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EDITORIAL

MAGIC, MENACE, MYTH AND MALICE

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In the 1950s, asbestos had so many uses that it was known as the *magic mineral.* However, in the next few years it was established that severe exposure to respirable asbestos fibres had led to respiratory fibrosis, or asbestosis, excess lung cancer, and mesothelioma. Ignoring the word 'severe' in the preceding sentence, an anti-asbestos lobby, based in the Mount Sinai School of Medicine of the City University of New York, promoted the fiction that asbestos was an all-pervading *menace*, and trumped up a number of *asbestos myths* for widespread dissemination, through media eager for bad news. These included: all workers ever exposed to asbestos will die prematurely of asbestos-related disease; all forms of asbestos are equally hazardous; one asbestos fibre can kill; any substitute for asbestos must be less harmful. Like all myths, they were inventions without foundation and entirely untrue, but they were welcomed by the media and promulgated so ardently that they were soon almost universally believed, engendering widespread 'fibre-phobia'; the anti-asbestos lobby (hereinafter The Lobby) became both influential and powerful.

When the first findings from Quebec were published they threatened to dispel the myths, and so The Lobby tried to denigrate them. As this could not be done on scientific grounds, a campaign of intense *malice* was conducted against the principal Quebec investigator. And of course any group, such as that at Tulane University in New Orleans, which found evidence contrary to the myths was also castigated or even ostracised. Nevertheless, honest scientists have continued their endeavours, and what follows provides some background to the study that is reported in the accompanying paper (p. 13).

The ancient Romans used a cloth made from a certain mineral fibre for the enshrouding of corpses before cremation to facilitate collection of ashes for burial and, because the shroud was cleansed by fire, they called the fibre *amiantus*, from the Greek for undefiled, namely αμιαντος—and this led to the French *amiante*. The English *asbestos*, which derives from the Greek α- (not) with σβεστος (quenchable), is really a misnomer due to an error in the *Natural History* of Pliny (A.D. 23–79) but was in use for centuries to denote a specific fibre now called chrysotile. Asbestos is now a collective term for a wide variety of minerals, but usually restricted to six naturally-occurring mineral silicate fibres.

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The earliest reported uses of asbestos were in Finland, where the indigenous, anthophyllite fibres were found in the clay of pots made 4000 years ago, having been added presumably because of their now well-known binding properties; and in China, for textiles, at least 3000 years ago. Several classical writers refer to what must have been chrysotile used for the wicks of temple lamps, and thereafter there were several references to amiantus (incorrectly amianthus), a fine soft incombustible cloth made from 'the long flexible pearly-white fibres of [chrysotile]', for example by Marco Polo (*ca.* 1250 A.D.) and Benjamin Franklin (in 1725).

The modern asbestos industry dates from the discovery in the 1870s of large deposit of chrysotile fibre in Quebec; slightly later came the commercial exploitation of chrysotile in Russia, and then of crocidolite, amosite and anthophyllite. Production grew from a few tons of Quebec chrysotile in 1878 to a peak a century later of around 6 000 000 tons. In 1977, the U.S.S.R. produced 45% (all chrysotile), Canada 27% (also all chrysotile), at least seven other countries* 15% (almost all chrysotile) and South Africa 13% (mainly crocidolite and amosite) (Ward, 1977).

Over 3000 uses of asbestos have been identified. It was introduced into the Paris Métro in 1903 to prevent the repetition of a disastrous fire, and its pre-eminence as a fire-resistant material has been recognized since at least 1932 when it was used in the interior of RMS *Queen Mary* to prevent the spread of any possible fire. From 1912, seamless asbestos-cement pipes have been very widely used for a multitude of purposes. Asbestos has been exploited in the form of building materials such as tiles and for cements and cement sheets, and was used for filter cloths, linings for certain chemical vessels, valve packings and gaskets. Asbestos is a major ingredient of brake linings and clutch facings. It has also had many domestic applications (in ironing boards and oven cloths, for instance) and it was a favourite material in the 'Do-It-Yourself' market. It is hardly surprising therefore that asbestos earned the sobriquet 'The Magic Mineral'.

It is quite difficult to understand how the various types of asbestos fibre could possibly be thought of as one substance because they differ so greatly—in their physical and chemical characteristics and in the shapes and sizes of their macroscopic forms, and so in their commercial uses, in the shapes and sizes of their microscopic forms, and in their durability. Nevertheless, asbestos is the generic name given to a group of six naturally occurring mineral silicate fibres of the serpentine and amphibole series: the serpentine chrysotile (white asbestos) and the amphiboles crocidolite (blue asbestos), amosite (brown asbestos), anthophyllite, tremolite and actinolite.

The main chemical constituent of all forms of asbestos is SiO_2, 42% of chrysotile and 51% of crocidolite and amosite. The other principal constituent of chrysotile is MgO (42%), with less than 3% FeO and Fe_2O_3; crocidolite and amosite contain less than 3% MgO, with about 35% FeO and/or Fe_2O_3. Chrysotile fibres are fine and highly flexible, with good heat resistance; crocidolite and amosite fibres are more brittle and of harsher texture. Most crocidolite has come from South Africa, and the source in Australia has ceased production; amosite was produced until recently in South Africa. Until 1974, anthophyllite was produced in Finland and used

*Including China, Cyprus, Italy, U.S.A. and South America, all chrysotile, and Australia, which was reported as producing chrysotile, but also mined crocidolite at Wittenoom Gorge.

extensively there. Some limited mining of tremolite was carried out in the U.S.A. and in the Far East; this fibre now exists primarily as a natural contaminant in other exploited materials (Ward, 1977).

Although 90% or more of asbestos usage has always been of chrysotile, other types of asbestos have often been specified for particular purposes. Until the 1950s, the British Admiralty specified crocidolite for certain materials used in the insulation of machinery in warships, and insulation of steel decks and hulls was mainly with crocidolite. Another specified use of crocidolite was for the filter pads in gas masks for allied service personnel in World War II, whereas chrysotile was used for civilian masks. Over 720 000 tonnes of chrysotile was used in the U.S.A. in 1974, together with 33 800 tonnes of crocidolite, 8300 tonnes of amosite, and less than 1000 tonnes of anthophyllite. Almost all the crocidolite, but more than five times as much chrysotile, was used for asbestos-cement pipe. The amosite was used mainly for asbestos-cement sheet and roofing products, but with over 20 times as much chrysotile. However, for thermal insulation, 1600 tonnes (20%) of amosite were used with 6600 tonnes (80%) of chrysotile (Ward, 1977).

Health effects arising from the inhalation of dust, albeit in hard-rock miners, had been recognized by Agricola (1495–1555) and had been called 'pneumonokoniosis' by Zenker in 1876. It should not therefore have been surprising that, in 1898, one of H.M. Inspectors of Factories for England and Wales drew attention to lung injury in asbestos sifting and carding (as well as in operations with silk and hemp); no action was taken. Eight years later, a departmental committee on compensation for industrial disease heard of the death in 1900 of a 33-year-old man with pulmonary fibrosis who had spent 14 years working with asbestos, mainly in the carding room. Although 10 men who started the same work at the same time had already died of a similar illness, the official view, accepted by the representatives of the unions, was that asbestos could not cause lung disease; still no action was taken.

In 1930, E. R. A. Merewether and Chas. W. Price, H.M. Medical and Engineering Inspectors of Factories, reported a survey of 363 workers exposed to asbestos mainly in the manufacture of textiles and insulation products (Merewether and Price, 1930). The prevalence of pulmonary fibrosis was high, and clearly related to duration and intensity of exposure, to the extent that almost all men reaching retiring age after a working life in asbestos textiles or the manufacture of asbestos insulating materials were expected to be diseased. As in those days *asbestosis* (the name now given to this disease) was frequently fatal, a substantial proportion of the workforce must have succumbed before reaching retirement age: the risk must have been unimaginably high. The first environmental control measures were devised in the light of Price's recommendations on dust suppression and embodied into the Asbestos Industry Regulations 1931, which came into force in 1933, defining 'scheduled areas' in specified parts of the British asbestos industry. The earliest studies of asbestos-related disease in the U.S. appear to have taken place in the mid-1930s, and a Threshold Limit Value was set by the American Conference of Governmental Industrial Hygienists endorsed by the same organisation in 1964.

As John Gilson wrote in 1982: "Most occupational diseases, including asbestos-induced lung cancer and mesothelioma, were first detected by astute observation by clinicians or pathologists; it has usually taken many years before their severity and

extent have been revealed by systematic surveys." From 1934, primary carcinoma of the lung, then a very rare condition, was occasionally recorded in asbestos workers, usually in association with asbestosis. Then, in 1955, Richard Doll showed that the average risk of *lung cancer* in men who had worked in scheduled areas for 20 years or more was 13 times that experienced by the general population (Doll, 1955). Several later occupational cohort studies of asbestos workers reported excess lung cancer, which is now generally accepted as a possible effect of asbestos exposure. Between 1943 and 1961, there had been several reports of cases of mesothelial tumour associated with asbestosis or with a history of exposure to asbestos. A landmark publication was in 1960 by Chris Wagner and colleagues (Wagner *et al.*, 1960) who reported 33 cases of diffuse pleural mesothelioma from the crocidolite mining area of the North Western Cape province in South Africa, and within a few years it was recognized that asbestos dust could cause *mesothelioma*, that is malignant mesothelial tumours of pleura or peritoneum. From the 1960s, there have been suggestions of associations between exposure to asbestos and *gastro-intestinal cancer*, and possibly malignancies at other sites, but none has been reliably demonstrated.

In February 1964, the Geographical Committee of l'Union internationale contre le cancer convened a Working Group on Asbestos and Cancer. The Group, of some 40 experts in three sections: epidemiology; pathology; and physics and chemistry, met for two days in October, and the agreed report, submitted in March 1965, was published later that year (Working Group on Asbestos and Cancer, 1965). It accepted that there was an association between exposure to asbestos dust and carcinoma of the lung and diffuse mesothelioma of the pleura and peritoneum; also that the latent period between first exposure to the dust and detection of the related tumours is many years, usually 20 or more and up to 60 years, so that even if dust exposures were greatly reduced, further cases of these associated tumours would be expected to occur for many years to come. Associated lung cancer was probably not limited to exposure to any one type of asbestos fibre, but further investigation was urgently needed. It could not be concluded that only crocidolite was concerned with mesothelioma, although that fibre appeared of particular importance, and further investigation was needed.

The recommendations of the Working Group were far-sighted and remarkably comprehensive: those of particular relevance here were as follows. "1. That the importance of fiber type on the risk of developing asbestosis, carcinoma of the lung, and mesothelial and other tumors be investigated." Studies of mining and other populations exposed to only one type of fibre were recommended, in particular in Canada, but also in many other countries, and 'intranationally'. "2. That the relationship of dust dosage (including concentration and duration of exposure) ... to the incidence of asbestosis, carcinoma of the lung, mesothelioma and other cancers be studied." And under 6.A.(a), that "... special attention should be directed to a detailed, social (including smoking habits), occupational, environmental, and medical history from early childhood, ..." Other recommendations included: "3. That the effects of removal from further exposure to asbestos dust be investigated. 4. That further investigations be made of past and all future cases of diffuse mesothelial tumors of the pleura and peritoneum to establish any association with asbestos and other factors. 5. That studies of morbidity and mortality be

extended to asbestos-exposed populations that have not so far been widely investigated."

Almost all these recommendations were incorporated into the comprehensive epidemiological survey of the entire Quebec asbestos-producing industry since its inception which was initiated by Corbett McDonald, of McGill University in Montreal, in 1966. The main component was the mortality investigation of the 1891–1920 birth cohort, and especially important in that were the estimates for each member of the cohort of what the Working Group had called dust dosage (concentration and duration of exposure) and the smoking habits elucidated by questionnaire for the great majority of subjects. Other components concerned radiographic changes, respiratory symptoms and lung function, and their interactions, relations between radiographic changes and mortality, removal from exposure, and many other subjects, all of which have been fully reported. Another prime concern has been with mesothelioma, and there have been many papers on this subject. However, of more than 8000 deaths by 1992 of members of the cohort, just 38 could be ascribed to mesothelioma, and only now is a full report justified; it is nearing completion. McDonald and his colleagues extended their investigations into many other populations, including chrysotile friction product workers and textile workers, workers in a mainly textile plant using amphiboles as well as chrysotile, vermiculite miners exposed to tremolite, and gold miners exposed to cummingtonite–grunerite, another amphibole although not fibrous.

When the first report on mortality in the Quebec cohort appeared (McDonald *et al.*, 1971), it immediately drew the wrath of The Lobby. Of the 9981 men traced, 7568 were still alive on 1 November 1966 and 2413 had died, 97 (4.0%) from lung cancer, still a rare condition in Quebec, and three (0.1%) from mesothelial tumours. These proportional mortality ratios (PMRs) were very much lower than in two previous studies: Irving Selikoff and colleagues from Mount Sinai, studying 632 insulation workers reported PMRs of 18.9% and 5.8%, respectively, among 380 deaths; the corresponding PMRs, within 436 deaths among 4806 workers in a crocidolite factory in Barking, were 9.6% and 4.6%. McDonald's report concluded: "[Our] findings strongly suggest that chrysotile is less likely to cause malignant disease of the lung and pleura than other forms of asbestos, such as crocidolite, or that workers engaged in insulation and processing are exposed to additional factors which explain the difference."

The Lobby simply denied this enormous difference, claiming that McDonald had created it because he had accepted a grant from the Quebec Asbestos Mining Association with its vested interests in chrysotile. (The possible bias arising from investigators within The Lobby having been financed by opposed vested interests was, needless to state, ignored.) A campaign of personal abuse was mounted and given very wide publicity, including a series of articles in *The New Yorker*, later published in book form.

In the mid-1970s, the Gouvernement du Québec set up a Comité d'étude sur la salubrité dans l'industrie de l'amiante. The members of the commission spent a day at McGill to hear about the scope of the investigation and decide how best to conduct the public hearing; in another full day, all senior members of McDonald's team made presentations. A little earlier, Selikoff's group, at the request of the unions, had made two surveys in Thetford Mines, one a small mortality enquiry, the

other X-raying men of long service. The commission agreed to the unions' request to hear this evidence, but stated in their report that they found nothing from Mount Sinai to detract from what they had learned from McGill. At a symposium masterminded by Selikoff in 1978, the McGill team was permitted only one paper, a brief account of mortality in the Quebec cohort up to 1975; this was immediately followed by a presentation, the first of many from the Mount Sinai group and associates, of the small mortality study in Thetford Mines, and after it the author of *The New Yorker* articles was heard to ask what price the differences now.

At the same meeting, The Lobby dropped a blockbuster (Selikoff *et al.*, 1979)—a report of a mortality survey of 17 800 insulation workers in the U.S.A. and Canada followed for 10 years 1967–1976. There had been 2271 deaths, 612 (37%) more than would have been expected on the basis of white male death rates in the U.S.A. We were asked to believe that there had been an excess of 843 deaths, made up of 175 due to mesothelioma, 380 due to lung cancer, 120 from other cancers, and 168 from asbestosis. Even apart from mesothelioma and asbestosis which are all excesses because there are no sensible reference rates, cancer excesses of more than 50%, were cited as follows: lung 360%, oesophagus 153%, stomach 54%, colon–rectum 55%, larynx 134%, pharynx and buccal cavity 108%, and kidney 136%. The difference between 843 and 612 deaths was a shortfall of 231, which was 17.3% of the number of deaths expected from causes other than cancer and asbestosis, that is including heart disease, stroke and accidents. Even when causes were 'recorded from death certificate information only' (as distinct from 'best evidence', cited above, but still not as coded for official statistics), the pattern remained fairly similar, except that the shortfall was much reduced.

No other investigations have indicated a blanket of excess cancer even approaching such an order, and it is difficult in the extreme to see the relevance of these findings to any of the problems faced today. Nevertheless, they have had enormous influence throughout the world.

All the above had been offered with the comment that insulation workers had been exposed to time-weighted average dust levels of 4–12 fibres/ml, whereas there is a great deal of anecdotal evidence that insulation was perhaps the dustiest of all occupations, probably as bad as in the mills of Quebec, where the mean concentration in the 1960s was of the order of 50 fibres/ml.* Furthermore, it was stated that insulation workers had been subject to only limited and intermittent exposure to asbestos, particularly as some insulation materials contained no asbestos and other substances, such as rock-wool and fibrous glass, were also used. Even so, it was claimed that chrysotile was the sole cause of all the excesses.

This claim had wide acceptance and was a main plank in The Lobby's platform. It was followed by two insidious 'arguments' of which the first was that it could be taken for granted that, if any occupational group showed excess cancer and there had been any exposure to asbestos, then it was the asbestos that caused the malignancies. The second was that if workers are exposed to a mixture of

*Estimated from two reports by G. W. Gibbs and M. Lachance in the *Archives of Occupational Health*, (1972) **24**, 189–197 and (1974) **28**, 69–71. A dust level of almost 10 million particles per cubic foot (inferred from Fig. 3 in the earlier paper) has to be multiplied by a ratio of 5.3 (the median of the 19 ratios of membrane filter counts to midget impinger counts for milling operations tabulated in the later paper).

asbestos fibres in which chrysotile predominates, then it is the chrysotile that is responsible for any deleterious health effects; in other words, the admixture of amphibole has been too slight to affect health, and all ill effects must have been due to the chrysotile. An analogous argument would be that a drunken lager-lout cannot have been intoxicated by alcohol because that is only a small percentage of the brew.

The Lobby made several attempts to get all forms of asbestos banned. A paper prepared for the Swedish government was full of factual error and rambling argument but helped to introduce a total ban except for circumscribed 'essential uses'. Another, for the West German Department of Health (B.G.A.), simply dismissed the findings from Quebec, by far the largest study, as 'anomalous'.* Then in 1986, largely on similar unsatisfactory evidence from The Lobby, the Environmental Protection Agency (E.P.A.) of the U.S. government promulgated a total ban, from 1989, on all forms of asbestos. On appeal, the ban was lifted, but only partially, in 1991.

By then, there had been reports from over 30 epidemiological studies of asbestos workers, which can be summarised, in terms of Standardized Mortality Ratios (SMRs) for lung cancer and PMRs for mesothelioma (Table 1). It would be naïve to claim these SMRs and PMRs *prove* anything, but they do illustrate several facts. First, those for Quebec miners and millers are in line with those for other workers of chrysotile only, while those for the 17 800 insulation workers are well out of line even with those for workers of amphiboles only. Secondly, there are startling differences in lung cancer and particularly mesothelioma between those exposed to chrysotile only and amphiboles only. However, while there were very few mesotheliomas among chrysotile textile workers, there was a marked excess of lung cancer, particularly at one plant in Charleston NC, which used fibre from Thetford Mines in Quebec. Thirdly, the 'others' with mixed exposures had a mesothelioma PMR fairly close to that for amphibole production. As the fibre

Table 1

	Lung cancer		*Mesothelioma*	
	No. of deaths	SMR	No. of deaths	PMR
Chrysotile only				
Quebec mining & milling	230	1.25	10	0.3%
Textiles	94	1.69	3	0.1%
Other workers	238	1.16		
Amphiboles only				
Manufacture and mining & milling	208	3.07	91	3.6%
Chrysotile and amphiboles				
17800 insulation workers	397	4.24	170	8.7%
Other workers	770	1.74	283	2.6%

*The German asbestos industry, in its paper for the B.G.A., grossly misused the Quebec data in an attempt to demonstrate that chrysotile, and hence all forms of asbestos, was innocuous. Initially, this misuse may perhaps have been due to a misunderstanding of the bases of the data, but once these bases had been explained by one of the McGill team, the failure to retract and, worse, the insistence on publication by the B.G.A. were thoroughly mischievous.

'mixtures' were all chrysotile-rich, The Lobby used the 'lager-lout argument' to claim that it was the chrysotile that caused the mesotheliomas.

Meanwhile, experimental studies had suggested that chrysotile was at least as dangerous to rodents as amphiboles. It is a precept of scientific method that an experiment should be designed to test a specific theory. Progress is made when experimental findings are not explained by the theory, which has to be discarded in its specific form: another theory is then developed, to be tested in its turn by a new experiment. Progress is also made when epidemiological findings are in conflict with those from experiment: the theory apparently supported by the experiment has to be replaced. It is contrary to the principles of scientific method to maintain the theory in the face of disproof, whether that disproof is demonstrated by experiment or by epidemiology.

Therefore, when the theory that chrysotile was at least as dangerous as amphiboles was disproved in man, although supported in animal studies, it was essential on scientific grounds that the human evidence should prevail over that from animals, and hence over theory. However, The Lobby adopted the unprincipled approach of ignoring the human evidence, thus perpetuating the myth that all forms of asbestos are equally carcinogenic. Similarly, the E.P.A. will only accept epidemiological evidence after it has been 'confirmed' by toxicology.

But now, after many electron microscope studies, there is a convincing explanation of the reasons for differential effects in animals and humans (Elmes, 1991). Massive doses of chrysotile fibres, once retained in the lung of a rat, split up into finer and finer bundles, and eventually into fibrils (the basic crystal units, which are less than 0.1 μm in diameter), and so provided a much increased surface area for biological reaction. Within the animal's life-span, clearance from the lung was not great, and doses were so enormous (at the limit of tolerance, well over 2000 fibres/ml) that substantial proportions of animals developed tumours. On the one hand, however, the same dose of amphibole fibres was retained virtually without increase in surface area, so that the effective dose of amphibole in rodent lung was much smaller than that of chrysotile. In man, on the other hand, although the initial build-up of surface area was much greater, exposure for exposure, with chrysotile than with amphibole, the chrysotile was largely cleared from the lung before tumours could develop, whereas amphibole was retained indefinitely. The effective dose of amphibole in the human lung was thus very much greater than of chrysotile.

A corollary is that inhalation studies can only investigate effects manifest in the lifetime of the experimental animal, say two years in rats. These are essentially short-term effects whereas in man the interval from first exposure to disease is some 20 times longer on average. This has important implications for the evaluation of any substitutes for asbestos, particularly as short-term comparisons against chrysotile, many of which have been carried out, are seen to be invalid. Further discussion is irrelevant here.

The differential clearance of chrysotile and amphiboles from the human lung has been confirmed by several (*post mortem*) lung tissues analyses over the years. One of the more interesting findings is of at least as many tremolite fibres as chrysotile in the lungs of former Quebec miners and millers despite the tremolite contamination having been only of the order of 1%.

It is impossible to discern the motives of The Lobby. Nevertheless, its adherents

persist, most recently in a paper 'proving' the claim, incorporated in its title, that chrysotile is the main cause of pleural mesothelioma (Smith and Wright, 1996). Here, in what purports to be a review of the epidemiological evidence, 43 studies of industrial cohorts exposed to asbestos are identified. For 25 of them, the mesothelioma PMR averaged 3.0%, ranging from 0.8 to 8.8%, and they provided satisfactory grist; however, in the other 16 studies, the corresponding PMRs were too low for the authors' mill. Therefore, these 16 studies which, by the definition of a low mesothelioma PMR, included every one of those cohorts exposed to chrysotile alone, were ignored. Further comment is surely unnecessary—although a great deal more would be fully justified.

This is not the place to expose the folly of removing asbestos from buildings nor the potential problems arising with substitutes, either because of inadequate testing for toxicity or of expense or of both. Comment is, however, required concerning asbestos-cement, which in the form of building materials and pipes is of great importance, and is much cheaper than replacements. The asbestos used is chrysotile, amounting to about 15% by weight of the product; if reasonable precautions are taken in handling the products, the airborne hazard is extremely slight. Further, despite frequent claims by The Lobby, there is no evidence that ingested chrysotile fibres cause gastro-intestinal cancer. To deny a developing country the use of asbestos-cement is to deny it adequate housing, drainage and water for domestic purposes, and hence protection against the potential disasters of waterborne infections such as cholera, to say nothing of water for agricultural purposes. And this is, of course, by no means the only way in which a ban on chrysotile works against the public good.

Thus The Lobby's attitude is antisocial: it is essential that the views of the great majority of scientists working in these fields come to prevail. Undoubtedly, crocidolite is highly toxic and there is sufficient doubt about amosite and tremolite, because of their persistence in the human lung after inhalation, that all amphiboles should be proscribed. However, chrysolite at current levels of occupational exposure is essentially innocuous, except possibly in textile manufacture, and non-occupational exposures are orders of magnitude less severe. It is probably too much to hope that the publication of the accompanying paper will go some way to achieve this purpose.

Corbett and Alison McDonald started this mammoth investigation over 30 years ago and after enormous sustained effort it is nearly concluded: only three reports, on lung cancer, mesothelioma, and 'the tremolite question' [see a recent letter to *Science* (McDonald and McDonald, 1995)] are outstanding and all are nearing completion.

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