Editorial

Highlights from the European Respiratory Society 2017 annual congress: epidemiology and environment (assembly 6)

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Submitted Aug 17, 2017. Accepted for publication Sep 29, 2017.
doi: 10.21037/jtd.2017.11.33
View this article at: http://dx.doi.org/10.21037/jtd.2017.11.33

How do smokers quit?

Vardavas *et al.* provided information on trends in and patterns of smoking cessation methods used in the EU between 2012 and 2014 (1). The authors analysed data from the 'Special Eurobarometer for Tobacco survey'. The survey data allows analyses which focus on changes in use of smoking cessation methods. The authors evaluated whether smoking cessation methods differed by socio-demographic characteristics. Several methods to quit smoking were applied less frequently over time: quitting without assistance; use of nicotine replacement therapy; healthcare professionals; and smoking cessation clinics. However, experimentation with e-cigarettes for the purpose of quitting increased, especially among younger people and those with a lower income. Interestingly, these changes were not consistent across EU member states.

As a conclusion, the authors suggest that a common evidence-based strategy to smoking cessation should be implemented across EU. Will this ever happen?

Smoke-free laws: have they reduced second-hand smoking?

Today, indoor air in public and work spaces is cleaner and life satisfaction is higher (2)—thanks to the European smoking ban! Most of the EU countries have implemented smoke-free environments to reduce exposure to second-hand smoke (SHS). However, it is not clear whether the smoking ban led to a reduction in SHS exposure. Filippidis *et al.* compared SHS exposure in 27,788 people, aged 15 years or more, in 2009 to 27,801 people, of the same age, in 2014 in 28 EU countries.

They found a significant 20% reduction in exposure to SHS in public places such as bars and restaurants, over the five-year period (3). However, the proportion of individuals exposed to SHS in the workplace increased by about 4% over the same period. The findings of this study reveal that many people in the EU continue to be exposed to SHS, and that the enforcement of smoke-free laws in the workplace is not completely adequate.

Can an airway challenge test predict chronic obstructive pulmonary disease (COPD)?

Measured with a challenge test and defined by a response beyond normal of the airways to nonspecific constrictor stimuli (4), airway hyper-responsiveness is a characteristic feature of asthma that is common among COPD patients too (5). Whether this feature precedes or is a consequence of COPD is unclear. Marcon *et al.* (6) explored this question by assessing the association of airway hyper-responsiveness with incident COPD in over 3,000 young and middle-aged adults from the multi-centre population-based European Community Respiratory Health Survey 2 (ECRHS2) (4,7), who were followed up for about 10 years (ECRHS3). The challenge test was carried out using methacholine, and airway responsiveness was treated either as categorical or continuous variable. COPD was defined using the ratio of forced expiratory volume-one second (FEV1) to forced vital capacity (FVC) either as binomial or continuous variable. They found that participants with airway hyper-responsiveness were more than twice more likely to develop COPD than those without airway hyper-responsiveness. This association was

evident also in a dose-response manner.

Despite some loss to follow up and changes in the spirometry protocol [i.e., pre-bronchodilator at ECRHS2 *vs.* post-bronchodilator at ECRHS3 (8)], the findings of this study suggest that: (I) airway hyper-responsiveness precedes COPD; and (II) an airway challenge test could be used to identify subjects at risk of COPD. Nevertheless, replication of these findings in independent cohorts is warranted.

Is there a role for DNA methylation in the association of airflow obstruction with occupational exposures?

Occupational exposures may account for 15–20% of the burden of COPD in the general population (9), and the mechanism underlying this association is thought to involve the release of free radicals leading to inflammation in the airways (10). van der Plaat *et al.* assessed whether the effect of occupational exposures (i.e., biological dust, mineral dust, gases/fumes) on airflow obstruction is mediated by DNA methylation (11). Instead of focusing their analysis on oxidative stress and inflammation-related genes, they carried out an epigenome-wide study (with Illumina's Infinium HumanMethylation450 BeadChip) on almost 1,600 participants from the Dutch LifeLines population-based cohort (12), who had pre-bronchodilator spirometry readings. They used bootstrapping to test the amount of mediation attributed to DNA methylation. None of the over 420,000 single CpGs (cytosine-phosphate-guanine sites) associated with any of the exposures at the genome-wide significance level. However, the exposures did associate with several differential methylated regions, some of which related to immune response.

The findings of this study suggest that the association of airflow obstruction with occupational exposure to dusts, gases and fumes is mediated by DNA methylation. What does this mean? Replication and further research is needed in this field.

Parabens, oral microbiome and lung function. What is this all about?

Epidemiological evidence suggests that airways obstructive disease may be somehow affected by poor oral health (13,14). One way of preventing the latter is to frequently brush the teeth and use an antibacterial mouthwash, which may or may not contain parabens. These chemicals have biocidal properties and for this characteristic they are widely used in cosmetics and personal hygiene products (e.g., toothpaste and mouthwash). To understand the relationships between the oral microbiome (i.e., genomes of microorganisms present inside the oral cavity), exposure to parabens, and lung function, Bertelsen *et al.* (15) analysed data from almost 300 Norwegian young adults, who took part in the RHINESSA study and had provided lung function measurements and samples of gingival fluid (for sequencing of the oral microbiome) and urine (for the levels of exposure to parabens). Consistent with other studies (16), parabens were found in more than 94% of the participants, and women showed higher concentrations than men. Bertelsen *et al.* showed that the different levels of urinary parabens correlated with the abundance of bacteria of two genera (*Enterobacter* and *Fretibacterium*). They found that the greater the microbiome diversity, the better the lung function of women, but not of men. Could this be a sex difference or just the result of the small study sample and insufficient control for confounders?

The authors of this study suggest that exposure to antibacterial chemicals, such as parabens, may modify the association between oral microbiome and lung function. However, how the associations 'parabens-bacteria' and 'bacteria-lung function' interlock with each other is not clear yet. Replication in a larger sample, with prospectively collected data, and further research will surely bring valuable information on this topic.

Acknowledgements

None.

Footnote

Conflicts of Interest: The authors have no conflicts of interest to declare.

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Cite this article as: Gagnat AA, Amaral AF. Highlights from the European Respiratory Society 2017 annual congress: epidemiology and environment (assembly 6). J Thorac Dis 2017;9(Suppl 16):S1554-S1556. doi: 10.21037/jtd.2017.11.33

