MICHIGAN DEPARTMENT OF ENVIRONMENTAL QUALITY

INTEROFFICE COMMUNICATION

March 10, 1998

TO: File for Kaolin (CAS No. 1332-58-7)

FROM: Michael Depa, Toxics Unit, Air Quality Division

SUBJECT: Screening Level Determination

The initial threshold screening level (ITSL) for kaolin is 20 µg/m³ based on an 8-hour averaging time.

The following references or databases were searched to identify data to determine the ITSL: IRIS, RTECS, ACGIH Threshold Limit Values, NIOSH Pocket Guide to Hazardous Chemicals, Environmental Protection Bureau Library, IARC Monographs, CAS Online (1967 - April 1, 1997) National Library of Medicine, Health Effects Assessment Summary Tables, and NTP Status Report. Review of these sources found that EPA has not established an RfC or RfD for kaolin. A search of Current Contents was also performed. Current Contents covers the periodical literature from 1993 to September 1997. The ACGIH and NIOSH have established occupational exposure limits (OELs) for the respirable fraction of kaolin at 2 mg/m³ and 5 mg/m³, respectively. The epidemiological literature was reviewed in order to determine whether the TLV of 2 mg/m³ is protective of occupational pneumoconiosis caused by respirable kaolin exposure.

Kaolin, or china clay, is a hydrated aluminum silicate ($Al_2O_3 \cdot 2SiO_2 \cdot 2H_2O$). Kaolin is an altered product of granite, and deposits usually consist of a soft aggregates of sand, mica, and kaolinite; the latter in the form of microscopic plate-like crystals consisting of aluminum silicate (Altekruse et al, 1984). The term clay does not refer to any precise chemical composition and kaolin may contain other constituents, such as silicon, iron, titanium, calcium, magnesium, copper and sulfur (Altekruse et al, 1984).

Most studies of kaolin mine workers show increased incidence of pneumoconiosis. However, these studies lack quality long-term exposure measurements, a major problem when using epidemiological studies for a quantitative risk assessment. One author (Sheers, 1989) stated that dust measurements were started in 1965 but a satisfactory standard of technique including personal sampling was not achieved until 1978. In a Georgia kaolin mine, Altekruse et al. (1984) showed that from 1977 to 1980, personal samples of respirable dust levels in the processing areas ranged from 4 to 5 mg/m³, but after 1980 dropped to about 1 mg/m³. Another problem with some kaolin exposure assessments in the past is the failure to quantitate crystalline silica content of the dust exposure. Crystalline silica is thought to be much more potent than kaolin in its ability to cause pneumoconiosis. Overall, there seems to be general agreement in the literature that the dust levels before 1971 were higher and that the working conditions have steadily improved, “as a result of enclosure, automation, and remote control, together with better housekeeping and personal protection” (Sheers, 1989).

In addition to poor exposure assessments in the past, the delayed onset of pneumoconiosis is also a problem with a kaolin dust risk assessment. This is important because the exposure levels in kaolin mines and processing areas has fallen recently and the cases of pneumoconiosis seen currently are thought to be a result of historically high exposures. In one study (Sepulveds et al, 1983a), the mean cumulative years of kaolin exposure in 8 workers with pneumoconiosis was 28 years. The authors stated that this, "supports the general notion of long exposure and latency period in this disease." The problems with effective kaolin risk assessments have been recognized by Sheers (1989) who said,

> [P]redictions of the risk of pneumoconiosis in post-1971 working conditions are based on relatively short exposures and will need to be readdressed after a further ten years before conclusions can be drawn with confidence.

Because of the problems of exposure measurements and the long latency period of kaolin pneumoconiosis, a robust quantitative risk assessment based on current epidemiological data is not possible at this time. Further analysis of the literature should be performed when better epidemiological studies of kaolin pneumoconiosis become available.

EPIDEMIOLOGICAL STUDIES

In 1990, an epidemiological study was performed in china clay workers in the United Kingdom (Rundle et al., 1993). The same study is also reported by Comyns et al. (1994), and as the authors state it confirms and extends earlier surveys in the same sample population (see Ogle et al., 1989). Unfortunately none of the studies of the kaolin dust exposed population cited above examined the co-exposure of kaolin and crystalline silica, an important consideration since both can cause similar forms of pneumoconiosis. A total of 4401 china clay workers participated in the study representing 70% of the then current employees and 17% of the pensioners. The survey consisted of x-ray film, lung function measurements and a questionnaire on respiratory symptoms and smoking habits. An estimate of total exposure was derived from representative dust concentrations for each job. Work histories were used to calculate the cumulative exposure which the authors defined as total occupational dust dose (TODD). These were based on measured dust concentrations after 1978 and on estimates before 1978. Concerning the measurement of exposure, the authors stated that a number of assumptions were necessary in assigning exposures to early employment before regular measurements began and these will gradually become less important as time goes on and the proportion of measured dust data increases. The authors made no attempt to criticize the sensitivity of their exposure estimate before 1978. Although both total and respirable dust were measured the authors chose to use TODD as the measurement of exposure. RESULTS: The authors concluded that a non-smoker will have on average reached the lower limit of major x ray film Category 1* at the age 60 after a total exposure of 85 mg/m^3-years (working

* Category 1 pneumoconiosis as identified by radiography is defined as: small opacites are present, but few in number; the normal lung marking (i.e., the images of the vascular structures) are usually visible. There are four categories of pneumoconiosis with Category 0 being the least severe and Category 4 the most severe.

continuously for 42.5 years) at an average dust concentration of 2 mg/m³ TODD. For a smoker, the total exposure would have to be less than 65 mg/m³-years, or less than an average exposure of 1.5 mg/m³ TODD. The authors stated that, “Dust concentrations have been reduced in recent years, averaging 1.7 mg/m³ for dryers in 1990 compared with 3.5 mg/m³ in 1978.” Furthermore, “The percentage of people with small opacities greater than major x-ray film category 1 was 0.8% (lower than in previous studies).”

A cross-sectional survey study was performed at a kaolin clay mine in Georgia, USA (Altekruse et al., 1984). The investigators conducted this epidemiological study to determine (a) kaolin dust concentrations in the various areas of kaolin production; (b) the relationship of kaolin pneumoconiosis to dust exposure and work history; and (c) the extent and type of any pulmonary impairment present in men with kaolin pneumoconiosis. Sixty-five workers employed in the processing (or “shed”) and mine areas were used in the study. Personal air samples were collected at various intervals from 1977-1981. The workers wore the sampling kit for eight hours. In 1980, the mean respirable dust level in the processing area was roughly 5.2 mg/m³ and 1.0 in the mine area. In 1981, the mean respirable dust level in the processing area had fallen to 1.74 mg/m³ and was 0.14 mg/m³ in the mine area. The mineral analysis of the dust showed the composition to be 94-98% kaolinite and 2-6% anatare (TiO_2) with no silica (either crystalline or amorphous). RESULTS: Five workers, all of whom had worked at the processing area, had radiographic evidence of kaolin pneumoconiosis. When the spirometric values were expressed as a percentage or the predicted values, the FVC and FEV_1 were significantly lower in the workers with kaolinosis than in other workers in the processing area. The FVC and FEV_1 also declined significantly with increasing years of work in the processing area. The authors stated that, “The FEV_1/FVC%, however, was not significantly altered either by the presence of kaolinosis or by an increasing number of years of work, indicating that the impairment was restrictive and hence likely to be a consequence of dust inhalation rather than smoking.” In this study the highest dust concentrations occurred in the processing area, and kaolin pneumoconiosis was limited to those who had worked there. All 5 men with pneumoconiosis worked in the processing area greater than 7 years. One of these workers had complicated pneumoconiosis and had worked his entire working life in the processing shed (36 years). Another worker with pneumoconiosis with the shortest exposure (7 years) had the least radiographic abnormality. The authors stated that measurement of dust levels before 1977 had been irregular and infrequent but that an earlier survey of several kaolin plants in Georgia showed dust concentrations over a hundred million parts per cubic foot. No conversion factor from parts per cubic foot to mg/m³ was offered. The authors concluded, “Thus there is little doubt that before regular dust sampling began dust concentrations had been far higher.” The authors stated that since there was no reliable data on dust concentrations for previous years, they did not feel justified in attempting to derive a cumulative dust exposure for each worker. The authors addressed the complication that cigarette smoking causes, “Since all five workers with pneumoconiosis were cigarette smokers, it is difficult to separate the contributions of smoking and pneumoconiosis.” The authors hypothesized that, “Cigarette smoking has been thought to delay dust clearance and thus increase the incidence of dust related pneumoconiosis.” The authors concluded that kaolin pneumoconiosis is associated with only mild effects on pulmonary function,

and that the complete absence of silica indicates that inhalation of kaolin alone can lead to pneumoconiosis.

In June 1981, 65 of the then current and former workers at a Georgia kaolin mine and mill were examined by chest radiography, spirometry, and a questionnaire (Sepulveda et al., 1983). Breathing zone samples of respirable dust and area samples of total dust were collected. Airborne dust was found to be composed of kaolinite (96%) and titanium dioxide (4%). Each personal respirable dust sample (n = 44) was analyzed for crystalline silica and none was detected. RESULTS: Five (13%) of 39 current workers and 3 (19%) of 16 former workers with 5 years or more of exposure had radiographic evidence of pneumoconiosis. The authors stated, “Seven of the 8 workers with pneumoconiosis had dust exposure limited to mill work, and mill tenure was largely in the dustier occupations.” The other employee with pneumoconiosis worked in the open-pit mine work area. Lung function tests showed significant reductions ($p<0.05$) in FVC, FEV, and peak flow rate in kaolin workers compared with that in a control group. In all job categories, mean exposure values were below the respirable nuisance particulate threshold limit value of 5 mg/m³. The respirable dust concentration was roughly 1 mg/m³ (n = 3) in the mill area during the days of the health testing. The average respirable dust concentration for the miners was 0.2 mg/m³. The total years of kaolin exposure in the 8 workers with pneumoconiosis was 15 to 39 years. The authors stated, “Workers with pneumoconiosis had also been employed at the facility at a time when the work environment was reported to have been considerably dustier.” Pneumoconiosis was found exclusively in workers who were current or former cigarette smokers. The authors stated that, “Meaningful correlations of lung function and symptoms by radiographic severity of pneumoconiosis were not possible in this study because of the small size of the population examined.”

ANIMAL STUDIES

In an animal study, 2 groups of 45 female Wistar rats were exposed to 0 or 300 mg/m³ respirable Cornish kaolin dust for 6 hours a day, 5 days a week for 3 months (Wastiaux, 1990). X-ray defraction and infra-red spectroscopy did not detect any crystalline silica in the sample. The pulmonary response was assessed on 8 animals at month 3, 6, 12, 24 and 28 after the beginning of the experiment. Before month, no significant gross changes were noticed in the lungs. Macroscopic tumorous lesions were present in one animal at month 24 and in three animals at month 28. At month 3, the mean weight of fresh lungs was slightly increased and this difference persisted until month 28. At month 3, the main histopathological lesions were lipoproteinosis and alveolitis with numerous dust-laden macrophages in the alveolar spaces. At month 12, epithelial hyperplasia and bronchiolar metaplasia were visible associated with interstitial fibrosis. Multinucleated giant cells granulomas were also encountered. At month 28 three squamous carcinomas were found among the nine animals. At the end of the exposure period (three months) the amount of kaolin retained in the lung averaged 20.7 mg per rat, with very slow clearance afterwards. At month 28, 17.9 mg per rat was measured in the lungs. Formed collagen increased from an average of 8.9 mg per rat at month 3 to 21.7 mg per rat at month 28. A progression of lesions was observed from macules to

interstitial fibrosis and collagenized granulomas. The author concluded that kaolin exhibited moderate fibrogenicity.

The ACGIH documentation for kaolin summarized two studies where test animals were exposed to air born concentrations of kaolin dust. ACGIH summarized the Gross et al. (1960) study where guinea pigs were exposed to 23.4 mg/m³ kaolin for 6 hours/day, 5 days/week for 1 year. ACGIH mentioned that the lymph nodes contained large masses of dust cells. Rats exposed at 27.1 mg/m³ developed scattered dust foci; collagenous fibers were also observed between the cells and the lymph nodes contained large masses of dust. The silica content of the kaolin was not specified.

The ACGIH also summarized an unpublished study performed by the United Kingdom Mining Research Council (MRC). Forty Fischer 344 SPF rats were exposed to 10 mg/m³ of either kaolin-filter grade or kaolin-coating grade dust. The animals ere exposed 7 hours/day, 5 days/week for up to 24 months. Four rats were sacrificed and examined every 4 months. The most severe pulmonary change observed was classified as grade 4 fibrosis (minimal) which showed minimal collagen deposit at the level of terminal bronchioles and alveolus. ACGIH stated that the increased bronciolization and associated mucoid debris suggested a glandular pattern. The authors concluded that occupation kaolin exposure would be unlikely to produce severe pulmonary disease.

DISCUSSION

The available scientific literature and the ACGIH documentation for threshold limit values (TLVs) were reviewed in order to determine the adequacy of the TLV for kaolin. The ACGIH TLV is 2 mg/m³ for respirable kaolin dust containing no asbestos and less than 1% crystalline silica. As mentioned above there are several problems with the kaolin pneumoconiosis database that hinder a fully defensible risk assessment. Inadequate exposure assessments are the biggest impediment to quantitating the risk of respirable kaolin exposure. Since Rundle et al. (1993) did not analyze the respirable dust for crystalline silica content this study cannot be used to assess the adequacy of the TLV. Altekruse et al. (1984) and Sepulveda at al. (1983) did not assess pre-1977 kaolin dust exposures, therefore, these studies cannot be used for a quantitative risk assessment of kaolin. However, Altekruse et al. (1984) identified a case of kaolin pneumoconiosis in one man who worked in the processing area for only 7 years where the mean respirable dust level in this area from 1977 to 1980 ranged from 4 to 5 mg/m³. The TLV of 2 mg/m³ does not seem provide a large margin of safety below the suspected exposure level of 4 to 5 mg/m³ reported to have caused the mild pneumoconiosis observed in this single case. However, there are problems with inferring that the TLV is not protective because of this case. There were no exposure measurements prior to 1977 when the exposures could very well have been higher than 4 or 5 mg/m³. Another problem with using this case to assess the safety of the TLV is that it was not reported whether the man worked in any other dusty jobs either at this kaolin plant or another prior to working in the processing area for 7 years. Unfortunately there is no sound data to infer whether or not the TLV is protective of kaolin pneumoconiosis. Therefore, the TLV of 2 mg/m³ will be used to develop the ITSL for kaolin according to Rule 232(1)(c). When more

complete epidemiological studies are available the adequacy of the TLV for kaolin will be re-evaluated.

ITSL = occupational exposure limit ÷ 100

ITSL = 2 mg/m^3 ÷ 100

ITSL = 0.02 mg/m^3

ITSL = 20 µg/m^3 (based on an 8-hour averaging time)

The ITSL for kaolin is 20 µg/m^3 based on an 8-hour averaging time.

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