**Hypersensitivity Pneumonitis (Extrinsic Allergic Alveolitis) and Other Granulomatous Lung Disease**

Epidemiological evidence on hypersensitivity pneumonitis (HP) is heterogeneous in regard to the occupational burden among all cases. The estimated occupational proportion of HP based on 15 reports published since 1983 are listed in Table 1 and range from 0% to 81.3% (1–15), whereas in many cases likely etiology identification failed, yielding 0% of occupational HP in case series. The weighted meta-proportion occupational HP prevalence was 19% (95% CI 12; 28) with high heterogeneity across studies (I2=97.6%). Only one study used a classic epidemiological approach yielding an OR from which a PAF could be calculated (20.2%) (2). One study was excluded from this data analysis (16) because its cases (n=199) were clearly subsumed in the cohort published by Selman (8). Other potential overlap among the series we did included cannot be excluded. Major limitation in the available data is that series that were exclusively derived from an occupational cohort or defined by a single risk factor (e.g., bird fanciers) could not be used to apportion etiology (17,18).

Published mortality profiles as an alternative approach to ascertain occupational burden in HP also showed that around 1/3 (38.4%) of all cases to have occupational origin (14), where farmer’s lung, bagassosis, suberosis malt worker’s lung, mushroom worker’s lung, maple-bark stripper’s lung we considered as occupational.

Inhalation of metal [aluminum, barium, beryllium, cobalt, copper, gold, rare earths (lanthanides), titanium, and zirconium] dust or fume can cause granulomatous lung disease that mimics sarcoidosis. Several studies of sarcoidosis patients have reported that disease prevalence is higher among workers in certain occupations, including firefighters, navy recruits, workers in the lumber industry, rock wool or glass wool workers, salespeople, and World Trade Center disaster emergency responders (19). A large case-control study of sarcoidosis patients screened not to have beryllium sensitization (ACCESS) found that workers with industrial organic dust exposures; retail workers who sold building materials, hardware, and gardening materials; and educators were at an increased risk compared to controls (20). Another ACCESS paper involving the same patient population reported that agricultural employment and occupational exposures to insecticides and microbial bioaerosols were associated with increased risk (21). A third study using the ACCESS occupational history questionnaire and a case-control design with African American siblings found that usual employment in metal machining and retail trade; ever working in metalworking and transportation services; and exposures to titanium, vegetable dust, and moldy environments were associated with risk of sarcoidosis (22). In a study of sarcoidosis prevalence in Switzerland, higher frequency was found in regions with metal industry and intense agriculture (23). In a large US study using national death certificate data, sarcoidosis mortality risk was significantly elevated in association with employment involving metal working, health care, teaching, sales, banking, and administration (19), and these results suggest occupational exposures increase risk for a more severe sarcoidosis phenotype (24). Estimated occupational prevalence of sarcoidosis ranged from 37% to 54% (19,20,22).

Epidemiological evidence on the proportion of chronic beryllium disease (CBD) that is misdiagnosed as sarcoidosis is limited to a few case series (25–29), as well as one case-referent study (30). In the case series the determination of CBD was based on pathology consistent with sarcoidosis plus beryllium sensitization based on a positive beryllium lymphocyte proliferation test. The case-referent study used the Glu-69 genetic variant as the marker of CBD in the context of physician-diagnosed sarcoidosis and a history of >10 years of occupational exposure to beryllium. Estimated occupational prevalence of CBD ranged from 0% to 69% (25–30).

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