Environmental Research 153 (2017) 126–134

Contents lists available at ScienceDirect

Environmental Research

journal homepage: www.elsevier.com/locate/envres

Occupational airborne exposure in relation to Chronic Obstructive Pulmonary Disease (COPD) and lung function in individuals without childhood wheezing illness: A 50-year cohort study

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ARTICLE INFO

Keywords:
COPD
Lung function
Occupational exposure
Airborne pollutants
Job exposure matrix

ABSTRACT

Background: Evidence from longitudinal population-based studies relating occupational exposure to the full range of different forms of airborne pollutants and lung function and airway obstruction is limited.
Objective: To relate self-reported COPD and lung function impairment to occupational exposure to different forms of airborne chemical pollutants in individuals who did not have childhood wheeze.
Methods: A prospective cohort study was randomly selected in 1964 at age 10–15 years and followed up in 1989, 1995, 2001 and 2014 (aged 58–64) by spirometry and respiratory questionnaire. Occupational histories were recorded in 2014 and occupational exposures assigned using an airborne chemical job exposure matrix. The risk of COPD and lung function impairment was analyzed in subjects, who did not have childhood wheeze, using logistic and linear regression and linear mixed effects models.
Results: 237 subjects without childhood wheeze (mean age 60.6 years, 47% male) were analyzed. There was no association between any respiratory outcomes and exposure to gases, fibers, mists or mineral dusts and no consistent associations with exposure to fumes. Reduced FEV_1 was associated with longer duration (years) of exposure to any of the six main pollutant forms - vapors, gases, dusts, fumes, fibers and mists (VGDFFiM) with evidence of a dose-response relationship (p-trend=0.004). Exposure to biological dusts was associated with self-reported COPD and FEV_1 < Lower Limit of Normal (LLN) (adjusted odds ratio [95%CI] 4.59 [1.15,18.32] and 3.54 [1.21,10.35] respectively), and reduced $FEF_{25-75\%}$ (adjusted regression coefficients [95% CIs] −9.11 [−17.38, −0.84] respectively). Exposure to vapors was associated with self-reported COPD and FEV_1 < LLN (adjOR 6.46 [1.18,35.37] and 4.82 [1.32,17.63]). Longitudinal analysis demonstrated reduced FEV_1 and $FEF_{25-75\%}$ associated with exposure to biological dusts or vapors.
Conclusions: People with no history of childhood wheezing who have been occupationally exposed to biological dusts or vapors or had longer duration of lifetime exposure to any VGDFFiM are at a higher risk of reduced lung function at age 58–64 years. Occupational exposure to biological dusts or vapors also increased the risk of self-reported COPD.

1. Introduction

Occupational factors play an important role in adult-onset asthma (population-attributable risk (PAR) 17%) and chronic obstructive pulmonary disease (COPD) (PAR 15–20%) (European Respiratory Society, 2013). Occupational exposure to the main airborne pollutant forms, i.e. vapors, gases, dusts or fumes (VGDF) has been linked to higher prevalence, incidence and severity of COPD (Fishwick et al., 2015), even after adjustment for smoking, in both occupational and population-based studies, the majority of which were cross-sectional (Omland et al., 2014). Evidence from longitudinal population-based studies is, however, scarce and less consistent (Sunyer et al., 2005;

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http://dx.doi.org/10.1016/j.envres.2016.11.018
Received 21 July 2016; Received in revised form 24 November 2016; Accepted 25 November 2016

Mehta et al., 2012; Humerfelt et al., 1993; Kauffmann et al., 1982). Most longitudinal studies have relatively short follow-up periods with only one study reporting changes in lung function in relation to occupational exposure to VGDF over 25 years (de Jong et al., 2014).

Long-term prospective studies are needed to explore the relationship between occupational exposure and adult onset of obstructive respiratory disorders which have not been influenced by preceding respiratory symptoms and/or healthy worker effect (Le et al., 2008). The Aberdeen-based WHEASE (What Happens Eventually to Asthmatic children: Sociologically and Epidemiologically) cohort 1964–2014 is one of the longest follow-up studies of children in the world with a focus on airways disease. Moreover, clinical assessment of this cohort provides an opportunity to link occupational exposures to respiratory outcomes in adult life in subjects without childhood wheezing illness.

This study investigates whether lifetime occupational exposure to different airborne pollutant forms increases the likelihood of self-reported COPD, spirometry-defined airflow obstruction, and lung function impairment at age 58–64 years among individuals with no history of childhood wheezing illness. This study also investigates the longitudinal impact of occupational exposures on lung function over 25 years of follow-up.

2. Materials and methods

2.1. Subjects

The WHEASE cohort has been described in detail elsewhere (Tagiyeva et al., 2016). Briefly, in 1964 a random sample of 2511 (20% of all Aberdeen schoolchildren aged 10–15 years), was selected (Dawson et al., 1969) and administered the parent-completed Medical Research Council (MRC) questionnaire (Fletcher, 1960). Those with parentally reported history of ever wheeze were clinically assessed by a pediatrician and categorized into having childhood asthma or childhood wheezy bronchitis – now commonly known as virus-associated wheeze. Children without parent-reported history of ever wheezing were categorized as childhood "non-wheezers". The cohort was invited for the follow up in 1989 (aged 35–40), 1995 (aged 41–46), 2001 (aged 47–52), and 2014 (aged 58–64) (Tagiyeva et al., 2016; Bodner et al., 1998; Edwards et al., 2003; Godden et al., 1994) (Table 1).

In 2014 all individuals with spirometry who took part in at least one previous follow up were traced and invited to take part. Only childhood "non-wheezers" were included in the current study of occupational exposure and adult airway disease, i.e. those whose parents in 1964 reported that their child had no history of ever wheezing. Subjects who in 1964 were categorized as having childhood asthma or childhood wheezy bronchitis/virus-associated wheeze were excluded from the current study.

2.2. Health assessments and outcomes

Each follow up between 1989 and 2014 included the updated version of the MRC respiratory questionnaire (Medical Research Council, 1986) administered during in-person interview and spirometry with recorded forced expiratory volume in one second (FEV_1) and forced vital capacity (FVC). Spirometry was performed according to internationally accepted guidelines. The 2014 spirometry followed ATS/ERS guidelines (Pellegrino et al., 2005) with pre- and post-administration of 400 μg salbutamol using a Vitalograph Compact II spirometer (Vitalograph, Buckingham, UK). Measurement of forced expiratory flow over the middle half of FVC ($FEF_{25–75}$), which reflects small airway function, was also recorded in 2014.

Socio-demographic characteristics related to health status, i.e. cigarette smoking and deprivation were ascertained at each follow up. A history of cigarette smoking was assessed by pack year smoking histories, with a pack year defined as twenty cigarettes smoked every day for one year. The highest educational qualification was used as an indicator of childhood and adulthood socio-economic status (SES).

Table 1
The WHEASE cohort recruitment and follow up.

Original cohort	
1964	The Medical Research Council random survey of Aberdeen school children Age 10–15 years 2743 invited, 2511 participated: response rate 92% 121 child asthma, 167 child wheezy bronchitis, 2223 child non-wheezers Spirometry measured in 288 child asthma and wheezy bronchitis
⇩	
What Happens Eventually to Asthmatic children: Sociologically and Epidemiologically (WHEASE) cohort	
1989	WHEASE 1 to follow all child asthma and wheeze cases and selected non-wheezers Age 35–40 years 455 traced and invited, 360 participated: response rate 79% Participants included: 97 child asthma, 132 child wheezy bronchitis, 131 child non-wheezers Spirometry measured in 272, including 93 child non-wheezers
1995	WHEASE 2 to follow all child non-wheezers Age 41–46 years 1758 traced and invited to postal survey, 1542 participated in postal survey: response rate 88% Participants in postal survey: 102 adult onset wheeze (AOW), 1440 never-wheezers Clinical assessment (including spirometry) carried out in 312: 102 AOW and 217 randomly selected never-wheezers
2001	WHEASE 3 to follow all in WHEASE 1 and those with spirometry in WHEASE 2 Age 47–52 years 605 traced and invited, 381 participated: response rate 63% Participants included: 46 child asthma, 65 child wheezy bronchitis, 270 child non-wheezers Spirometry measured in 372, including 270 child non-wheezers
2014	WHEASE 4 to follow all WHEASE 1–3 with previously measured spirometry Age 58–64 years 583 traced and invited, 330 participated: response rate 57% Participants included: 38 child asthma, 53 child wheezy bronchitis, 239 child non-wheezers Spirometry measured in 329, including 239 child non-wheezers Occupational histories collected from 328, including 237 child non-wheezers

2.3. Occupational exposure

The 2014 follow up assessment also included a record of lifetime occupational history, including job titles, main job tasks, industry type, year when jobs started and stopped, and type of employment for each job (full- or part-time), obtained during the interview. Occupational histories were obtained for all jobs held for at least 12 months.

Free-text descriptions of occupations and industries for all lifetime jobs were coded into four-digit Standard Occupational Classification (SOC2000) codes developed by the UK Office for National Statistics (Office for National Statistics, 2000) using the Computer Assisted Structured Coding Tool (CASCOT) (Jones and Elias, 2006). This generates certainty scores (1–100%), indicators of the degree of certainty, that the given code is correct. The chosen coding strategy was to accept CASCOT derived codes scored $>50\%$ in a fully automatic mode and to code manually those scored $\leq 50\%$. In the automatic mode, the code with the highest score was automatically accepted. This coding strategy has been shown to have 91% agreement with manually coded occupations (Tagiyeva et al., 2010).

Occupational exposure to the complete range of airborne pollutant forms was assigned to each SOC code for each job held using the airborne chemical exposure job exposure matrix (ACEJEM). The