The role of cigarette smoking-derived pollutants in the risk of mortality in idiopathic pulmonary fibrosis

Received: 15 May 2021
Accepted: 28 May 2021

To the Editor:

We read with great interest the work reported by Yoon *et al*. [1]. The authors found that a high concentration of nitrogen dioxide at an individual's residential address is significantly associated with increased mortality in idiopathic pulmonary fibrosis (IPF) patients when all confounders are included in the health analysis model. However, an important question that remains unclear in the study is whether active or passive cigarette smoking-associated release of nitrogen dioxide or particulate matter plays a role in the clinical outcome of IPF patients.

Consistent evidence reported in previous studies showing that smokers are at high risk of developing IPF and/or acute exacerbation of the disease underscores the relevance of smoking as a confounding factor in the clinical outcome of IPF patients [1–3]. Also, based on the literature, there is no doubt that tobacco smoke is a source and an important contributor to increased indoor levels of nitrogen dioxide and particulate matter [4].

In the study, the authors stratified their patients by age and sex for subgroup analysis and found a similar significant association between nitrogen dioxide and IPF mortality. However, subgroup analysis by the smoking history of the patients was not reported. Tobacco smoke exposure was included only as a confounder in the health analysis model (models 3 and 4) [1]. The effect of tobacco smoke in this study is of particular interest because of the person-focused research design used to measure the environmental pollutants. Theoretically, the person-centred approach would increase the power of the model to deny or confirm the influence on the outcome of a confounding factor closely associated with the patient's lifestyle.

In addition to active cigarette smoking history, the model would become even more powerful if the history of smoking by family members or neighbours is also included. Among all patients (n=1114) included in the study, the number of ever-smokers (n=846) was significantly higher than that of smokers. However, the population of patients with a history of smoking (n=268) was sufficiently large to negate or confirm the involvement of smoking-derived pollutants in the clinical outcome of IPF.

Thus, we believe it would be appropriate and valuable to perform and provide the results of subgroup analysis by tobacco exposure. This information would help to design future epidemiological studies and therapeutic strategies to predict and prevent the effect of smoking-derived pollutants on mortality in IPF patients.

Shareable abstract (@ERSpublications)
The role of pollutants derived from cigarette smoke in the mortality of patients with pulmonary fibrosis https://bit.ly/3pbuPDc

Cite this article as: Yasuma T, D'Alessandro-Gabazza CN, Hataji O, *et al*. The role of cigarette smoking-derived pollutants in the risk of mortality in idiopathic pulmonary fibrosis. *Eur Respir J* 2021; 58: 2101372 [DOI: 10.1183/13993003.01372-2021].

Taro Yasuma[1], Corina N. D'Alessandro-Gabazza[1], Osamu Hataji[2], Tetsu Kobayashi[3] and Esteban C. Gabazza[1]

[1]Dept of Immunology, Mie University Faculty and Graduate School of Medicine, and Mie University Hospital, Tsu, Japan. [2]Respiratory Center, Matsusaka Municipal Hospital, Matsusaka, Japan. [3]Dept of Pulmonary and Critical Care Medicine, Mie University Faculty and Graduate School of Medicine, and Mie University Hospital, Tsu, Japan.

Corresponding author: Esteban C. Gabazza (gabazza@doc.medic.mie-u.ac.jp)

Author contributions: Approval and writing the draft of the correspondence: T. Yasuma, C.N. D'Alessandro-Gabazza, O. Hataji, T. Kobayashi and E.C. Gabazza.

Conflict of interest: T. Yasuma reports grants from Shionogi, outside the submitted work. C.N. D'Alessandro-Gabazza reports grants from Shionogi Pharmaceutical Inc., Astellas Pharmaceuticals Inc. and Asahi Kassei, outside the submitted work. O. Hataji has nothing to disclose. T. Kobayashi reports grants and personal fees from Chugai, Pfizer, ONO, Boehringer Ingelheim and Eli Lilly, grants from TAIHO, outside the submitted work. E.C. Gabazza reports grants from Shionogi Pharmaceutical Inc., Astellas Pharmaceuticals Inc. and Asahi Kassei, outside the submitted work.

References

1 Yoon H-Y, Kim S-Y, Kim O-J, *et al.* Nitrogen dioxide increases the risk of mortality in idiopathic pulmonary fibrosis. *Eur Respir J* 2021; 57: 2001877.
2 Sack C, Raghu G. Idiopathic pulmonary fibrosis: unmasking cryptogenic environmental factors. *Eur Respir J* 2019; 53: 1801699.
3 Steele MP, Speer MC, Loyd JE, *et al.* Clinical and pathologic features of familial interstitial pneumonia. *Am J Respir Crit Care Med* 2005: 172: 1146–1152.
4 Vardoulakis S, Giagloglou E, Steinle S, *et al.* Indoor exposure to selected air pollutants in the home environment: a systematic review. *Int J Environ Res Public Health* 2020; 17: 8972.