Editorial

Highlights from the European Respiratory Society 2018 Annual Congress: environment and epidemiology (assembly 6)

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Lung function-associated genetic variants: can they predict chronic obstructive pulmonary disease (COPD)?

Measures of adult lung function, which are used in the assessment of COPD, have been associated with several dozens of genetic variants. Wain *et al.* using an unweighted risk score based on 95 variants have previously shown that these variants predict COPD (1). This time after identifying over a hundred new variants from data on 400,000 adults from UK Biobank and SpiroMeta, they built a weighted risk score to predict COPD in about 6,000 cases and found that this score improved disease prediction (2). What is the clinical relevance of these findings? What do these variants tell us about the underlying mechanisms of COPD? Hopefully the future will bring answers to these questions.

Children's lung function and… the exposome: what else?

It is well accepted that early life exposures to tobacco smoke and poverty have a role in determining lung function throughout the life course (3,4). However, there is relatively little research done on other environmental factors acting early in life. Siroux *et al.* used data from 1,033 mother-child pairs from the Human Early-Life Exposome consortium to agnostically assess the effect of over 400 early life exposures on the FEV1 percent predicted of 8-year olds (5). In this environment-wide association study, which did not produce any statistically significant finding, poverty, phthalates, per- and polyfluoroalkyl substances, parabens and traffic-related exposures came out as potential early life factors affecting lung function in children. These results should not discourage us from carrying out such studies, especially in larger samples.

Do changes in BMI throughout life affect lung function?

BMI has been associated with lung function (6,7). However, little is known about the impact that changes in BMI over time may have on lung function. Peralta *et al.* (8) assessed the association of BMI trajectories in more than 3,500 adults from the ECRHS that were followed up for about 20 years. The main findings of this analysis suggest that people who are and remain overweight or obese throughout adult life may experience greater lung function decline compared to those with normal weight.

To have a better picture of the impact of BMI trajectories on lung function it would be interesting to know whether these findings hold true in a birth cohort expanding the age range from early childhood to late adulthood.

Young adults with airflow obstruction: what to expect?

Lung function is a good predictor of survival, even among people with no respiratory symptoms (9,10). Çolak *et al.* (11) assessed whether young and middle-aged adults with airflow obstruction from the Copenhagen General Population Study are more likely to develop cardiorespiratory disease and die earlier than those without airflow obstruction. Not surprisingly, they found that obstruction increased the risk of cardiorespiratory morbidity and early death. However, this was true only when obstruction was defined using the

lower limit of normal (FEV1/FVC < LLN) and not the fixed ratio (FEV1/FVC <0.70). Should clinicians keep using the fixed ratio?

Are animal research facilities doing enough to prevent laboratory animal allergy (LAA)?

Thousands of people work with laboratory animals worldwide, and about a third of them may be affected by LAA (12). With mice replacing rats as the main animal model for diseases and with improvements made to the cages housing these animals, an updated Code of Best Practice for animal research seems essential. To support a new code in the UK, Feary *et al.* assessed the prevalence of LAA in a survey of 750 laboratory animal workers taking part in the SPIRAL study and measured their exposure to a mouse urinary protein linked to allergy (13). They found that the prevalence of LAA was larger among workers using conventional cages than workers using individually ventilated cages, and that exposure to the mouse urinary protein was partly explained by modifiable factors. Are new guidelines really needed or workers just need to adhere to existing ones?

Does the nasal proteome change after inhalation of occupational agents?

The proteome is the set of proteins encoded by the genome and expressed at a given time as response to certain conditions and exposures (14). Whether the nasal proteome changes under exposure to several occupational agents is what Suojalehto *et al.* tried to find out (15). They collected nasal brush samples from 24 non-smokers with work-related asthma before and after inhalation to protein allergens, isocyanates and welding fumes. The samples were analysed using electrophoresis and mass spectrometry. Suojalehto *et al.* did not find major changes in the nasal proteome, but they found that it changed differently depending on the exposure. What does this mean? What can we learn from these findings in terms of the biological mechanism of work-related asthma?

Quitting smoking?—let the money talk

What is the best way to convince working smokers to quit smoking? van Schayck *et al.* carried out a randomised trial of smoking cessation with 640 employees from 61 Dutch companies (16). They tested the use of financial incentives on top of regular smoking cessation group training sessions against just the training sessions and used CO-validated continuous abstinence at 6 months as their main outcome. van Schayck *et al.* found that even small amounts of money can make smokers quit and have a lasting effect on their abstinence. However, it is unclear for how long the financial incentives should be paid so that people do not go back to smoking.

Smoking status and aging

The most common way to know whether someone smokes or not, how much they smoke and for how long they have been smoking or smoked is to ask them. Recent exposure to tobacco smoke can also be assessed by measuring cotinine in biological samples. Clinicians not always ask their clients whether they smoke or not and rarely measure their cotinine levels. With this in mind, Skjodt *et al.* used biochemistry records from routine blood tests of 149,000 people to identify smokers (17). They used deep neural network analysis to run their models and found that red blood cell and serum lipid parameters were enough to predict smoking status with good accuracy. The approach taken by Skjodt *et al.* may well be used to fill the holes in electronic health record databases.

Using similar statistical methods and the same study population, Skjodt *et al.* assessed the association of biological aging with smoking status (18), showing that smokers aged faster regardless of fasting blood glucose and serum cholesterol levels. Their results are not novel, but they show how deep neural network analysis of routine records is a powerful tool.

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None.

Footnote

Conflicts of Interest: AF Amaral is one of the authors of abstract "*Body mass index trajectories during adult life and lung function decline*" (Peralta *et al.*).

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