

## EPIDEMIOLOGY OF BERYLLIUM DISEASE - 40 YEARS EXPERIENCE OF A MAJOR PRODUCER

O. P. PREUSS

*Brush Wellman Inc., Cleveland, Ohio, U.S.A.*

### ABSTRACT

A total of 189 cases of acute and 42 cases of chronic disease among employees and 23 chronic cases among non-employees were analysed. Beryllium related acute respiratory disease (chemical tracheitis, bronchitis and pneumonitis) showed a high incidence only between 1938 and 1948 when the need for environmental controls was not yet fully recognized. They were observed only among employees exposed to water soluble extraction intermediaries (beryllium sulfate, fluoride and chloride) and declined sharply with the first exposure control efforts in 1949. Subsequently, only a few, mostly mild cases occurred sporadically due to accidental breakdowns in control equipment or neglect of safe operating procedures, and none since 1968.

The incidence of chronic beryllium disease (interstitial granulomatous pneumonitis) also was high only as long as exposure controls did not exist (22 cases in 10 years). With institution of controls and an even much larger work force, only 20 cases were observed during the subsequent 28 years (1949 to 1977). Of these, nearly all occurred in employees hired when production and control technology were still in a developmental stage and there were difficulties in maintaining the recommended standard of two micrograms per cubic meter of air on a daily weighted average. Only two new cases were diagnosed among employees hired after 1960. Both were caused by accidentally excessive, in retrospect preventable, exposures.

All so called neighborhood or "air pollution" cases turned out to be contact cases. They occurred exclusively among non-employees living only one or two blocks away from the plant, with direct exposure to rather high concentrations of beryllium dusts and vapors, and/or continued contact with beryllium oxide dust carried home on the work clothes of family members or friends. No further cases were observed near modern production facilities having adequate exposure and exhaust control installations and obligatory work clothing exchange and laundry facilities on the plant premises.

These observations indicate that acute or chronic beryllium disease would occur with exposure to beryllium concentrations far in excess of the recommended control standard. With the exception of two due to accidentally high exposure levels, no new cases occurred in production facilities where the atmospheric concentrations could be kept at or below two micrograms per cubic meter of air.

The extremely low incidence (at the most 1%) of chronic beryllium disease even among highly exposed workers, together with long latent periods and evidence of altered lymphocyte and macrophage reactivity should serve also as further confirmation that immune biological mechanisms play a major role in the pathogenesis of chronic beryllium disease.

The applications of beryllium and its oxide, and especially its copper alloy, are today much more widespread and varied than generally known. Also, no epidemiological studies have been published on the effectiveness of the threshold

limit value of two micrograms of cubic meter of air, as recommended in 1948 by the United States Atomic Energy Commission.

Having experienced a high incidence of beryllium related health problems early in its production history, Brush began with the development of a very comprehensive preventive program as soon as these recommendations had been made. In a continual search for the most efficient containment technology and work practices, it aimed at reducing the worker's exposure to or below these levels. However, demands for increased production, triggered primarily by the space program, made its attainment often quite difficult.

Beryllium dermatitis and acute respiratory problems, such as chemical bronchitis and pneumonitis, were the easiest to prevent. Caused exclusively by

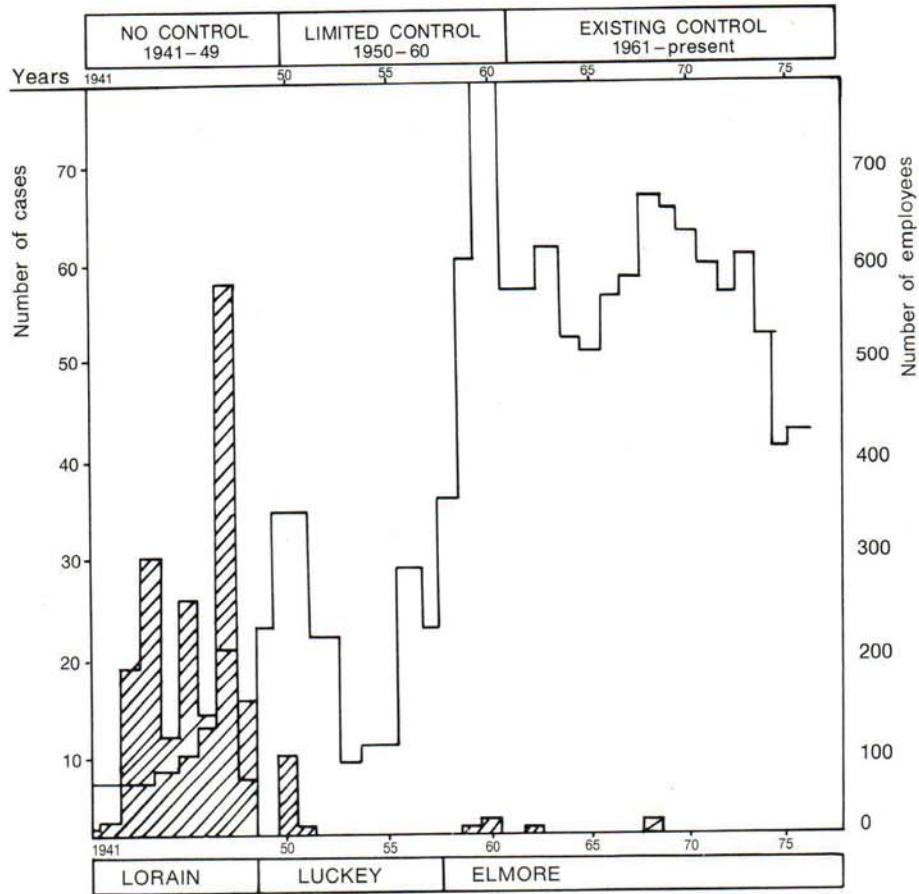


FIG. 1 - Crossed bars represent the number of acute respiratory cases (tracheitis, bronchitis and pneumonitis). Empty bars represent the number of employees.

water-soluble beryllium salts, they were virtually eliminated with the first major control effort, namely the construction of a new plant and installation of a closed, well ventilated extraction system in 1950 (Figure 1).

In contrast, cases of chronic interstitial pneumonitis continued to manifest themselves during the following two decades (between 1950 and 1970) with disturbing regularity (Figure 2). Being hard to trace back to the actual exposure sources, because of their long latent periods, these cases seemed to disprove both the validity of the recommended two microgram TLV and the efficacy of all ongoing control efforts.

Only after plotting the year of diagnosis against that of employment by plant location, did it become obvious that every diseased worker had been hired either during the earliest production phase (when beryllium disease and methods

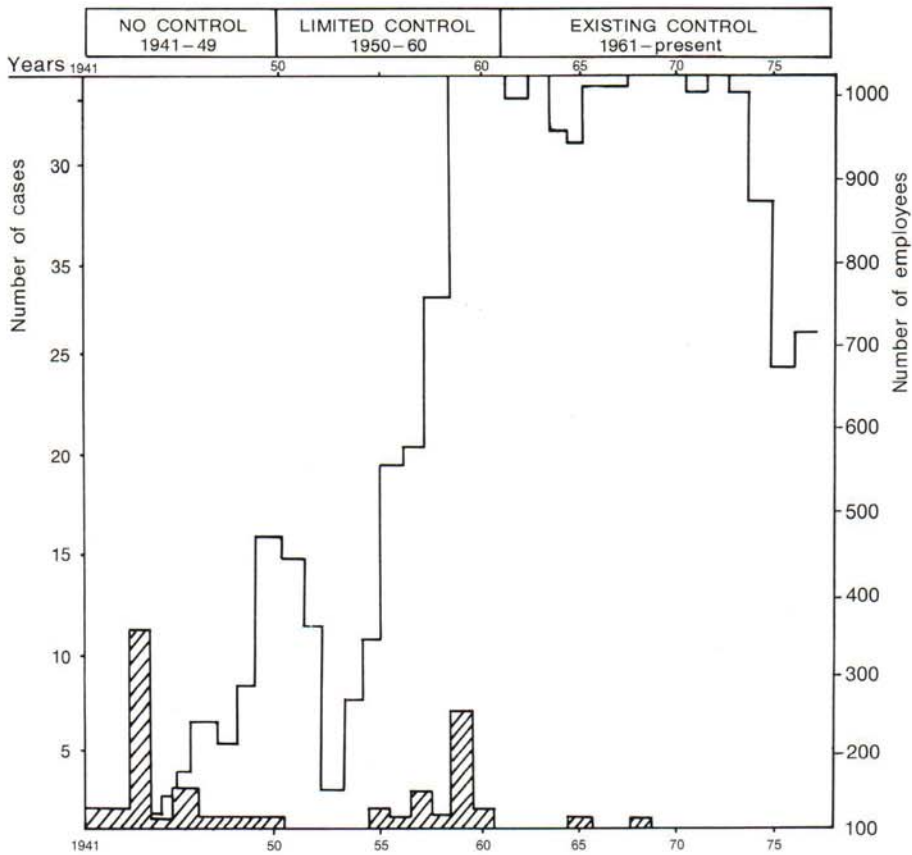


FIG. 2 - Crossed bars represent the number of chronic berylliosis in all plants. Empty bars represent the number of employees.

for its prevention were still unknown to most) or during later periods when technical problems interfered temporarily with adequate containment (Figure 3).

This method made it quite clear that all of the 42 employees with chronic beryllium disease had been exposed at one time to respirable beryllium concentrations exceeding at least 20 times the TLV. Furthermore, nearly all of these cases had experienced their critical exposures before 1960, during the

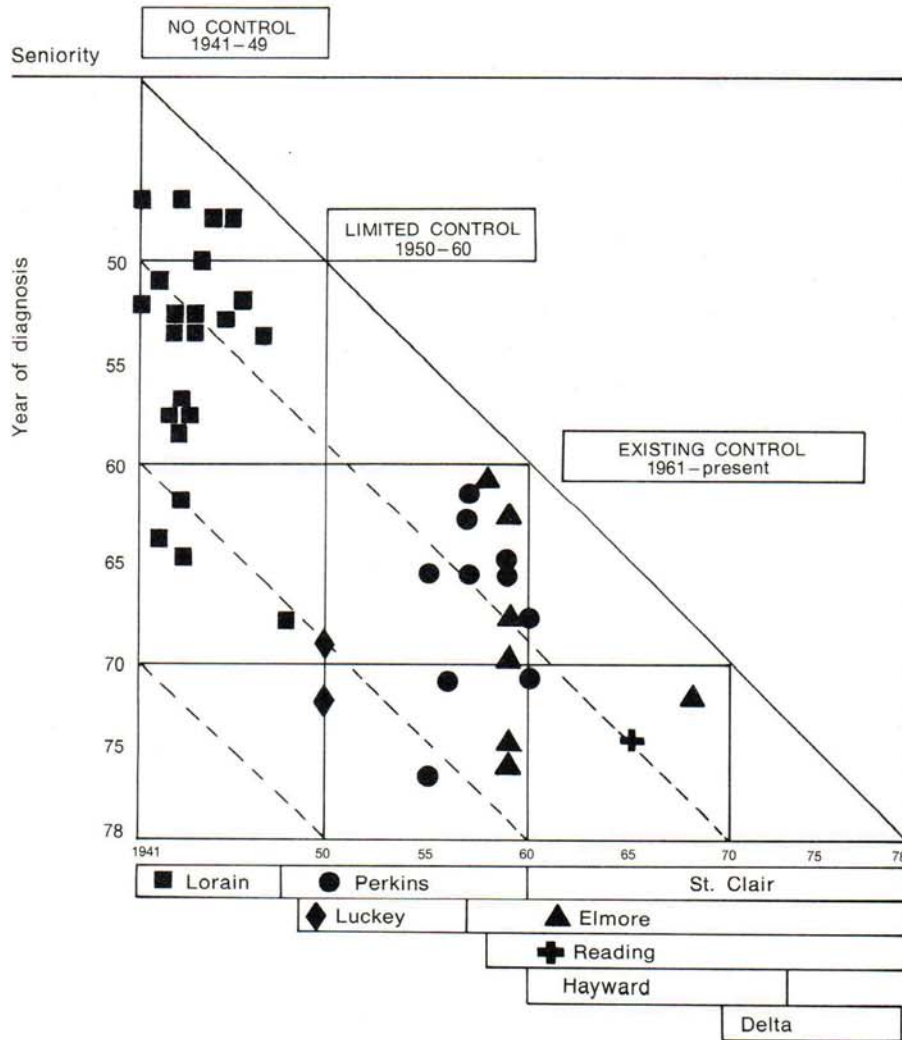


FIG. 3 - Chronic berylliosis among workers from individual plants. Dotted lines represent the latent period.

earliest production period, which was characterized by rapid changes in methods and production volume and only in its latter half by the development of a comprehensive containment program. The following 18 years however, showed clearly the fruits of these efforts. Of the many employees hired during that period, only two acquired chronic beryllium disease and both only because of truly accidental circumstances. So called "air pollution cases," observed during the initial two decades in the neighborhood of our first and rather inadequate plant, also ceased to occur as soon as operations were moved into modern, well controlled production facilities. Just as important for the prevention of such cases were the obligatory wearing, exchange and laundering of work uniforms on the plant site. The amount of beryllium oxide dust carried home regularly on the workers clothing had been large enough to expose relatives and friends to atmospheric concentrations far in excess of the two microgram threshold limit.

In summary, the data presented here support the following:

- a. The extremely low incidence of chronic beryllium disease, namely 42 cases out of 7 000 exposed within a 40 year period, and evidence of altered lymphocyte and macrophage reactivity in diseased individuals, confirm that immunobiological mechanisms play a major role in its pathogenesis.
- b. None of the workers continuously exposed to concentrations below two micrograms has ever acquired chronic beryllium disease.
- c. Only respirable dusts, fumes or vapors can produce the disease. However, the exposure has to be high (at least 20 times the TLV or significantly more short term) and must involve a predisposed and sensitized individual.
- d. Mere contact and handling of metallic beryllium, beryllium oxide and beryllium copper alloy, including stamping and slitting of the latter, will cause neither lung disease nor dermatitis.

In view of the well-known literature of the preceding two decades which concerns studies in animals, the question of the carcinogenicity of beryllium for man has been much discussed recently. However, our own empirical data do not show such evidence and contrary statements submitted at the August 1977 OSHA hearing have too many shortcomings to hold up under critical analysis.