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Biogenic amorphous silica (BAS) is a natural constituent of living matter (eg, unicellular organisms and crop plants). Diatoms, whose siliceous remains are the geologic precursors to diatomaceous earth, actively process soluble silica into BAS. In some plants, a portion of the BAS exists externally as pointed or irregularly shaped fibers. Although silica-related adverse health effects are usually attributed to crystalline forms, such effects could occur as a result of exposure to BAS at high temperatures (above 800°C), where crystalline silica, a known human toxicant is formed. BAS fibers from food crops can be ingested and lead to adverse health effects due to irritative processes. Airborne BAS fibers from rice can be inhaled during burning or incineration. Fibrous or nonfibrous BAS can adsorb toxic organic compounds and facilitate their entry into the lung. Recommendations for research are suggested to address the issue of potential health effects due to exposure to BAS.

Key terms biogenic amorphous silica, crop plants, diatomaceous earth, environmental exposure, fibers, inhalation, occupational exposure, review.

Amorphous silica exists in more than one form. Some forms are associated with nonvegetative matter, such as volcanic glasses and various manufactured sols, gels, powders, and glass fibers (1—3), while other forms (ie, biogenic amorphous silica) are found in living matter [eg, viruses, bacteria, fungi, diatoms (a group of algae), sponges, and plants] (1, 4). For the current discussion, the focus will be on the biogenic amorphous silica (BAS) associated with fossilized diatoms and crop plants.

Amorphous silica is considered less toxic than crystalline silica, to which are attributed the silica-related adverse health effects, silicosis, and lung cancer (5). Amorphous silica is currently not classifiable as to its carcinogenicity to humans (5). The noncancer toxicity associated with amorphous silica is more ambiguous than for crystalline silica, although in vitro cytotoxicity and in vivo lung lesions have been observed with some industrial amorphous silica in rats (6, 7). The current threshold limit value for amorphous silica is 10 $mg \cdot m^{-3}$ (total dust) for uncalcined diatomaceous earth, precipitated silica and silica gel; 2 $mg \cdot m^{-3}$ (respirable dust) for fumed silica; and 0.1 $mg \cdot m^{-3}$ (respirable dust) for fused silica (8).

Exposure to amorphous silica from diatomaceous earth and crop plants may present unique situations not examined in most studies. The purpose of this paper is to describe the conditions under which human exposure to amorphous silica from diatomaceous earth and crop plants occurs. Where information is available, potential exposure-related health effects are discussed. Recommendations for research are also suggested.

Diatomaceous earth

Diatomaceous earth is the geologic product of decayed unicellular organisms called diatoms (1, 9). As part of the normal life cycle, diatoms take up soluble silica, probably as silicic acid [$Si(OH)_4$] from the surrounding water and transport it across the plasma membrane in saturable, energy-dependent steps (9, 10). The transported $Si(OH)_4$ then undergoes a series of reactions that ends in biomineralization producing biogenic amorphous silica and deposition in the diatom valve (frustule). The BAS levels in diatoms vary with species and range from less than 1 to almost 50% by weight (1). Over geologic time, the siliceous frustules of the diatoms accumulate and become diatomaceous earth.

Exposure to amorphous silica in diatomaceous earth occurs among workers who process the raw material into manufactured products. During a heating (calcining) step at temperatures of 982—1093°C, which includes the addition of a flux agent such as sodium carbonate, some amorphous silica in diatomaceous earth is transformed into a polymorph of crystalline silica, cristobalite (11). The temperature-induced transformation is a known phenomenon and has been studied with industrial silicas, including precipitated and vitreous silicas (12, 13). Hence, at the heating step of the manufacturing cycle, diatomaceous earth workers can experience inhalation exposure to high levels of respirable cristobalite and can therefore be at risk for adverse pulmonary health effects associated with this silica polymorph (14).

Crop plants

Like the silica in diatoms, the accumulation and deposition of silica in certain vascular (higher) plants is a normal biological process. Little is known, however, about the specific steps in uptake, accumulation, and deposition. For the grasses (including rice and sugar cane), silicic acid is taken up from the soil by active transport or passive diffusion (1). In rice, the concentration of

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silicic acid in the vascular tissue may reach 650 ppm as it is transported towards the leaves (4).

The amorphous silica in rice undergoes a temperature-dependent transition to crystalline silica (cristobalite), similar to that observed for diatomaceous earth. When rice hulls are incinerated at temperatures above 800°C, cristobalite is formed, and the conversion to this silica polymorph is prevented when furnace temperatures are lowered (15). If precautions are not taken, cristobalite can be inhaled by facility workers in proximity to the ash as it is removed from the baghouse or where it is stored on site. A potential for environmental exposure also exists if uncovered ash is disturbed by high winds.

Some of the amorphous silica in plants exists as fibers, and the fibrous forms vary by crop. In wheat and millet, silica fibers exist externally as regular hair-like projections with sharp pointed ends (4, 16). In sugar cane and rice the fibers are less regular, sometimes resembling twigs (17, 18). Concern about amorphous silica fibers is based on the adverse health effects associated with exposure to asbestos mineral fibers, particularly those with aspect ratios (length divided by diameter) greater than three. Although extensive epidemiologic data exist on the relationship between exposure to asbestos fibers and human disease (2, 19), little health research has been done on exposure to fibers of biogenic amorphous silica. Sinks et al (20) reported the absence of mesothelioma among Hawaiian sugar cane workers; however, other pulmonary end points were not addressed.

Exposure to fibers of amorphous silica may occur environmentally through the ingestion of silica-containing plants. The suggestion has been made that in the parts of the world where such plants are a staple food item, high esophageal cancer rates may be related to the ingestion of the amorphous silica fibers (16). However, the results of a mouse bioassay were contradictory (21). Skin tumors developed after direct dermal exposure to the amorphous silica from the grain or to the fiber-bearing grain, whereas gastrointestinal tumors did not occur after the latter was ingested. Although the mean aspect ratio of the fibers from the grain was 10 before the grain was added to the diet, the value after the grain was ground was not reported. The evidence thus suggests that tumor formation in relation to amorphous silica exposure is probably at the level of irritative and reparative processes.

Inhalation exposure to amorphous silica fibers occurs during harvesting, crop burning, or incineration. Such exposure can be occupational or environmental. Environmental exposure would occur during crop burning due to the air transport of residual particulate matter. Airborne fibers with aspect ratios greater than three were determined during sugar cane burning (17), rice straw harvesting and burning (18), and rice hull incineration (22). These fibers were not equivalent, however, to the long, thin, pointed structures associated with the external tissue of many grasses.

To date, laboratory studies on the biological effects of amorphous silica fibers have not been carried out in large part due to the unavailability of sufficient material. An epidemiologic study (23) on the respiratory health of California rice farmers has taken place, and the preliminary X-ray evidence is consistent with exposure to respirable silica dust. The nature of the specific exposure giving rise to the X-ray findings could not be determined however.

Interactions

The foregoing discussion has been restricted to exposure to amorphous silica separate from simultaneous exposure to other contaminants, although such exposures are known to occur. For example, polycyclic aromatic hydrocarbons (PAH) are detected in the smoke from rice straw burning in California, and they may be partially responsible for the mutagenesis observed in standard bacterial assay systems (24). Dioxins and dibenzofurans are also found in the smoke from rice straw burning in Japan (25). Particulates, in general, may facilitate the uptake and distribution of toxic substances, such as the carcinogenic PAH to the lung. One result may be enhanced tumor formation, as observed by Saffiotti et al (26) and Kimizuka et al (27). Other changes, such as increased retention time in the lung and altered PAH metabolite profiles (28, 29), may also occur.

Although the mechanisms for the particle-associated enhanced or altered PAH-induced biologic effects are not understood, in vitro studies may provide some insight. The facilitated transport of a PAH across lipid bilayers or isolated cell membranes was demonstrated if the PAH is first adsorbed onto amorphous silica particles that are respirable (30, 31). Hence the presence of PAH (and perhaps other organic compounds) in the environment of a population exposed to amorphous silica could exert interactive influences on the biological outcome. In the case of exposure to amorphous silica, the presence of organic toxins should be taken into consideration when the relationship between amorphous silica exposure and adverse health outcomes is studied.

Research needs

Many gaps exist in the knowledge about the relationship between exposure to amorphous silica and potential health effects. To assess the health risk due to exposure to amorphous silica adequately, studies are needed on the mechanism of disease processes associated with exposure to silica in general and to biogenic amorphous silica in particular. For example, more research is needed on the consequences of the interactions between soluble toxins and respirable silica particles. More information is also needed on exposure levels of amorphous silica. To address this concern, improved methods for preparing large amounts of purified amorphous silica fibers need to be developed. The result from studies such as the ones suggested in this presentation will add to our knowledge of the role of amorphous silica in silica-related disease and enable risk assessors to evaluate risks due to exposure to amorphous silica more rationally.

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