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minimum value during that cycle (Fig. 1C). The cycles of $\dot{V}_{O_2}$ were closely followed by oscillations in T_{th}, with the peaks in $\dot{V}_{O_2}$ slightly preceding the peaks in T_{th} (Fig. 1C). We assume that the cycles in $\dot{V}_{O_2}$ and T_{th} coincide with cycles of activity in the thoracic flight muscles. The T_{ab} values sometimes showed similar, but smaller oscillations which lagged behind those of the thorax. Our experiments usually lasted 2 to 3 hours, and during this interval the beetles were almost completely motionless and T_{th} was continuously elevated. We did not determine the maximum duration of the homeothermic response of *Megasoma*.

As long as T_b remains constant, heat production is necessarily equal to heat loss. Heat loss is proportional to the difference between T_b and T_a (ΔT); in the case of endothermic insects T_b refers to T_{th}. A mammal with a T_b of 37°C at a T_a of 23°C should have the same heat loss as a *Megasoma* of the same mass with a T_b of 27°C at a T_a of 13°C. (This parity requires that the mammal and beetle have similar thermal conductances.) It is instructive to compare the $\dot{V}_{O_2}$ (which is proportional to heat production) of a homeothermic *Megasoma* with that of placental mammals of similar mass experiencing the same ΔT (Fig. 2). For comparative purposes we have selected two small rodents whose metabolic rates were measured with an open-flow respirometry system similar to that used for *Megasoma* (*8*). The rodents, a kangaroo mouse (*Microdipodops pallidus*) and a pocket mouse (*Perognathus californicus*) have lower $\dot{V}_{O_2}$ than *Megasoma* of the same mass when ΔT is greater than 5° to 7°C (that is, at T_a below the thermal neutral zone of the mammals). Below thermoneutrality, the mass-specific thermal conductances of the mammals did not change with T_a, whereas the mass-specific thermal conductances of the beetles decreased linearly with T_a. For example, the conductance of an 11-g beetle was 0.42 ml of O_2 per gram per hour per degree Celsius at a T_a of 26°C but was only 0.30 ml of O_2 per gram per hour per degree Celsius at a T_a of 8°C. The decrease was apparently caused by a decline in T_{ab} from 27.1° to 15.5°C while T_{th} remained constant. Consequently, heat loss from the abdomen decreased with decreasing T_a.

The dearth of information on the natural history of *Megasoma elephas* makes ecological interpretation of the homeothermic metabolic response problematic. We assume that when T_b is regulated above T_a during terrestrial activity, the pattern of T_b regulation is the same as the pattern we observed in the laboratory at T_a below 20° to 22°C; that is, that variation in heat production rather than variation in heat loss to the abdomen is the primary mechanism of control of T_b. *Megasoma* is certainly capable of supporting normal activity levels by regulating a high T_b at T_a below 20° to 22°C (T_a values as low as 15°C have been recorded on Barro Colorado Island), but we do not know whether the beetles could fuel the metabolic increase required, should T_a remain low for more than a few hours.

To our knowledge, among insects, a homeothermic metabolic response to decreases in T_a in the absence of locomotor activity has only been reported in worker honey bees (*9*) and in incubating queen bumble bees (*10*). The homeothermic responses of the beetles reported here were not as fine-tuned as those of birds and mammals. In the beetles, T_b declined to about 20° to 22°C before there was any regulation. Thereafter, their metabolic response to a decrease in T_a was qualitatively similar to that of birds and mammals. However, instead of maintaining T_b constant, there was an oscillation over a range of 3° to 4°C with the frequency but not the amplitude of the oscillations increasing with ΔT. The beetles appear to have a definite set point below which they "defend" T_b. The greater the ΔT, the more rapid the heat loss and the more rapid the oscillations in $\dot{V}_{O_2}$ and T_b.

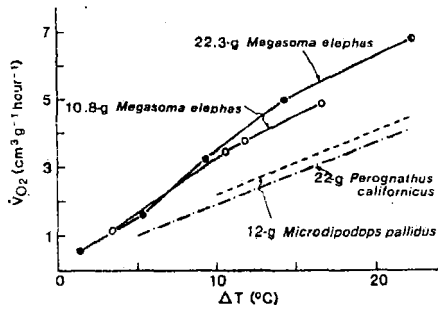


Fig. 2. A comparison of the relation of mass-specific $\dot{V}_{O_2}$ to ΔT in scarab beetles and heteromyid rodents of similar size. The curves for rodents are the regressions of their euthermic energy metabolism below the thermal neutral zone (*8*). The points for the beetles are the means of 16 or more measurements taken while T_{th} was constant at a given T_a.

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Indoor Airborne Asbestos Pollution: From the Ceiling and the Floor

Abstract. *Electron microscopic measurements of the concentrations of airborne asbestos were carried out inside and outside an office building having ceilings sprayed with a crocidolite-containing material and floors covered with vinyl-chrysotile tiles. Under normal conditions in this building, constructed 10 years ago, the two asbestos-containing materials released fibers into the air. This is the first measurement of elevated (up to 170 nanograms per cubic meter) concentrations of indoor airborne asbestos associated with the weathering of asbestos floor tiles during their service life. Asbestos flooring is used in a large number of buildings and represents the third largest use of asbestos fibers in the United States and in Europe, ranking after roofing and asbestos-cement pipe.*

The potential hazard arising from exposure to airborne asbestos inside buildings sprayed with asbestos-containing materials is currently the subject of much concern. The Environmental Protection Agency has undertaken a large-scale program to control friable asbestos-containing materials in school buildings (*1*). The Conseil Supérieur d'Hygiène Publique de France has recommended controlling the concentrations of airborne asbestos inside buildings sprayed with asbestos (*2*). Since 1975, analytical facilities for the measurement

of asbestos, with an analytical transmission electron microscope (ATEM), have been available in our laboratory (*3*). Thus far, 493 air samples from 122 buildings have been analyzed. In December 1980, we were asked to monitor the airborne asbestos within a building, the ceilings of which had been sprayed in 1970. In this building, an additional and unexpected emission source of airborne asbestos was discovered: the asbestos floor tiles.

The building, located 20 km south of Paris, was occupied by an engineering company that was marketing optimized electronic computer systems. The 300 workers were all white-collar employees, working under clean conditions inside offices, graphic studios, and workshops where electronic machines were being tested. The six floors (900 m^2 each) showed similar architectural configurations with peripheral workrooms and a central core occupied by elevators, stairs, corridors, and blind storerooms. Measurements of the indoor airborne asbestos pollution were requested because of the presence on the corridors' ceilings of a suspicious, friable, blue material that had been sprayed on. This material was located in an open plenum where it could be eroded by the circulating return air. Another possible cause of deterioration was the fact that electricians sometimes operated on the plenum electric lines.

We took five bulk samples by penetrating the total depth of the sprayed material with large-mouth containers in five randomly predetermined locations. Analysis of their fiber content with the polarized light microscope (PLM) revealed that they were almost pure crocidolite (*4*, *5*). Neither man-made mineral fibers nor chrysotile were detected. Further analysis with the ATEM (Fig. 1, a and A) did not reveal the presence of fine chrysotile fibers unresolved by the PLM.

Air was sampled simultaneously at four indoor sites and on the roof where fresh air entered the air-conditioning system. Air-sampling units had been operating for five consecutive working days but only during building activity periods. Air sampling during activity periods is of primary importance, since it has been observed that the concentrations of indoor airborne asbestos measured during periods of active use are much higher than those measured during nonuse periods (*3*). It is believed that human activity not only contributes to the degradation of materials but is also a factor in the fragmentation and secondary dispersal of settled dust (*6*).

Airborne asbestos measured outside the building showed the usual features encountered in the urban area of Paris and its suburbs. Only chrysotile was encountered, with a mass concentration of 0.1 ng/m^3. Earlier measurements of 249 air samples from 35 locations in this area revealed a homogeneous background of ambient airborne chrysotile. Concentrations were in the range of 0.1 to 9 ng/m^3 (median, 0.4 ng/m^3), with 95 percent below 3 ng/m^3. Commercial amphibole-type asbestos fibers have not as yet been detected in significant quantities in urban air samples.

Inside the building, both crocidolite and chrysotile were present in the air samples (Fig. 1B). Concentrations were 0.2, 0.5, 0.9, and 33 ng/m^3 for crocidolite and 8, 21, 25, and 170 ng/m^3 for chrysotile at sites 1, 2, 3, and 4, respectively. Sites 1, 2, and 3 were corridors; site 4 was a workshop including part of a corridor. The concentrations rose from site 1 to site 4 in the same order for crocidolite as for chrysotile.

Obviously, the airborne crocidolite fibers within the building were released from the crocidolite-containing material sprayed on the ceilings. The concentrations can be compared with those measured earlier inside a series of 36 buildings sprayed with a material containing amphibole-type asbestos, generally amosite or crocidolite (Fig. 2). Fifty percent of such buildings showed detectable concentrations of amphibole-type asbestos fibers in the air; only 22 percent of the buildings showed concentrations in excess of 33 ng m^3, which was the maximum concentration of amphibole-type asbestos fibers in the air of the building reported here. Bearing in mind these reference data and because the architec-

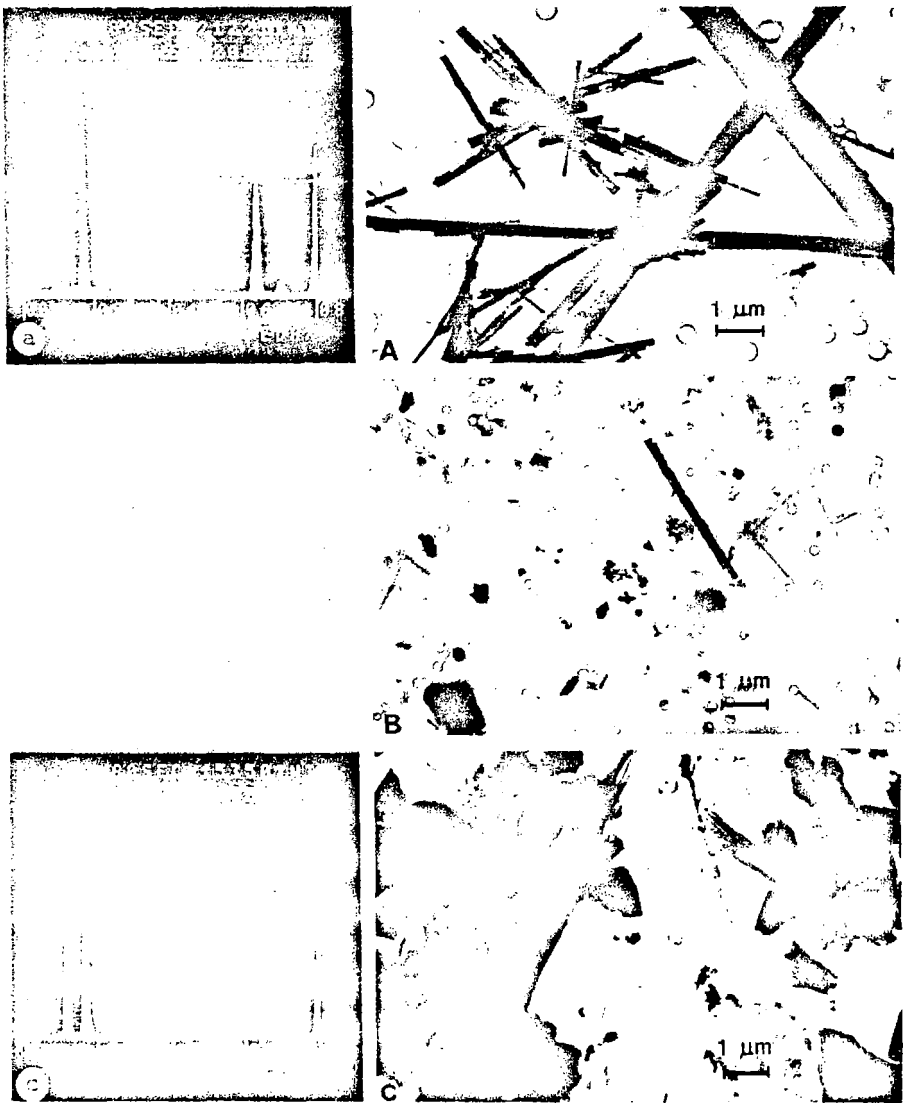


Fig. 1. Characterization with the analytical transmission electron microscope [a conventional transmission electron microscope fitted with an x-ray energy-dispersive spectrometer (EDS)] of asbestos fibers isolated from the material sprayed on the ceilings (a and A), an air sample (B), and a vinyl floor tile (c and C). The EDS spectra of individual fibers are typical of crocidolite (a) and chrysotile (c). Fibers of both asbestos types were encountered in the air samples (B).

tural configuration of the building was not favorable, we recommended that some corrective action be taken (*7*) to prevent further crocidolite fibers from becoming airborne.

The elevated concentrations of indoor airborne chrysotile were very surprising in view of the absence of detectable chrysotile in the sprayed material. A further warning sign was the shortness of the airborne chrysotile fibers; no fiber longer than 3 μm was encountered on the membrane filters. Further investigations were undertaken to discover the origin of such fibers. Inquiries revealed no use of asbestos products by the workers. The air-conditioning system was carefully investigated; no asbestos-containing material was discovered. Water used to dampen the conditioned air was analyzed with the ATEM; no chrysotile fiber was detected. The only material in the building that was found to contain chrysotile fibers was the vinyl floor tile. The fiber content of a floor tile was analyzed with the ATEM and showed very abundant chrysotile fibers (Fig. 1, c and C). The owner of the building confirmed that the 5400 m^2 of floor were covered with asbestos vinyl tiles. These tiles were black and showed a decorative relief on their surface. Damage to some tiles was clearly apparent (Fig. 3). In the absence of any other explanation, we concluded that the short chrysotile fibers present in the air of the building had escaped from the vinyl matrix of the floor tiles. The fact that the highest concentrations were measured in the workshop on the third floor, where the activity was greatest, reinforced our conclusions. It might be hypothesized that, in this workshop, breakdown forces such as walking, scraping, and machine scrubbing were higher than in other places. High activity might also be an explanation for the higher concentration of airborne crocidolite at this site. The company that occupies this building now intends to cover the asbestos floor tiles with another flooring material.

It is well known that the building industry is the primary consumer of asbestos products, such as asbestos-cement pipes, asbestos-cement sheets for roofing and cladding, asbestos vinyl and asphalt floor tiles, insulation products, special-purpose decorative textiles, and asbestos-coating and paint compounds (*8*). The interest of environmentalists in possible indoor airborne asbestos pollution caused by the weathering of asbestos-containing materials began in the 1970's. Considerable attention has been devoted to buildings insulated with sprayed asbestos, where the concentrations of indoor airborne asbestos can be several orders of magnitude higher than the ambient background (*3*, *9*). Some countries such as the United States and France have now banned the use of spray-applied asbestos-containing materials for insulation in buildings.

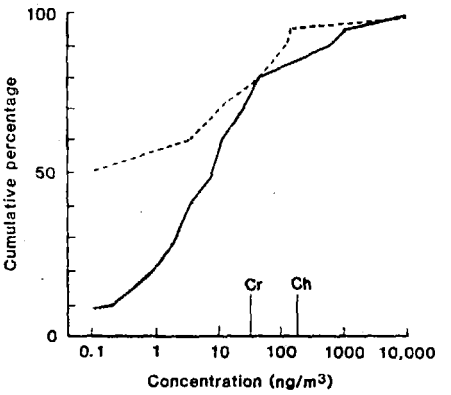


Fig. 2. Distribution of maximum concentrations of airborne amphibole-type asbestos fibers measured inside 36 buildings sprayed with a material containing amphibole-type asbestos (dashed line). Corresponding curve for chrysotile asbestos inside 58 buildings (solid line). The vertical lines indicate the maximum concentrations of airborne asbestos (*Cr* for crocidolite and *Ch* for chrysotile) fibers inside the building examined in this study.

It has been reported that the sanding of old asbestos floor tiles that are to be replaced is an important source of asbestos exposure (*10*). To our knowledge, this report presents the first measurement of elevated concentrations of indoor airborne asbestos associated with the weathering of asbestos floor tiles during their service life. Daly *et al.* (*11*) have predicted that the average asbestos concentration in the air above an asbestos floor in service can theoretically be increased by 80 ng/m^3. Some concentrations measured in this particular building were close to the predicted level of 80 ng/m^3. Up to now, in spite of this prediction, the presence of asbestos flooring within a building has not been considered a serious source of potential indoor airborne asbestos. In the Environmental Protection Agency's control program in schools (*1*), the nature of the floor is not taken into consideration. In their survey of the asbestos-containing materials in 336 schools in Vermont, Novick *et al.* (*12*) did not report the presence of asbestos floor materials. In France, up to now, our measurement program in buildings was not designed to document the presence or absence of asbestos floor tile.

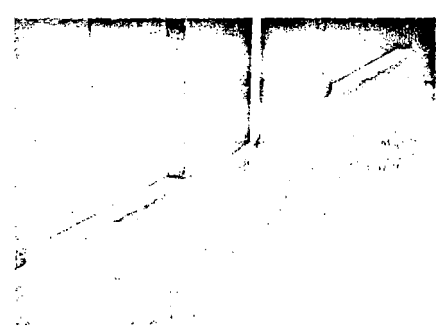


Fig. 3. View of the asbestos vinyl flooring in the building. Note the toning down of the black color and of the surface relief, as well as scraping marks.

The weathering of asbestos flooring under normal use patterns can yield concentrations of indoor airborne asbestos similar to those measured in sprayed buildings (Fig. 2), which are the subject of much concern. In 1976, flooring materials represented about 15 percent of the total U.S. market for asbestos, making it the third largest asbestos consumer, after roofing and asbestos-cement pipes (*8*). If this first measurement is confirmed in other places and by other laboratories, the use of asbestos flooring, a very common practice, could become an important environmental issue.

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York River Destratification: An Estuary-Subestuary Interaction

Abstract. *Destratification in the York River during high spring tides is the result of the interruption of normal two-layer estuarine flow by the advection of relatively fresh water into the river mouth from the Chesapeake Bay. This advection is due to the presence of a longitudinal salinity gradient in the bay and a difference in the tidal current phase between the river and the bay. Similar behavior is seen in other subestuaries of the Chesapeake Bay and may be common in subestuary-estuary interactions.*

Vertical homogeneity in subestuaries of the Chesapeake Bay, which normally exhibit moderate stratification, has been shown by Haas (*1*) to be correlated with high spring tides. Identified by a surface-to-bottom salinity difference of less than 1 per mil in contrast to normal values as high as 10 per mil, these episodes have been termed destratification events (*2*). For example, the lower York River becomes vertically homogeneous 3 to 4 days after the predicted tide height exceeds 0.8 m (*1*), and homogeneity persists for three or more days. These events are not correlated with changes in the flow of fresh water into the river (*1*). Some of the significant consequences of destratification events in the York River include a periodic resupply of oxygen in the bottom water with an accompanying renewal of nutrients near the surface (*3*) and changes in primary productivity including blooms of dinoflagellates and other phytoplankton (*4*).

Two theoretical discussions of spring-neap tidally related stratification variations have been presented (*5*). Both of these models describe reductions in stratification that coincide with the occurrence of strong tidal currents and do not persist in the absence of such currents. This coincidence is in contrast to destratification in the lower York River, where vertical homogeneity first appears a few days after the onset of strong tidal currents and persists for several days thereafter (*1*, *2*).

A conceptual model for the onset and disappearance of vertical homogeneity in the York River is as follows. (i) Destratification commences when spring tides exceed a critical height and relatively fresh water from the Chesapeake Bay is advected into the mouth of the York River. (ii) This produces a reduction or possibly a reversal of the pressure gradient driving estuarine circulation. The concomitant diminution of two-layer circulation reduces the tendency toward stratification by limiting the importation of more saline bottom water. (iii) This permits the establishment of homogeneity by the unopposed action of normal mixing processes enhanced by strong spring tidal currents. (iv) Destratification ceases when the decrease in tide height after spring tides halts the advection of the fresher water into the York River. (v) This allows the reestablishment of a normal horizontal salinity gradient, which produces the eventual reinitiation of two-layer estuarine circulation and vertical salinity stratification.

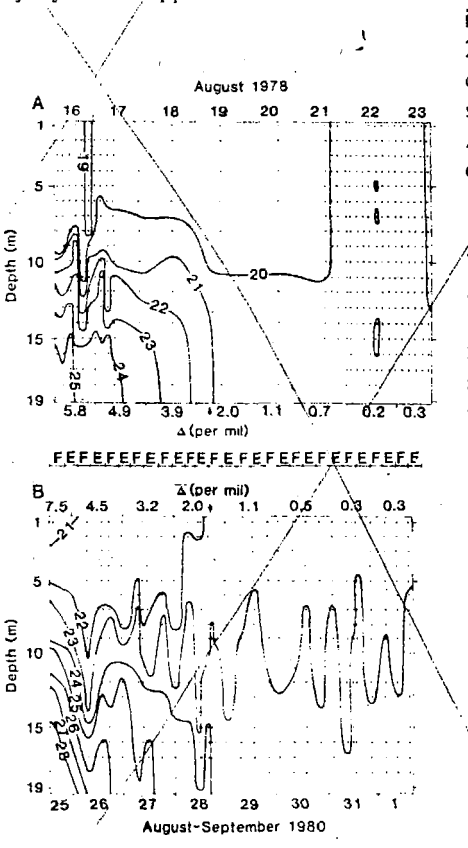


Fig. 1. Salinity data (per mil) from the York River mouth for the periods 16 throught 23 August 1978 (A) and 25 August through September 1980 (B). The arrows indicates the dates of maximum spring tide; $\bar{\Delta}$ is the daily mean of differences in salinity from 1 m to the bottom. Periods of ebb (*E*) and flood (*F*) are indicated. Points indicate measurements (*11*).

This hypothesis developed from an examination of salinity data (Fig. 1, A and B) collected during intensive studies of two destratification events that were predicted on the basis of earlier work (*1*, *2*). The intrusion of relatively fresh water, which initiated the destratification process, is indicated by the sharp downward displacement of isohalines on 16 August 1978 and 26 August 1980. In each case, this was followed by a progressive reduction of stratification in the water column. As expected, the introduction of fresher water into the river mouth caused a reversal of the longitudinal salinity gradient, producing a midriver salinity maximum. This condition is illustrated in Table 1, where York River salinity values at 1-m depth are shown for the period from 0 to 3 days after the intrusion of fresher water observed on 25 August 1980 (Fig. 1B). The data indicate a reversal of the normal longitudinal salinity gradient as far as 18 km upriver. A similar reversal was observed on several occasions between 15 and 20 August 1978 (*2*).

The reversal of the longitudinal salinity gradient is also evident from the behavior of the isohaline at 23 per mil in the 1980 data (Fig. 1B). The assumption is made that the salinity changes at the station are caused in large part by the advection of water of differing salinity up and down the river by tidal currents. Depressions in the isohaline, indicating the presence of fresher water, coincide with slack before ebb through 28 August, the date of highest tidal heights. After that date, the isohaline at 23 per mil shows a phase reversal. The depressions are coincident with slack before flood, indicating the reestablishment of the normal longitudinal salinity gradient. While tidal heights were increasing, there was a continuing source of fresher water. As tidal heights recede, more saline water is once again present in the river mouth.

The only reasonable source for the relatively fresh water is the Chesapeake Bay. An upriver source is discounted first because the water is introduced into the river mouth on flood tide (Fig. 1B) and second because the nearest riverine source of water of comparable salinity is