Sawyers and timber workers	2
Baker	1
Postman	1

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Letters to the editor

Gelatin emulsions in the formation of microcapsules

Sir, – Dr A. Courts in his lecture on 'Gelatin emulsions in the formation of microcapsules' before the SCI Food Symposium in March 1972 and reprinted in *Chemistry and Industry*[1] states that the most profitable application for microcapsule technology is to be found in the carbonless copy paper manufacture.

We are convinced that the readers of *Chemistry and Industry* will find Dr Court's lecture extremely valuable because it presents a fair picture of the present knowledge on gelatin based micro encapsulation technique.

There are, however, some errors which we would like to correct. Dr Courts states 'The capsules are coated onto the underside of a sheet of paper and this is then contacted with a surface coat of attapulgus clay on the following sheet. When the capsules are broken by the pressure from a typewriter key, or a pen, the dye is released and reacts with the clay surface, which induces oxidation leaving a blue coloured print. One dye is oxidised immediately and the second dye provides the more lasting effect'.

In actual fact[2,3] the primary mark on breaking the capsules is brought about by the immediate ring opening of crystal violet lactone in contact with the clay surface. No other chemical reaction is involved and a blue image of limited light stability is produced. The lasting blue-green image occurs slowly through the decomposition of benzoyl leuco methylene blue in a complex mechanism[4] involving oxidation as well as the action of light.

Yours faithfully

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