Chronic effects of occupational exposure to mineral fibres and recurrent chest infections in insulators

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To the Editor:

Mineral fibres, which are types of commercially produced man-made vitreous fibres (MMVFs) with a woolly consistency, are industrially manufactured by passing air through molten glass, rock or slag, and are commonly known as glass, slag or rock wool. Although all these materials are grouped together as mineral fibres, there are differences in their composition and physical properties. Glass wool/fibreglass is made from borosilicate glass, which is composed of sand, soda ash dolomite, limestone, ulexite and anhydrite, while rock and slag wools are produced from naturally occurring igneous rock (basalt or dolomite rocks) and molten furnace slag, respectively [1]. These disorganised, interlocking fibres were reported to deliver potentially deleterious health effects, particularly on the skin and upper respiratory tract, to workers occupationally exposed to these materials, such as during installation or removal of insulation [2, 3]. While a few studies reported no evidence of pneumoconiosis in the industrial workers exposed to glass, rock or slag wool [4], several case reports appeared in recent years describing a long biopersistence of MMVFs leading to the development of pulmonary fibrosis at a later stage [5–8]. However, evidence of mineral fibre-associated respiratory tract infections has been limited. In this study, we aimed to investigate whether occupational exposure to mineral fibres was associated with recurrent chest infections.

We used baseline and follow-up data in this longitudinal study of unionised insulators that were enrolled in the Wellness of Workers programme in Alberta (Canada) between 2011 and 2017. Details of the study population and methodologies were reported previously [9]. Briefly, out of 990 workers who were recruited at the baseline visit, we removed participants who had chest infections at baseline (n=457), were diagnosed with asthma or COPD (n=206), or were lost to follow-up (n=55). We thus recruited 272 insulators for this current analysis. The demographic profile, smoking history (ever *versus* never smokers, and smoking pack-years), exposure to second-hand smoke (SHS) in childhood (yes/no), and family history of lung disease in either or both the parents (yes/no) were recorded using a structured questionnaire as described elsewhere [9]. Occupational exposure to mineral fibres was self-reported (yes/no) and was recorded at the baseline visit. Self-reported chest infection, as defined previously [9], was recorded at the follow-up visit, and episodes of chest infections in the past 3 years were considered as the primary outcome. In a subsample, we also collected venous blood samples at the baseline and follow-up visits, and complete blood cell counts were performed.

We created univariable and multivariable Poisson regression models (for count outcome variable) to examine the association between baseline mineral fibre exposure and episodes of recurrent chest infections at follow-up. Multivariable models were adjusted for baseline age, sex, ethnicity, body mass index, marital status, education, smoking pack-years, the time between baseline and follow-up visit, and use of personal protective equipment (PPE). We checked for multicollinearity among the confounding variables using variance inflation factor. The goodness of fit of the models was tested by Akaike information criterion. We also performed some secondary analyses. We tested the potential effect modification of smoking habits (active *versus* former/never smoker), exposure to SHS in childhood, and parental lung disease on the association between mineral fibre exposure and chest infections. In a subgroup (n=157), we also tested the mediation effect of lymphocyte/monocyte ratio (LMR) and neutrophil/lymphocyte ratio (NLR) on the association between mineral fibre exposure and chest infections. All analyses were performed as a complete case approach in STATA v.17 (StataCorp, College Station, TX, USA).

Shareable abstract (@ERSpublications)
Exposure to mineral fibres (man-made forms of vitreous fibres often used as insulating material) is a risk factor for recurrent chest infections among workers, underscoring the necessity of workplace surveillance for protection from hazardous substances https://bit.ly/38cUpmA

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This study was approved by the Health Research Ethics Board of Alberta (HREBA.CTC-17-0067) and Health Research Ethics Board (Pro00079792), University of Alberta, and was conducted according to the Declaration of Helsinki. All participants provided signed informed consent forms before taking part in the study.

Descriptive statistics of the study participants are presented in table 1. Most participants were male (91%) with a mean±SD age of 46.9±13.6 years. 64% were active smokers at baseline, with a median (interquartile range (IQR)) of 3 (0–16.4) pack-years. Participants were followed up at a median (range) interval of 3.8 (3–8) years from their baseline visit. In the follow-up visit, 95% of the participants reported occupational exposure to mineral fibres at baseline and reported a median (IQR) of 2 (0–2) episodes of chest infections that occurred between the baseline and the follow-up visits.

In the univariable analysis, we found that being exposed to mineral fibres was associated with increased episodes of chest infections (regression coefficient (β) 1.56, 95% CI 0.16–2.95). After adjusting for confounders in the multivariable model (except for PPE), the association remained consistent, although the magnitude of the estimate was increased marginally (β 1.95, 95% CI 0.55–3.35). However, adding the use of PPE in the model did not alter the magnitude of the estimate significantly (β 1.84, 95% CI 0.41–3.26). We did not observe any effect modification by smoking, exposure to SHS in childhood, or parental lung disease on the association between mineral fibre exposure and recurrent chest infections. In a subgroup analysis, we did not observe any mediating effects of LMR or NLR on the association between mineral fibre exposure and recurrent chest infections.

Our study demonstrates that mineral fibre exposure can lead to recurrent chest infections in a group of insulators. We did not observe any potential involvement of active or passive smoke exposure, family

TABLE 1 Demographic, exposure and clinical characteristics of study participants

Participants	272	
Demographics		
Age, years[#]	46.9±13.6	
Male	248 (91)	
BMI, $kg \cdot m^{-2}$[#]	29.6±6.0	
Ever smokers	173 (64)	
Smoking pack-years	3 (0–16.4)	
Caucasian ethnicity	219 (81)	
Educational qualifications		
Grade school	8 (3)	
High school	36 (13)	
Trade school	128 (47)	
College/university	100 (37)	
Marital status		
Single/unmarried	82 (30)	
Married/with a partner	172 (63)	
Divorced/separated/widowed	18 (7)	
Exposure history		
Childhood smoke exposure	182 (67)	
Parental lung condition	74 (27)	
Exposure to mineral fibres[#]	259 (95)	
Use of PPE for mineral fibres[#]	161 (59)	
Clinical profile		
Episodes of chest infection in the past 3 years[¶,+]	2 (1–2)	
White blood cell profile	**Baseline**	**Follow-up[+]**
Neutrophil %	58±10	59±10
Eosinophil %	3±2	3±2
Lymphocyte %	30±8	30±9
Monocyte %	8±2	8±2
Lymphocyte/monocyte ratio[§]	3.84±1.31	3.76±1.45
Neutrophil/lymphocyte ratio[§]	2.26±1.45	2.31±1.19

Data are presented as mean±SD or median (interquartile range) for numerical variables, and n (%) for categorical variables. BMI: body mass index; PPE: personal protective equipment. [#]: data taken at the baseline visit; [¶]: data taken at the follow-up visit; [+]: median interval between baseline and follow-up visit was 3.8 years; [§]: ratio of absolute count.

history of lung diseases, or immune cell modulators (LMR or NLR) on this association. Our findings of mineral fibre exposure-associated chest infections are supported by the only available report of increased risk of infectious pneumonia due to exposure to inorganic dust containing mineral fibres [10]. A case of pneumonia with anthracofibrosis was reported in which the investigators found that the patient had a history of mineral fibre exposure [11]. While previous studies have demonstrated LMR and NLR as biomarkers for influenza and pneumonia-like infections [12–15], in our subgroup analysis we did not observe any mediating role of LMR or NLR on the pathway between mineral fibre exposure and recurrent chest infection. This presumably indicates that mineral fibre exposure might trigger infections other than pneumonia or influenza, although the detailed pathophysiology of such infections is not yet fully understood [11]. One possible hypothesis of such aggravated risk of chest infections is that MMVFs can damage alveolar macrophages [16], thus reducing the lung's capacity to counter pathogens. Lastly, our study also emphasises the use of proper PPE for controlling exposure. As we mentioned previously [9], PPE should be quality-checked for different exposures, as PPE is product-specific, and not all PPE provides uniform protection from different classes of materials.

One of the strengths of this study lies in the longitudinal design of the study, which suggests an association between mineral fibre exposure and the event of concomitant chest infections for the first time. Moreover, we investigated other factors (potential effect modifiers and mediators) that could influence the association between mineral fibre exposure and chest infections. However, the study has some limitations. First, the exposure was self-reported, and we could not determine individual exposure to mineral fibres and were also unable to calculate the cumulative exposure index, as participants were unable to provide exact information about their exposure history (years of said exposure). Secondly, chest infections were self-reported and were not clinically examined. Due to the longitudinal nature of the study design, we could not determine the exact time of onset of such infections and could only obtain reports of chest infections at the time of follow-up. Therefore, the possibility of over- or under-reporting could not be eliminated. Thirdly, blood cell profiles were not available from all participants, and we did not have information about other immunological markers or bacterial/viral infections in participants. Moreover, we could not determine any possible deviation in the LMR and NLR values of our study population, due to the lack of standard reference ranges of those variables in healthy adult populations. Lastly, the plausible involvement of other factors, such as exposure to physical, chemical or biological agents in the workplace or residence, could not be estimated, and thus more robust studies are required in the future.

In summary, we may conclude that our study provides a potential link between occupational exposure to mineral fibres and recurrent chest infections in insulators. This underscores the deleterious yet undetermined health effects of such insulating materials and warrants proper and safe handling of insulating materials. Moreover, continuous surveillance of workplaces as well as personnel health is necessary to minimise the risk of exposure to such hazardous substances, as well as facilitate compensation and medical assistance in case of adverse events due to exposure.

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Data sharing policy: The datasets used and/or analysed during the current study contain sensitive personal data and cannot be made publicly available. However, a de-identified dataset with limited variables is available from the corresponding author on reasonable request.

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Author contributions: S. Moitra carried out the formal analysis, validated and interpreted the results, and drafted the original version of the manuscript. A. Farshchi Tabrizi was involved in data curation and revised the manuscript. L. Henderson was involved in project administration and revised the manuscript. F. Khadour was involved in the clinical investigation, project administration, resource acquisition and reviewing the draft. M. Osman provided intellectual content and reviewed the draft. L. Melenka conceptualised and administered the project, acquired funding, and was involved in clinical investigation and revision of the draft. P. Lacy was involved in project administration and resource acquisition, supervised the project and edited the draft.

Conflict of interest: S. Moitra reports personal fees from Synergy Respiratory and Cardiac Care (Canada) related to this work; and from Apollo Gleneagles Hospital (Kolkata, India) and Permanyer Inc. (Spain) outside this work. P. Lacey reports grants from the Wellness of Workers programme, Local 110 Heat and Frost Insulators and Allied Workers, and Synergy Respiratory Care Limited (Canada) that supported this work; and personal fees from AstraZeneca (Canada) and GlaxoSmithKline (Canada) outside this work. The other authors do not have any conflict of interest to declare with this work.

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