Occupational lung disease

S1 A NEW, EFFICIENT WEB-BASED TOOL TO COLLECT AND CODE LIFETIME JOB HISTORIES IN LARGE POPULATION-BASED STUDIES: THE COPD PROJECT IN THE UK BIOBANK COHORT

¹S De Matteis, ¹D Jarvis, ¹M Wheatley, ¹H Azhar, ²A Young, ²H Young, ¹L Rushton, ¹P Cullinan. ¹Imperial College London, London, UK; ²Oxford University, Oxford, UK

10.1136/thoraxjnl-2014-206260.7

Introduction and objectives The manual collection and coding of job histories is the standard method for assessing occupational exposure, but may be infeasible for large population-based studies such as the UK Biobank cohort.

Our aim was to develop a new web-based tool to automatically collect and code individual lifetime job histories in the UK Biobank cohort for investigating the causes and burden of work-related COPD in the UK.

Methods UK Biobank is a population-based cohort of 502,682 subjects, aged 40–69 years, recruited in 2006–2010. Baseline spirometry data, current employment and smoking histories were collected. We developed a job questionnaire based on the hierarchical structure of the standard occupational classification (SOC) 2000 to allow participants to automatically self-collect and code their lifetime job histories. The web-based prototype (www.imperial.ac.uk/biobank/questionnaire) was pre-piloted in May–August 2013 among key job sectors using snowball sampling together with a feedback survey.

Results 171 subjects participated in both the pre-piloting and feedback survey. 91% completed the questionnaire in <20 min, 85% considered the instructions clear, and 80% found their job categories/titles easily. Overall, 96% judged the questionnaire to be clear and easy. A revised questionnaire has now been designed and will be accessible from different media including PCs/laptops, tablets and smart phones to encourage high response. A demonstration version will be made available to conference participants.

Conclusions Our web-based job questionnaire is an efficient new standard tool for collecting and automatically coding lifetime job histories in large population-based studies and is adaptable for use in many occupational and environmental health research projects.

S2 DEVELOPMENT OF A JOB EXPOSURE MATRIX FOR SOC 2000 LISTINGS TO IDENTIFY OCCUPATIONAL CAUSES OF COPD

¹S Sadhra, ²D Fishwick, ³OP Kurmi, ²H Chambers, ¹KBH Lam, ⁴S Hutchings, ⁴D Jarvis, ⁴S De Matteis, ⁴L Rushton, ¹JG Ayres, ⁴P Cullinan. ¹University of Birmingham, Birmingham, UK; ²Health and Safety Laboratory, Buxton, UK; ³University of Oxford, Oxford, UK; ⁴Imperial College London, London, UK

10.1136/thoraxjnl-2014-206260.8

Introduction Occupational exposures are associated with the presence of a significant proportion of chronic obstructive pulmonary disease (COPD). The majority of the previous population studies have relied on self-reported exposures to vapours, gases, dusts and fumes (VGDF), which could lead to substantial misclassification. We aim to develop an occupational inhalation job exposure matrix (JEM) developed for use specifically with

SOC 2000 occupational codes covering a wider range of occupational airborne pollutant types.

Methods The development of airborne chemical exposure JEM (ACE-JEM) involved a four-stage approach; first, exposure (yes/no) to each of the six different airborne pollutants types (vapours, gases, dusts, fumes, fibres and mists; VGDFFM) was assessed for each of the 353 SOC codes, then three levels of exposure estimates (low, medium and high) (L-JEM1) and four levels of proportion exposed (0–4%, 5–9%, 20–49% and ≥50%) (P-JEM2) were assigned to the exposed codes and for each pollutant type. The two P and L JEMs were then combined to produce the final ACE-JEM. The estimated exposure of the 6 pollutant types was expanded to include biological dusts, mineral dusts, metals, diesel fumes and asthmagens.

Results For L-JEM1 186 (53%) of the codes were assigned as exposed to at least one type of VGDFFM. The most common exposure was dust (40% of all SOC codes) followed by fumes (26%). Over 68% of all codes were assigned as not being exposed to fibres, gases or mists. The pollutant with the highest proportion in the high exposure group was dusts (13%), and 33% of the codes were assigned as exposed to asthmagens. Overall, 53% of the codes were assigned as exposed to CGDF, with 22% assigned as having medium or high exposure to VGDF.

Discussion An expert assessment derived JEM has been developed, using a strict set of a priori defined rules. This JEM will assist attribution of possible harmful workplace exposures in future epidemiological studies. The ACE-JEM could also be applied to studies to assess risks of other respiratory diseases, including asthma and extrinsic allergic alveolitis.

S3 IDIOPATHIC PULMONARY FIBROSIS, MESOTHELIOMA, AND ASBESTOSIS MORTALITY TRENDS FOR ENGLAND AND WALES: IS ASBESTOS EXPOSURE ASSOCIATED WITH IPF?

¹C Reynolds, ²C Barber, ¹P Cullinan. ¹Department of Occupational and Environmental Medicine, National Heart and Lung Institute, London, UK; ²Centre for Workplace Health, University of Sheffield, Sheffield, UK

10.1136/thoraxjnl-2014-206260.9

Introduction and objectives Idiopathic pulmonary fibrosis (IPF) is more common in older people, in men, in manual workers, and in those living in more industrial areas of the country.

It has been hypothesised that workplace asbestos exposure is an under-recognised cause of IPF. Studies of mesothelioma patients have shown that asbestos exposure is poorly recalled. If asbestos exposure is not known, asbestosis can be misclassified clinically as IPF because both can present with progressive breathlessness and radiological UIP.

Our aim was to investigate a possible association between IPF and known asbestos-related mortality. Our objective was to visualise age-standardised annual mortality trends for IPF, mesothelioma, and asbestosis for men and women.

Methods Age, sex, and region stratified mortality data for IPF, mesothelioma, and asbestosis were obtained for England and Wales from the Office of National Statistics for the period 1974–2012. Data were age-standardised and visualised using the Python Pandas data analysis library and matplotlib.

Results The incidence of IPF continues to increase and is higher in men (male:female = 1:1.6) and in the north west of England. IPF and asbestos-related deaths are rare before the age of 40 and more common in elderly people.

Abstract S3 Figure 1

Conclusions New antifibrotic treatments for IPF throw into sharp focus the question of whether or not a proportion of IPF is due to occult asbestos exposure; patients known to have asbestos exposure are currently not considered to be candidates for antifibrotic treatments. Our data are consistent with a proportion of IPF being attributable to asbestos exposure but are not conclusive and further research is needed.

S4 RELATIONSHIP BETWEEN HISTORIC UK ASBESTOS IMPORTS AND ANNUAL MORTALITY DUE TO MESOTHELIOMA, ASBESTOSIS AND IDIOPATHIC PULMONARY FIBROSIS

CM Barber, RE Wiggins, D Fishwick. *Centre for Workplace Health, Health and Safety Laboratory, Buxton, UK*

10.1136/thoraxjnl-2014-206260.10

Background Previous studies from a number of countries have demonstrated that the rising mortality due to mesothelioma and asbestosis can be predicted from their historic asbestos usage. Mortality due to idiopathic pulmonary fibrosis (IPF) is also rising in the UK, without any identified explanation. This analysis compared annual male and female mortality due to IPF, asbestosis and mesothelioma, and examined the relationship between mortality and national asbestos import data.

Methods Mortality data for IPF and asbestosis in England and Wales were available from the Office for National Statistics (ONS). Data for mesothelioma deaths in England and Wales and historic UK asbestos import data were available from the Health and Safety Executive (HSE). The numbers of annual deaths due to each condition were plotted separately by gender, against UK asbestos imports 48 years earlier. Pearson correlation co-efficients were then calculated.

Results Correlation co-efficients for each condition are shown in Table 1. The annual number of deaths due to mesothelioma and IPF were significantly correlated with historical asbestos imports

Abstract S4 Table 1 Correlation co-efficients for historic national asbestos import data, plotted against annual mortality due to mesothelioma, asbestosis, and idiopathic pulmonary fibrosis (IPF)

	Mesothelioma	Asbestosis	IPF
Male	0.96 ($p < 0.001$)	0.87 ($p < 0.001$)	0.96 ($p < 0.001$)
Female	0.94 ($p < 0.001$)	-0.15 ($p = 0.32$)	0.97 ($p < 0.001$)

for both genders. For asbestosis mortality, a similar relationship was found for male but not female deaths.

Conclusion The strength of the association between IPF and historical asbestos imports was similar to that seen for mesothelioma mortality. This finding suggests that the role of occupational and environmental asbestos exposure in the aetiology of IPF requires further consideration.

S5 PREVALENCE OF IGE SENSITISATION TO 'IMPROVER MIX' ENZYMES AMONG SYMPTOMATIC BAKERS WORKING IN UK SCRATCH BAKERIES

¹M Jones, ¹J Welch, ¹J Turvey, ²J Cannon, ²B Fitzgerald, ¹J Szram, ¹P Cullinan. ¹Imperial College, London, UK; ²Royal Brompton and Harefield NHS Trust, London, UK

10.1136/thoraxjnl-2014-206260.11

Introduction and objectives Bakers working in supermarket scratch bakeries in the UK are exposed not only to flour and fungal alpha amylase but also other enzymes, incorporated in a pre-mixed bag of 'dough improvers'. Bakers and their employers are usually unaware of which enzymes are being used within the 'improver mix'. We have examined the question of whether supermarket bakers develop sensitisation to 'improver mix' enzymes other than fungal alpha amylase.

Abstract S5 Table 1 Sensitisation to 'improver mix' enzyme in bakers stratified by sensitisation to either flour and/or alpha amylase

	Quantity of enzyme used in 'improver mix' (ppm)	Sensitisation to 'improver mix' enzyme		
		All bakers*	Bakers co-sensitised to flour and/or alpha amylase*	Bakers not-sensitised to flour and/or alpha amylase*
'Improver enzyme' used in bakery				
Maltogenic amylase	40–50	12/260 (5%)	10/84 (12%)	2/176 (1%)
Cellulase	25	11/119 (9%)	8/26 (31%)	3/93 (3%)
Fungal Xylanase				
		40/260 (15%)	36/84 (43%)	4/176 (2%)
	20			
	4	32/141 (23%)	31/58 (53%)	1/83 (1%)
Fungal Xylanase	X 5 conc			
		25/260 (10%)	24/84 (29%)	1/176 (0.6%)
Lipase	20			
		35/260 (13%)	35/84 (42%)	0/176 (0%)
Bacterial Xylanase	10			
		32/260 (12%)	30/84 (36%)	2/176 (1%)
Fungal α -amylase	10			
		29/260 (11%)	27/84 (32%)	2/176 (1%)
Glucose Oxidase	5–10			
		15/260 (6%)	4/26 (17%)	1/93 (0.6%)
Bacterial α -amylase	0.5			