The biodurability of chrysotile asbestos

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ABSTRACT

Lung cancer, asbestosis, and, to a lesser extent, pleural mesothelioma have been linked to inhalation of chrysotile [Mg_3Si_2O_5(OH)_4] asbestos. The duration and intensity of exposure, along with fiber size and shape and mineral type (surface and chemical properties) appear to play important roles in the development of these diseases. The fluids in lung tissue contain very low concentrations of Mg and Si, and therefore they are undersaturated with respect to chrysotile. Therefore, chrysotile’s persistence in lung tissue is simply a result of its dissolution kinetics in the various biological environments of the lung. The dissolution reaction for chrysotile for pH < 9 is Mg_3Si_2O_5(OH)_4 + 6H^+ = 3Mg^{2+} + 2H_2SiO_4 + H_2O. This reaction proceeds in two steps. First, the magnesium hydroxide layer of the chrysotile dissolves, leaving behind silica that dissolves at a slower rate. Therefore, the fiber lifetime (i.e., biodurability) depends upon the rate of silica release. For the range of undersaturation found in lung tissue, the rate of silica release, 5.9 (± 3.0) × 10^{-10} mol/m^2s, is independent of pH. A shrinking fiber model predicts that a fiber of chrysotile 1 μm in diameter will completely dissolve in 9 (± 4.5) months.

INTRODUCTION

Throughout our lives we are exposed to a wide variety of mineral dusts. Fortunately most of these dusts are benign, but a few are known to produce lung diseases in animals (Guthrie, 1992). The public is most aware of lung diseases related to exposure to the serpentine and amphibole asbestos minerals. However, in order to find minerals and mineral-related substances to replace commercial asbestos, we must be able to identify the characteristics of the minerals that promote lung diseases. The shape, size, and mineral type of the particles are important etiologic factors in diseases related to mineral dust. (The mineral type controls the chemical and surface properties as well as the durability.) Stanton et al. (1981) concluded that the shape of particles influenced the development of tumors in rats because rats exposed to pulverized nonfibrous forms of chrysotile did not develop tumors. Stanton found that the probability of tumor development correlates with the fiber dimensions. In order for asbestos-related respiratory diseases to occur, the fibers must be small enough to enter the lungs. Fibers with diameters less than about 3 μm are respirable. The macrophage cells cannot easily engulf fibers longer than 10 μm, unless they coalesce to form giant cells, so these fibers tend to remain in the lower respiratory tract or penetrate the pleural membrane and enter the interpleural space (Davis, 1981). Middleton et al. (1979) reported that the deposition of chrysotile was much lower than that of amphibole fibers, but the clearance rates were similar. Other studies showed that the amount of amphibole deposited in the lungs of rats continued to increase with exposure time, whereas the amount of chrysotile deposited reached a plateau and did not continue to increase (Jones et al., 1989; Wagner et al., 1974). In the lungs, chrysotile undergoes morphological and chemical alteration, including longitudinal splitting along the x axis. On the other hand, amphibole fibers are relatively more resistant to biochemical change (Bellman et al., 1986). Note that all of these studies are relatively short term compared with the possible decades-long residence time of dust particles in human lung. Thus, it is useful to consider biodurability, that is, the resistance of the fiber to dissolution over a lifetime, as a factor in dust-related diseases.

The purpose of this study was to estimate the dissolution time of a respirable-size fiber of chrysotile in human lung tissue, with the ultimate goal of developing a general test for mineral dust biodurability. The fiber size considered most hazardous to the lung tissue has a diameter of less than 1 μm and is greater than 10 μm long (Davis, 1981; Bellman et al., 1986). The fluids in the lung tissue contain very low concentrations of Mg and Si. As a result they are quite undersaturated with respect to chrysotile. In order to determine the amount of time needed to dissolve a respirable chrysotile fiber, a dissolution rate constant was determined and applied in a shrinking particle model. There are several previous studies of chrysotile dissolution rates (Gronow, 1987; Churg et al., 1984;
leached, these studies cannot be used for the calculation of a fiber lifetime. Because a silica framework remains after the Mg has been leached, the studies report rates of leaching of Mg from the fibers. Most of them has produced rate constants that can be used to estimate the lifetime of chrysotile in lung tissue. Because a silica framework remains after the Mg has been leached, these studies cannot be used for the calculation of a fiber lifetime. The dissolution reaction for chrysotile for pH < 9 is

\[
\text{MgSi}_2\text{O}_5(\text{OH})_2 + 6\text{H}^+ = 3\text{Mg}^{2+} + 2\text{H}_2\text{SiO}_4 + \text{H}_2\text{O}
\]

and the equilibrium constant (Nordstrom et al., 1990) for this reaction at a human body temperature of 37°C is

\[
K = \frac{a_{\text{Mg}^{2+}} a_{\text{H}_2\text{SiO}_4}}{a_{\text{H}_4\text{O}^+}} = 5.9 \times 10^{30}
\]

The silica, Mg, and H ion concentrations in lung tissues determine whether chrysotile will tend to dissolve. The pH of the alveolar macrophage cells is comparable with an acid solution of pH 4, and the mesothelial cells are comparable with a solution of pH 7 (Jaurand et al., 1984). The pH of plasma is near 7, and its average Mg concentration is 8.7 × 10^{-4} m (range: 5.4 × 10^{-4} to 11 × 10^{-4} m) and the average Si concentration is 15 × 10^{-5} m (range: 1.5 × 10^{-5} to 28 × 10^{-5} m) (Iyengar et al., 1978; Altman, 1961). The ionic strength of plasma is about 0.12, as estimated from the Na (0.138 m) and chloride (0.102 m) concentrations (Iyengar et al., 1978). Given these ranges of silica and Mg concentrations, chrysotile would be in equilibrium with fluids in lung tissue at about pH 8. However, body fluids are constantly flushed through the system and never rise to pH 8, so the chrysotile in contact with these fluids should dissolve. The activity quotient (Q) for chrysotile is defined as

\[
Q = \frac{a_{\text{Si}^{4+}} a_{\text{H}_4\text{SiO}_4}}{a_{\text{SiO}_2} a_{\text{H}_2\text{O}}}
\]

and with the average concentrations of Mg and Si found in blood plasma, Q is

\[
1.4 \times 10^{-3} \text{ at pH 7 and } 1.4 \times 10^{-4} \text{ at pH 4.}
\]

The degree of saturation, S = Q/K, at pH 7 is 2.4 × 10^{-8}, and at pH 4 is 2.4 × 10^{-24}. Thus, from a thermodynamic perspective, solutions in lung tissues are extremely undersaturated with respect to chrysotile, and it should dissolve. Its persistence is simply a result of a slow dissolution rate.

**Experimental Methods and Results**

Chrysotile from the Bell Asbestos mine, Thetford Mines, Quebec, Canada, was cut into approximately 1-cm lengths and then ground in a tungsten carbide shatterbox for 10 min. This procedure introduced a minor amount of tungsten carbide contaminant into the sample. A size fraction of <350 μm was recovered by sieving and kept for the experiments. The specific surface area of this material was determined using a Quantachrome surface area analyzer to be 33.5 m^2/g using a three-point N₂ BET isotherm (Brunauer et al., 1938).

The experiments were conducted at pH values ranging from 2 to 6 (dilute HCl solutions) at 37°C for 3 h. At specific intervals, a sample was removed and centrifuged and the supernatant was filtered using a 0.25-μm syringe filter. The pH of the filtered solution was measured, and the silica was determined using the molybdate blue method (Govett, 1961). The Mg concentration was determined by atomic absorption spectrophotometry.

Graphs of the concentration of SiO₂ in solution vs. time (e.g., Fig. 1) were linear for values of Q between 1.45 × 10^4 and 1.45 × 10^6 (solution composition ranges for all experiments were 3.4 < pH < 7.4, 2.5 × 10^{-6} < m_{\text{SiO}_2} < 1.3 × 10^{-3}, 6.2 × 10^{-6} < m_{\text{Mg}^{2+}} < 5.8 × 10^{-5}), within the range of values expected in lung tissue. The rate of silica release is independent of the concentration of NaCl, H⁺, Mg²⁺, and H₂SiO₄. This indicates that the rate law for silica release over this range is zeroth order, i.e.,

\[
r = \frac{d[n_{\text{SiO}_2}]}{dt} = (A/M)k
\]

and the apparent reaction rate constant (k') is the slope of the silica concentration vs. elapsed time line. The integrated form of the rate law is

\[
m = m_0 + (A/M)k \Delta t,
\]

so a graph of silica concentration vs. elapsed time should be linear with a slope of (A/M)k. The normalized rate constant, k (mol/m²s), for each experiment was calculated from the following equation (Rimstidt and Barnes, 1980), where k’ is the apparent rate constant (mol/s), A is the surface area (m²), and M is the mass of solution (kg),

\[
k = k'/(A/M).\]

The results from 14 experiments with starting pH values ranging from 2 to 6 are shown in Table 1. These data were combined to find the average rate constant; k = 5.9 (± 3.0) × 10^{-10}. The entire data set is presented in Hume (1991).

**Discussion and Conclusions**

When chrysotile reacts with acid solutions, Mg leaches quickly, leaving behind a silica structure (Morgan et al., 1977; Clark and Holt, 1960). Thus, it appears that the destruction of chrysotile fibers proceeds by two steps. First, the magnesium hydroxide layer is removed by rapid leaching; then the silica layer dissolves at a slower rate. Therefore, the lifetime of a chrysotile fiber is controlled by the dissolution rate of the silica layer.

We can model (see Hume, 1991, for the complete der-
Table 1. Summary of conditions and results for chrysotile dissolution experiments

<table>
<thead>
<tr>
<th>Expt. no.</th>
<th>pH</th>
<th>$k^*$ (mol/s)</th>
<th>$A/M$ (m$^2$/kg)</th>
<th>$k$ (mol/m$^2$/s)</th>
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<td>$1.67 \times 10^{-10}$</td>
<td>0.134</td>
<td>$1.24 \times 10^{-9}$</td>
</tr>
</tbody>
</table>

Average | $5.9 (\pm 3) \times 10^{-10}$

*Experiments performed in solutions with an ionic strength of 0.12 M (NaCl).

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Significant increases in lung cancer rates occur 10–14 yr after the first exposure; significant increases in asbestosis rates occur 15–20 yr after the first exposure, and significant increases in mesothelioma rates occur 20 yr after first exposure to asbestos minerals (Ross, 1984). Thus, the average time for diagnosis and death from these diseases is at least an order of magnitude greater than our biodurability estimate for a fiber of 1 μm diameter. These are complicated diseases, and the steps in their progression are not all clear. However, we can conclude from this study that any damage to the lung tissue caused by chrysotile must take place soon after exposure, since this mineral dissolves quickly. Any model of these diseases must explain this discrepancy between chrysotile fiber lifetime and the time until the onset of disease symptoms.

The biodurability test developed here is simple to implement and interpret. The rate constants for some minerals of interest are already available in the geochemical literature. For example, the rate data from Rimstidt and Barnes (1980) can be used to estimate that the lifetime in lung tissue of a particle of quartz 1 μm in diameter would be $1.7 \times 10^6$ yr; this suggests that quartz grains in lung tissue will not change significantly over a human lifetime. Unfortunately, it seems that no rate data exist for the Fe-rich amphiboles such as crocidolite. However, once the rate data for these become available, it will be possible to assess the relationship between their biodurability and their disease-producing tendencies.

Acknowledgments

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