PLAINTIFF'S EXHIBIT

UC-5224

CABOT BERYLCO

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TO: T. J. Concannon

DATE: September 2, 1982

FROM: J. P. Butler

SUBJECT: Beryllium and Asbestos: Some Comparisons

cc: D. J. Richards/F. P. Giannakos

Manville Corp.'s choice of Chapter 11 as a buffer against asbestos litigation may stimulate questions about CBD's vulnerability to product liability suits for damage to health by beryllium. The two situations are different. In sum, asbestos has been the cause of more diseases and has placed more people at risk than has beryllium.

Damage from asbestos exposure may take five forms: asbestosis, a noncancerous lung disease; mesothelioma, a cancer of the pleura or peritoneum; and cancers of the lung, larynx, and gastrointestinal tract. Mesothelioma, principally if not exclusively caused by asbestos, has afflicted persons living near asbestos plants or with asbestos workers.

Speaking of asbestos, Dr. William Blot of the National Cancer Institute has said: "It appears to be the greatest single source of occupational cancer" (Newsweek, September 6, 1982). The same magazine reports that researchers at Mount Sinai School of Medicine, New York, predict at least 5000 cancer deaths annually from asbestos in the United States until the end of the century.

A case against beryllium as a producer of cancer in man has not been established. Both an independent review panel convened by the Center for Disease Control and the Second Annual Report on Carcinogens of the Public Health Service have found fault with studies purporting to show an excess of lung cancers in beryllium workers for failure to rule out the effects of factors other than beryllium. Dr. J. Donald Millar, Director of NIOSH, has said in an interview (Occupational Health and Safety, April, 1982) that the study in this group performed by his agency "included weaknesses which otherwise wouldn't have been there" if scientific objectivity had not given way to advocacy. In a paper, Avoidable Risks of Cancer (Journal of the National Cancer Institute, June, 1981), Sir Richard Doll and Richard Peto make specific reference to their decision to exclude beryllium from a list of established human carcinogens.

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Occupational exposure to beryllium is the predominant cause of chronic beryllium disease (berylliosis). Nonoccupational exposure, once considered a major hazard, is now a diminshing influence in disease propagagtion. Most nonoccupational patients were women who laundered contaminated work clothing of beryllium plant employees. A small number were nonemployee neighbors of beryllium plants. Clothing issue, plant laundries, and pollution control engineering have all but eliminated nonoccupational berylliosis. The isolated cases that have appeared in recent years are the results of exposure before adequate controls and were delayed in development by the latency which can defer onset.

Acute health effects from exposure to soluble beryllium compounds comprise lung, upper-respiratory, skin, and eye conditions. For a number of reasons, they exert no influence on product liability: they are readily prevented; if they occur, their duration is short and recovery is complete; the causative agents, soluble beryllium compounds, of limited industrial use at best, are used not at all by CBD.

According to OSHA estimates cited in the Second Annual Report on Carcinogens, the numbers of workers exposed, respectively, to beryllium and asbestos are 25,000 and 2,500,000. The report also states: "Asbestos is so widely used that the entire population is exposed to some degree."

The greatest consumption of beryllium is in stamping beryllium copper parts from strip, an activity productive of only insignificantly low exposures and with no history of berylliosis. More dangerous conditions, unless properly controlled, exist in primary producers' plants; in alloy foundries; in shops grinding, polishing, buffing or welding alloys; and in fabrication plants handling pure beryllium and beryllium oxide. These establishments are of small to moderate size and relatively few in number. Exposures are as a rule well-controlled.

For chronic beryllium disease to occur, a susceptible person must receive overexposure. Estimates of the susceptible portion of overexposed populations place it at less than five percent. This rate of attack is derived from the experience of groups of workers subjected to uncontrolled exposure in the early history of the beryllium industry. (By contrast, of a group of 2800 beryllium employees hired in a recent 20-year period for work in a controlled environment only two developed berylliosis, an attack rate of 0.07 percent. Each case was the result of documented accidental exposure far above acceptable limits.)

There is no built-in brake of limited susceptibility under conditions of high exposure to asbestos. In the monograph, Asbestos and Disease, Selikoff and Lee make this observation:

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"Where high levels of asbestos dust prevailed 10-20 years ago, a high incidence of asbestosis can be expected, even up to 100% of those exposed for more than 10 years, but more usually at the rate of 50% of those exposed for more than 20 years, with increasing frequency with longer employment."

CBD's best course under present conditions is sustained vigilance in preventing excessive exposures to beryllium and in warning users of the beryllium hazard.

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