ERJ open research

Biometric monitoring in silicosis to detect early disease and monitor lung injury: not quite there

To the Editor:

Any attempt to prevent the development of silicosis in exposed workers by identifying occult disease undetectable by conventional methodology, is a welcome and worthy cause. The recently published paper by Ophir *et al.* [1] addresses this issue by using biometric monitoring of functional (spirometry and diffusion capacity of the lung for carbon monoxide) and inflammatory parameters (percentage of neutrophils in a sample of induced sputum) in exposed workers. Using novel technology, they also quantitated the amount of silica in induced sputum in a cross-sectional study of 68 workers, 43 of whom already had silicosis, and compared their findings with those in 48 unexposed controls. These numbers are from the abstract of the paper; in the methods section in the body of the paper it is stated that 116 exposed individuals were screened, with no mention of the number of unexposed controls. 116 is the sum of 68 plus 48; subjects plus controls. We assume that this inaccuracy and the omission of the number of controls, are an oversight of the authors.

However the conclusion of the authors that their findings identify "populations at high risk of advanced inflammatory and functional deterioration as the result of continuing exposure to artificial stone dust" and their concluding statement that "the novel functional and inflammatory biomarkers in induced sputum samples can effectively detect and monitor lung injury" remains to be proved.

The study demonstrated the presence of inflammatory markers in exposed workers, the majority of whom already had silicosis. This negates the claim to "early identification of workers at high risk of developing advanced stages of silicosis"; one cannot claim to early identification in a worker who already has silicosis! Also, the higher percentage of neutrophils found in exposed workers may be due to the presence (and severity) of existing silicosis or related to smoking-related inflammation, and not necessarily represent "early" disease. No data regarding the type and severity of ongoing silicosis are provided. If indeed, the neutrophil burden, as termed by the authors, represents exposure-induced inflammation, there should be a relationship between the stage and severity of silicosis and the degree of inflammation. Whether inflammation of the nature found leads to permanent injury as the authors infer, needs to be shown prospectively. The assumption that this will occur is still only conjecture and has to be shown by a prospective cohort study and cannot be inferred from the cross-sectional study which was performed.

A cohort study of exposed *versus* non-exposed healthy workers and follow-up over time with serial tests to detect a temporal relationship between increased levels of inflammatory biomarkers and the risk of silicosis is needed to confirm the speculation that the biometric findings are truly harbingers of future disease. This is the main thrust of our critique which does not question the exciting novelty of the methodology described but addresses the clinical and epidemiological inferences of the conclusions.

Briefly, we would also like draw attention to other methodological shortcomings of this study. The authors did not stratify the results (including lung function testing) according to smoking history, an important cause of inflammatory reaction in the lung, (nor did they provide a definition of "past smoker"). Such stratification is crucial; smoking induces inflammation, the biomarker identified in the study and may be present in past smokers [2]. Furthermore, the study and control groups were significantly different in terms of average age, sex and weight. The degree of exposure to environmental pollutants and socioeconomic status are also important confounders that should have been addressed. Not considering these confounders considerably hampers the validity of the study. Finally, the study makes a point of various metals found in the dust which was analysed and considered to represent the "raw material". A sample collected from the floor of a shop is not truly the raw material since Caesarstone contains neither zirconium nor aluminium.

Daniel Weiler Ravell[1,2] and Hashem Bishara[3]
[1]Faculty of Medicine, Technion, Haifa, Israel. [2]Caesarstone Ltd, Haifa, Israel. [3]Faculty of Medicine, Bar Ilan University, Technion, Zefat, Israel.

ERS EUROPEAN RESPIRATORY SOCIETY every breath counts

Correspondence: Daniel Weiler Ravell, Migdan Lung Clinic, 29 Tshernichovsky, Haifa, 3570134, Israel.
E-mail: weiler@actcom.co.il

Received: July 31 2016 | Accepted: Aug 05 2016

Conflict of interest: Disclosures can be found alongside this article at openres.ersjournals.com

@ERSpublications
Biometric monitoring in exposure to silica dust http://ow.ly/qRrB303lMDg

References

1 Ophir N, Bar Shai A, Alkalay Y, *et al.* Artificial stone dust-induced functional and inflammatory abnormalities in exposed workers monitored quantitatively by biometrics. *ERJ Open Res* 2016; 2: 00086-2015.
2 Rutgers SR, Postma DS, ten Hacken NH, *et al.* Ongoing airway inflammation in patients with COPD who do not currently smoke. *Thorax* 2000; 55: 12–18.