

## Comment on Barbieri et al. Pleural plaques and lung asbestos burden. *Med Lav* 2019, 110: 353-362

I read with interest the paper of Barbieri et al on the relationship between pleural plaques (PP) and asbestos lung burden. The article confirms the results of a similar study which found the existence of a relationship between the extent of PP and pulmonary asbestos body concentration in 207 lung cancer patients (4). The Japanese authors considered the PP extent useful as a proxy for pulmonary asbestos body concentration. In this sense the study of Barbieri et al is of considerable utility by providing new data (including fibre concentration in lung tissue) on a very different working population. However attention should be focused on a couple of key issues.

As in the case of the Japanese authors, it is a necroscopy study on patients who died for asbestos-related diseases (likely mainly neoplastic diseases). It means the studied population was made up of heavily exposed workers. This is also confirmed by the heavy asbestos lung burden. Actually for malignant and non malignant pleural asbestos-related diseases it is commonly accepted that there is a dose-dependence at high intensity exposures, while this does not seem to occur with lower doses. Thus the relationship between PP and asbestos lung burden should be confirmed for less intense exposures currently much more frequent in the EU. In previous studies in less exposed populations we found an asbestos fiber concentration in bronchoalveolar lavage fluid significantly higher in patients with asbestosis than in subjects with only PP (2, 3).

Another issue is the use of total fiber concentration as a marker of asbestos lung burden. Unless to compare the lung burden data to the benchmarks set by the Helsinki Criteria,

having chrysotile and amphibole exposure a different meaning, it would be better to consider them separately. Particularly useful is the amphibole lung burden to assess the risk of developing asbestos-related diseases (1).

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## Response to the “Comment on Barbieri et al. Pleural plaques and lung asbestos burden Med Lav 19, 110: 353–362” by Sartorelli Pietro

Dear editor,

we thank Prof. Sartorelli for his interest in our recent work (1) and we are pleased to answer his letter, *Comment on Barbieri et al., Pleural plaques and lung asbestos burden* (5) regarding two critical aspects that, according to the author, we should have taken into consideration in our study.

First, Sartorelli comments that we examined “heavily exposed workers”. This is not so. The Helsinki Consensus Document (6, 7) suggested a cut-off of 1 million amphibole fibers (length  $>1 \mu\text{m}$ ) as measured by electron microscopy, to identify subjects with a high probability of (occupational) exposure to asbestos. In our study (Table 3), the concentration of amphibole fibers was  $<1$  million in 26 out of 124 (21.0%) subjects (1). Moreover, 53 subjects (42.7%) had concentrations between 1 and 10 million fibers, that we can confidently define as “moderate” based on decades of experience of one of us (PGB) and also taking into account that there were many subjects with much higher doses. Therefore, we believe that in our study we covered a wide range of doses/exposures, from low to high.

Second, Sartorelli suggested to consider chrysotile and amphibole fibers separately. We agree with him. However, as reported in our article (Table 1), the lung fiber burden was made up almost entirely of amphiboles. In particular, we reported that only in 10.5% of cases the concentration of amphiboles was  $<80\%$  (1). For this reason, we performed no separate analyses. Our findings were largely expected if we consider the much shorter biopersistence of chrysotile (2). For this reason, no reference values for chrysotile have been suggested in the Helsinki Consensus Document (6, 7).

Finally, we would like to note one imprecision in Sartorelli's letter. He stated that “for malignant and non malignant pleural asbestos-related diseases it is commonly accepted that there is a dose-dependence at high intensity exposures, while this does not seem to occur with lower doses”. We do

not concur with this statement. Systematic reviews of epidemiological studies that have explored the exposure-response relationship in pleural malignant mesothelioma have shown the presence of a positive association over the whole range of exposures, i.e., regardless of exposure/dose magnitude (3, 4). The results of our study (1) suggest that this holds also for non-neoplastic pleural diseases.

**Pietro Gino Barbieri**

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