

# BARRIERS TO THE IDENTIFICATION OF OCCUPATIONAL ASTHMA

*By*

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## DEDICATION

This is for my father Derek Walters (1940-2011)... who would have kept a copy on the bookshelf, but probably wouldn't have read it unless it was about Frederick the Great or the Stuart monarchy... which unfortunately it isn't.

## ABSTRACT

Occupational asthma accounts for 1 in 6 cases of new-onset adult asthma and is associated with an estimated societal cost in the UK of £100 million per annum. The cost is somewhat avoidable if workers with occupational asthma are identified quickly and removed from exposure to a sensitizing agent. However many workers with occupational asthma go undiagnosed or experience a lengthy delay in diagnosis. The aim of this work was to identify the barriers to diagnosis of occupational asthma on the part of the worker and of the healthcare professional. The first study evaluated current practice in assessing working-age asthmatics for occupational asthma in a West Midlands primary care population, using UK national guidelines as a reference standard. The recorded prevalence of occupational asthma was much lower than expected (0-0.8%) and there was poor enquiry regarding occupation (14% of cases) and the effect of work on asthma symptoms (2% of cases) by primary healthcare professionals. The second study used a qualitative methodology to explore and define health beliefs and behaviours in workers with occupational asthma symptoms. The major influences on workers' health seeking behavior were determined from four themes: (1) workers' understanding of their symptoms, (2) working relationships, (3) workers' course of action with symptoms and (4) workers' negotiation with healthcare professionals. The third study aimed to define the important barriers to identifying occupational asthma from the point of view of healthcare professionals. High levels of enquiry about the nature of patients' work and work-relatedness of symptoms were reported by GPs, nurses and non-occupational lung specialist physicians, though the sample was biased towards enthusiasts. Despite this, low awareness and adherence to occupational asthma guidelines was evident in all non-specialist groups. The fourth study evaluated the feasibility of introducing an electronic occupational asthma screening tool for primary care. Healthcare professionals who used the tool found it to be quick and easy to implement and user-friendly (clear, concise, logical), without impacting on the length of a consultation. With adequate training for users the screening tool could be rolled out nationwide in the UK and its clinical effectiveness measured.

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## ABBREVIATIONS

ABC	Area Between the Curves
ANOVA	Analysis of Variance
ARNS	Association of Respiratory Nurse Specialists
BOHRF	British Occupational Health Research Foundation
BTS	British Thoracic Society
CD4	Cluster of Differentiation 4
CI	Confidence Interval
CL	Confidence Limit
COPD	Chronic Obstructive Pulmonary Disease
COREQ	Consolidated Criteria for Reporting Qualitative Research
COSHH	Control of Substances Hazardous to Health
CWI	Centre for Workforce Intelligence
DV	Diurnal Variation
ELISA	Enzyme Linked ImmunoSorbent Assay
EMIS	Egton Medical Information Systems
ERS	European Respiratory Society
FE <sub>NO</sub>	Fractional Exhaled Nitric Oxide
FEV <sub>1</sub>	Forced Expiratory Volume in 1 second
FOM	Faculty of Occupational Medicine
FVC	Forced Vital Capacity
GHM	General Hospital Medicine
GINA	Global Initiative for Asthma
GP	General Practitioner
GU	Genito-urinary
HCW	Healthcare Worker
HDI	Hexamethylene Diisocyanate

HMW	High Molecular Weight
HSE	Health and Safety Executive
IBM	International Business Machines
ICS	Inhaled Corticosteroid
IgE	Immunoglobulin E
IIA	Irritant Induced Asthma
IMD	Index of Multiple Deprivation
INPS	In Practice Systems
IT	Information Technology
LCL(W)	Lower Control Limit at Work
LMW	Low Molecular Weight
MDI	4,4 Methylene Diphenyl Diisocyanate
NHS	National Health Service
NIHR	National Institute for Health Research
NRES	National Research Ethics Service
NSBR	Non-Specific Bronchial Reactivity
OA	Occupational asthma
OASYS	Occupational Asthma System
OASYS-2	Occupational Asthma System 2
OH	Occupational Health
OLD	Occupational Lung Disease
ONAP	Observatoire National des Asthmes Professionnels
ONS	Office for National Statistics
OPRA	Occupational Physicians Reporting Activity
OR	Odds Ratio
PAR	Population Attributable Risk
PCRN	Primary Care Research Network
PCRS-UK	Primary Care Respiratory Society UK

PC <sub>20</sub>	Provocative Concentration inducing a 20% fall in FEV <sub>1</sub>
PD <sub>20</sub>	Provocative Dose inducing a 20% fall in FEV <sub>1</sub>
PEF	Peak Expiratory Flow
QOF	Quality Outcomes Framework
RADS	Reactive Airways Dysfunction Syndrome
RAST	Radioallergoabsorbant Test
RCGP	Royal College of General Practitioners
RCP	Royal College of Physicians
REC	Research Ethics Committee
RNV3P	Reseau National de Vigilance et de Prevention des Pathologies Professionnelles
R&D	Research and Development
SABRE	Surveillance of Australian Workplace Based Respiratory Events
SD	Standard Deviation
SENSOR	Sentinel Event Notification System for Occupational Risks
SHO	Senior House Officer
SIC	Specific Inhalation Challenge
SIGN	Scottish Intercollegiate Guidelines Network
SLRD	Short Latency Respiratory Disease
SOM	Society of Occupational Medicine
SPSS	Statistical Package for the Social Sciences
SPT	Skin Prick Test
SWORD	Surveillance of Work-related and Occupational Respiratory Disease
TDI	Toluene Diisocyanate
THOR-GP	The Health and Occupation Research Network in General Practice
TPP	The Phoenix Partnership
UK	United Kingdom
USA	United States of America
VCD	Vocal Cord Dysfunction

WA	Washington (State)
WEI	Work Effect Index
WEL	Work Exposure Limit

## CHAPTER 1: INTRODUCTION

### 1.1 Defining occupational asthma

#### 1.1.1 Overview

Occupational asthma is a disease characterized by variable airflow limitation and/or hyper-responsiveness that is caused by inhalation of an agent in the workplace (Baur et al., 2012). It is the most frequently reported occupational respiratory disorder in western industrialized populations (McDonald, 2000) and represents 1 in 6 cases of new-onset adult asthma in the UK (Nicholson et al., 2010). Occupational asthma includes:

1. Occupational asthma by sensitization: cases with an allergic mechanism where there is sensitization to an agent by an Immunoglobulin E (IgE) mediated, or different immunological or non-immunological mechanism, which occurs after a latent period of exposure (Baur et al., 2012), and
2. Irritant-induced asthma: resulting from a single high exposure to a major respiratory irritant without a latent period (Brooks et al., 1985), known classically as the Reactive Airways Dysfunction Syndrome (RADS).

Occupational asthma, along with work-exacerbated asthma which is characterized by worsening of pre-existing asthma caused by, but not arising de novo, from workplace inhalational exposures (Tarlo et al., 2008), come under the broad umbrella term of work-related asthma.

It is helpful to consider some general aspects of asthma, since identifying a case of occupational asthma requires a physician to diagnose a worker with asthma, as well as establish the relationship with work. The majority of workers will present to a physician with asthma symptoms, that is, any of chest tightness, cough, wheeze and breathlessness, the

nature of which are not specific to occupational asthma (BTS/SIGN, 2012). Work-related asthma should be considered in any adult with new onset asthma symptoms, worsening asthma symptoms or recurrence of childhood asthma (Fishwick et al., 2012; Nicholson et al., 2010; Tarlo et al., 2008). The Global Initiative for Asthma (GINA) (Bateman et al., 2008) defines asthma as a:

*“chronic inflammatory disorder of the airways in which many cells and cellular elements play a role. The chronic inflammation is associated with airway hyper-responsiveness that leads to recurrent episodes of wheezing, breathlessness, chest tightness, and coughing, particularly at night or in the early morning. These episodes are usually associated with widespread, but variable, airflow obstruction that is often reversible either spontaneously or with treatment”.*

There is considerable heterogeneity in the clinical presentation of asthma, and diagnosis requires a thorough clinical history and physical examination, as well as pulmonary function testing (BTS/SIGN, 2012). Variable and reversible airflow obstruction is a key feature of asthma, though lung function is often normal between exacerbations, or with treatment (Tarlo et al., 2008). However a >400ml increase in forced expiratory volume in 1 second (FEV<sub>1</sub>) as a response to inhaled beta-agonist or a treatment trial of corticosteroid (Hunter et al., 2002), or a peak expiratory flow (PEF) diurnal variation of >20% after PEF monitoring (Quackenboss et al., 1991), are strongly supportive of a diagnosis of asthma. In patients with normal or near-normal spirometric values, assessment of airway responsiveness by inhalational challenge with histamine or methacholine should be undertaken. A methacholine PC<sub>20</sub> (provocative concentration inducing a 20% fall in FEV<sub>1</sub>) of <8 mg/ml, or PD<sub>20</sub> (provocative dose) of <4 micromols, has a sensitivity of between 60-100% in detecting asthma (BTS/SIGN, 2012). In addition a prior history of atopy, family history of asthma and/or atopy, or peripheral blood eosinophilia make a diagnosis of allergic asthma more likely (BTS/SIGN, 2012).

### 1.1.2 Occupational asthma by sensitization

More than 400 respiratory sensitizers causing occupational asthma have been identified, with new agents continually reported (Pralong et al., 2013a; Lemi re et al. 2012). Compendia of causative agents can be found in publications (Malo and Chan-Yeung, 2009; Fishwick, 2012; Currie and Ayres, 2005; Baur 2013) or on websites (OASYS group, 2013). Common causes of occupational asthma by sensitization are shown in Table 1.1.

	<b>Agent</b>	<b>Occupational exposure</b>
High-molecular weight agents	Flour, grain and baking enzymes Laboratory animals Seafood (fish, molluscs, crustacea) Flower pollen Green coffee and castor bean Latex Wood dust (Western red cedar, pine, iroko)	Baking, pastry making, food processing Laboratory workers, animal handlers Seafood workers Gardening industries Food production Healthcare workers Woodworkers, millworkers
Low-molecular weight agents	Isocyanates  Platinum salts Colophony (pine resin) Acid anhydrides Acrylates  Drugs (antibiotics, cimetidine, anaesthetic agents) Dyes Persulphate Metals chromium, nickel, cobalt, zinc Metalworking fluid Cleaning agents (enzymes, quaternary ammonium compounds) Glutaraldehyde	Spray painting, moulding, core making, printing, assembly, printing, rubber manufacture Platinum refinery, chemical manufacture Soldering Spray painting, epoxy manufacturing Plastics manufacturing, orthopaedic surgery, dentistry Chemical manufacturing, healthcare workers  Textile workers Hairdressers Metalworkers, metal production, welding Metalworkers Cleaners, healthcare workers  Healthcare workers

**Table 1.1. Common causes of occupational asthma by sensitization, along with occupations where exposure usually occurs. Adapted from Currie and Ayres (2005), Nicholson et al. (2010) and Walters et al. (2013).**

The agents can be categorized on size, as either high molecular weight (HMW) and >10,000 Dalton, or low molecular weight (LMW) and <1000 Dalton (Lummus et al., 2011). HMW agents, of which about 190 have been identified (Lummus et al., 2011) are molecules, usually proteins but some are complex polysaccharides, which are capable of inducing specific IgE antibody-responses and are usually associated with sensitization to animals, plants, or microorganisms. Some agents have functional characteristics that promote their allergenicity, such as protease activity in the case of detergent enzymes (Schweigert et al., 2000; Jacquet, 2011); others possess receptors that enhance the innate immune response, such as toll receptors in the case of house dust mites (Nathan et al., 2009). The majority of HMW agents have been purified and expressed recombinantly to make solutions useful for allergy testing (Lummus et al., 2011). Approximately 80 LMW agents have been described as causes; these are usually chemical molecules and include methylene diphenyl diisocyanate (MDI), acid anhydrides, amines, metals, and reactive dyes (Nicholson et al., 2010; Lummus et al., 2011). In the case of LMW agents, certain characteristics promote reactivity and allergenicity, particularly functional groups containing 2 or more reactive nitrogen or oxygen atoms, or the ability to conjugate with lysine (Jarvis et al., 2005). Isocyanate and acid anhydride functional groups have been demonstrated to form conjugates with larger molecules such as human albumin protein (haptens), causing rearrangement changes which are then capable of causing sensitization (Lummus et al., 2011).

Many cases of occupational asthma are due to specific IgE-mediated sensitization, where IgE binds to mast cell receptors, leading to degranulation and release of asthmatic mediators in susceptible individuals. Sensitization occurs by inhalation and uptake of the antigen by antigen-presenting cells, often dendritic cells, which then migrate to regional lymph nodes where they are presented to CD4 T-helper cells that initiate an immune response (Lummus et al., 2011). However cell-mediated immunity or delayed-type hypersensitivity has been postulated as a possible mechanism for some LMW chemical induced asthma, for example, due to persulphates (Yawalkar et al., 1999), or hexamethylene diisocyanate (HDI)



(Wisnewski et al., 1999). Additionally there is evidence that suggests that some isocyanate chemicals stimulate innate immune responses and this may contribute to sensitization (Lummus et al., 1998; Wisnewski et al., 2008). Holgate has suggested that the primary defect is in airway epithelium (Holgate, 2008; Holgate, 2010), and the immune response is a secondary phenomenon to impaired barrier function, allowing greater access for environmental allergens, microorganisms, and toxicants; which in turn triggers allergic-type inflammation (Holgate et al., 2000; Holgate et al., 2009). There may be internal (genetic) or external factors (occupational exposures) that modulate the normal epithelial damage-repair cycle of the human airways (Holgate et al., 2007).

### 1.1.3 Irritant-induced asthma

Occupational asthma with latency is usually caused by sensitization (Newman Taylor, 1980). However Brooks *et al.* (1985) described the Reactive Airways Dysfunction Syndrome (RADS), that is, acute onset of new asthma symptoms within 24-hours of a sudden exposure to a major respiratory irritant (vapour, gas, fume or smoke) and persistence of symptoms and bronchial hyper-responsiveness for at least 3-months after the incident. Workers with RADS do not become sensitized to the causative agent and so subsequent low-dose exposures do not cause problems.

RADS is now included in the spectrum of irritant induced asthma (IIA). Agents in the workplace causing IIA are many and include acids (sulphuric, hydrochloric), alkalis (sodium hydroxide, calcium oxide), gases (chlorine, bromine, ammonia), organic solvents, diesel exhaust, aluminium pot-room fumes, sulphur dioxide from apricot sulphurization, and also known sensitizers such as glutaraldehyde and metalworking fluid in very high concentrations (Brooks, 2013; Shakeri et al., 2008). Crucially almost all cases are associated with unanticipated release often after explosion or fire, or in confined spaces, therefore it is rarely possible to quantify the level of exposure causing IIA.

Brooks et al. (1998) subsequently reported a series of cases they described as "not-so-sudden-onset" IIA, with a latent period of days or weeks between repeated inhalation exposures and new onset asthma symptoms. The exposures could be intermittent and less intense than those of RADS when it was originally described, but symptoms needed to occur within 4-months of initial exposure. Burge et al. (2012) described a third group of IIA with low-level irritant exposure and development of asthma symptoms after a symptomless latent interval, which could be several years. When so-called "IIA with latency" has developed, usual level exposures trigger asthma symptoms, as for occupational asthma with hypersensitivity. At diagnosis, IIA with latency is indistinguishable from hypersensitivity occupational asthma with respect to any pre-existing asthma or atopy, smoking, latent interval and non-specific reactivity (Adewole et al., 2009; Burge et al., 2012). Whether the prognosis of IIA with latency differs from hypersensitivity occupational asthma is unknown and requires further study.

#### 1.1.4 Differential diagnosis of occupational asthma

The symptom profile of chest tightness, breathlessness, cough and wheeze is not specific for asthma and there are a number of conditions that co-exist with, exacerbate or mimic asthma, which must be considered when making a diagnosis; many of these can also be work related. Following odorant and irritant exposures, a spectrum of clinical syndromes can occur, termed "sensory hyper-reactivity" (Tarlo et al., 2008) which include increased cough reflex (Gordon et al., 1998), odour-driven panic, increased sensory awareness (Shusterman et al., 1991; Dager et al., 1987) and more ill-defined upper airway symptoms (Ternesten-Hasseus et al., 2002), none of which are associated with increased bronchial hyper-responsiveness.

Additionally, vocal cord dysfunction (VCD) closely mimics asthma by vocal cord adduction during inspiration leading to airflow obstruction. Odorants and irritants can precipitate episodes of VCD (Tarlo et al., 2008), so episodes may be work-related. The mechanisms of VCD are unknown, though gastro-oesophageal reflux and psychogenic factors are likely to

contribute. Diagnosis of VCD is through direct visualization of the vocal cords by laryngoscopy.

Another differential diagnosis is eosinophilic bronchitis, a condition where subjects experience asthma symptoms accompanied by sputum eosinophilia, but without demonstration of airway hyper-responsiveness to methacholine or histamine (Cartier and Sastre, 2011; Pala et al., 2012). It has been reported in workers exposed to methyl methacrylate (Quirce et al., 2004), latex (Quirce et al., 2003), mushroom spores (Tanaka et al., 2002) and lysozyme (Escudero et al., 2003).

#### 1.1.5 Occupational asthma guidelines

Preconditions for making a diagnosis of occupational asthma are the typical diagnostic findings of asthma, along with an association of asthma symptoms with exposure at work (Aasen et al., 2013). A detailed occupational history should be taken (Baur et al., 2012; Nicholson et al., 2010; Fishwick et al., 2012) which will enable the physician to determine the degree of pre-test probability of occupational asthma. In its evidence review and guidelines, the British Occupational Health Research Foundation (BOHRF) details work that it considers to be high-risk (see Table 1.1), which should create a high index of suspicion; this includes spray painting, health and dental care, chemical processing, baking and pastry making, food processing, welding, soldering, metalworking, woodworking, plastics and rubber manufacture, laboratory animal work, textiles, farming and hairdressing (Nicholson et al., 2010).

European Respiratory Society (ERS) guidelines assert that the diagnosis of occupational asthma should be made in 3 steps: (1) make the diagnosis of asthma, (2) identify the workplace as the cause of the patient's asthma and (3) identify the specific agent causing occupational asthma (Baur et al., 2012; Aasen et al., 2013). In UK hospitals the availability of resources necessary for full investigation of a patient with occupational asthma varies

between 3% and 100% depending on the resource examined (Barber et al., 2008). For example, only 3% of centres have the ability to perform specific bronchial challenge or workplace challenge, and only 15% have computerized peak expiratory flow (PEF) analysis software. Therefore, if a diagnosis of occupational asthma is suspected, the British Thoracic Society (BTS) recommends early referral to a specialist centre (Fishwick et al., 2012).

A diagnosis of occupational asthma should not be made on history alone and objective evidence should be gathered and triangulated: (1) objective evidence of exposure to known sensitizers (or airway irritants in the IIA), (2) demonstration of an association between exposure and airflow limitation at work, by serial PEF measurement, and (3) demonstration of a specific immunological reaction to the occupational agent (Baur et al., 2012; Aasen et al., 2013; Fishwick et al., 2012).

## 1.2 Epidemiology of occupational asthma

### 1.2.1 Reporting schemes

Occupational asthma has become the most frequently reported occupational respiratory disease in industrialized populations (Elder et al., 2004; McDonald et al., 2000; McDonald et al., 2005; Provencher, 1997; Newman Taylor, 2002). Its prevalence is not clearly defined due to inconsistent case definitions and a lack of surveillance data in the general population and exposed populations (Nicholson et al., 2010). Potential sources of data include workers' compensation records and health surveys; however these were often established to serve other functions, and therefore occupational information is incomplete or not collected, limiting the ability to identify cases (Hannaford-Turner et al., 2010). A number of reporting registries for occupational asthma exist worldwide, the majority of which are voluntary rather than mandatory.

One of the first voluntary reporting schemes was the Surveillance of Work related and Occupational Respiratory Disease (SWORD) scheme in the UK (Meyer et al., 2001), with

monthly submission of newly diagnosed cases of occupational respiratory illness which, in the opinion of specialist chest and occupational physicians, are caused by work. SWORD is coupled with a separate scheme, Occupational Physicians Reporting Activity (OPRA), which takes submissions from a larger number of occupational physicians (Cherry et al., 2000).

The Midland Thoracic Society's Rare Respiratory Disease Registry Surveillance Scheme of Occupational Asthma (Shield) is the only regional voluntary reporting scheme for occupational asthma in the UK, and is based in the West Midlands (Gannon and Burge, 1991; Gannon and Burge, 1993), taking reports from regional occupational health physicians and respiratory physicians.

In Finland it is mandatory for all cases of occupational asthma to be notified to the statutory Finnish Registry of Occupational Diseases (Karjalainen et al., 2000), and in Norway physicians are obliged to report occupational diseases, including occupational asthma, to a national compensation scheme (Leira et al., 2005). Elsewhere, the Surveillance of Australian workplace Based Respiratory Events (SABRE) exists in some Australia regions (Elder et al., 2004; Hannaford-Turner et al., 2010), the project SENSOR exists in Michigan, USA (Baker, 1989), and there are other schemes in Canada (Provencher et al., 1997), South Africa (Hnizdo et al., 2001) and Spain (Orriols et al., 2006). Incidence estimates are available from these surveillance schemes, and asthma rates vary between countries and regions depending on the distribution of industry and occupation, the case definition for occupational asthma and method of data collection.

### 1.2.2 Incidence data

Blanc and Toren (1999) carried out a synthesis of the available literature to estimate the proportion of adult asthma in the general population attributable to workplace factors, by calculating the population attributable risk (PAR) for occupational asthma, when considering new-onset disease and reactivation of pre-existing asthma. They obtained 43 PAR estimates

from 19 countries and the median value was 9%, though this increased to 15% when using studies of higher quality. The estimated incidence of occupational asthma varied widely among countries from 1.2-17.4/100,000 person-years with a median incidence of 4.7 cases/100,000 person-years. The highest rate of 17.4/100,000 was observed in Finland from asthma medication registry data (Karjalainen et al., 1998). They noted that where PARs were calculated from incidence data, values were lower than those derived from available population data, which suggested under-reporting of incident cases of occupational asthma (Blanc and Toren, 1999). The same authors updated their calculation of median PAR for occupational asthma to 17.6% (Toren and Blanc, 2009), using newer large general population-based longitudinal studies, allowing for more reliable risk estimates. Thus 1 in 6 cases of new-onset adult or recurrent-childhood asthma is likely to be due to workplace factors (Toren and Blanc, 2009; Nicholson et al., 2010; Baur et al., 2012).

Incidence measures should be interpreted with caution for a number of reasons. Systematic changes in membership of surveillance schemes over time can produce false trends: for example, early scheme entrants may have more of an interest in occupational lung disease than later entrants, and new members will report prevalent cases initially as well as incident cases. Additionally there is variation in case definition between specialist centres and individuals: Baldwin et al. (2002) showed that even occupational asthma experts interpret diagnostic tests differently. There is also variation in method of reporting, whether compulsory or voluntary, as well as differences in the exposed populations between regions or countries, and changes over time.

The incidence of occupational asthma was 174 cases per million workers in Finland, according to what is considered to be the most complete surveillance system (Karjalainen et al., 2000). However other estimates have varied: in a prospective pan-European population-based cohort of young adults aged 20-44 (European Community Respiratory Health Survey: 1990–1995) the annual incidence of occupational asthma was 248–303/million workers per

annum, estimated using asthma-specific job exposure matrices (Kogevinas et al., 2007). From 1996-1999, mean annual incidence of occupational asthma was 24 cases per million workers calculated from the French Observatoire National des Asthmes Professionnels (ONAP) voluntary surveillance programme for occupational and chest physicians. This number of reported cases was two-fold higher than the number of cases of occupational asthma compensated over the same period in France (Ameille et al., 2003). Similar figures were retrieved from SWORD 1992-2001 (22 cases per million workers; McDonald et al., 2005), SHIELD 1991-2005 (42 cases per million workers; Diar Bakerly et al., 2008), project SENSOR from Michigan, USA (27 cases per million workers; Henneberger et al., 1999), a mandatory physician reporting scheme in California (25 cases per million workers; Reinisch et al., 2001) and from the Belgian workers' compensation board (29 cases per million workers; Vandenplas et al., 2011).

### 1.2.3 Under-reporting of occupational asthma

Data from Europe shows that occupational asthma is under-reported to surveillance schemes. Leira et al. (2005) showed that occupational asthma is grossly under-reported to the mandatory Labour Inspection Authority registry in Norway, where only 223 reports were made between 1995 and 1999, from an expected 1,110 reports per annum based on estimates from a population-based cohort study (Karjalainen et al., 2001). A voluntary surveillance system implemented in Catalonia in Spain taking reports from 142 chest and occupational physicians was noted to give a four-fold higher total number of cases of occupational asthma in 2002 (174 cases) than the Catalonian compulsory reporting system (42 cases), implying a large degree of under-reporting (Orriols et al., 2006). In 2003 after the voluntary register had been adopted by 3 provinces (Orriols et al., 2010), the discrepancy persisted as a two-fold increase in cases (166 voluntary versus 80 compulsory). A wide variation in the incidence of occupational asthma was noted (median incidence=57 cases per million workers/annum; range 35.6-161.4).

#### 1.2.4 Incidence trends

There has been a decline in notifications to various reporting schemes for occupational asthma in the UK. Carder et al. (2012) analyzed reports of work-related respiratory disease from the SWORD reporting scheme for respiratory physicians (1996-2011), and observed that the mean annual decrease in the incidence of occupational asthma was greater than that observed for total respiratory disease at -7.7% (95% CI: -9.2 to -6.2), with reporter fatigue indicated by a sharper decline in notifications from general respiratory physicians (-9.1%; 95% CI: -12.7 to -5.3) when compared with core specialist reporters (-7.4%; 95% CI: -9.0 to -5.7), and by an increase in nil returns and non-response. McNamee et al. (2008) had previously compared more limited SWORD data (1996-2005) with that of OPRA and observed that the magnitude of annual change in incidence was often inconsistent: for occupational asthma it was -1.9% (95% CI: -5.2 to 1.4) and -12.1% (95% CI: -19.5 to -4.1) using respiratory physician and occupational physician reports, respectively. The authors concluded that the differences were in part consistent with differential reporting behaviour, specifically greater reporter fatigue in occupational physicians than in core reporting respiratory physicians. However, it should be noted that preventative workplace interventions would likely be more prevalent in workplaces with attending occupational physicians than in the general workforce, and referral patterns to specialist respiratory physicians within the NHS may be subject to change over time.

Stocks et al. (2013a; 2013b) demonstrated that preventative workplace interventions and control measures could, at least partly, account for reduced incidence of occupational asthma in the UK. A significant reduction in SWORD notifications of short latency respiratory disease (SLRD - including occupational asthma) was seen for all agents where work-exposure limits (WELs) had been introduced through COSHH (2002) legislation. Reductions in SLRD were also observed with colophony, attributed to a Health and Safety Executive (HSE) inspection pack for exposed workers, and with glutaraldehyde and latex in healthcare workers, attributed to substitution in National Health Service (NHS) clinical areas.



Diar Bakerly et al. (2008) reported that the incidence of occupational asthma in the West Midlands was stable between 1991 and 2005 (median incidence=42 cases per million workers; 95% CI=37 to 45). However, a more up to date analysis showed a statistically significant 5.5% year-on-year decrease in annual incidence (1991-2011) when a negative binomial regression model was applied (Walters et al., 2013). Incidence was highest in 1995 (58 cases per million workers), and fell to 14 cases per million workers per year by 2011. Notifications to the SHIELD surveillance scheme due to most causative agents declined year on year, with the largest reductions seen for latex, glutaraldehyde and colophony, where workplace substitution has been possible. Only the incidence of occupational asthma due to cleaning agents showed a year-on-year increase, which is consistent with similar reports from the SWORD surveillance scheme and from a French registry (Stocks et al., 2012a; Paris et al., 2012). Some cleaning agents have been demonstrated to be respiratory sensitizers, principally quaternary ammonium compounds (Vandenplas et al., 2013a). However, it remains unclear as to whether increasing reports represent better awareness amongst healthcare professionals or greater biocide potency (Pechter et al., 2005; Arif et al., 2012; Delclos et al., 2007). Reporter fatigue amongst occupational physicians and general respiratory physicians was observed, with very few notifications from anywhere other than the Birmingham specialist occupational lung disease clinic seen after 2000. Overall notifications from the specialist clinic fell significantly year-on-year by 3.9% after the effect of reporter fatigue had been taken into account. The authors suggested that for many agents this demonstrated increasing lack of recognition in the workplace and amongst healthcare professionals, rather than a true reduction in incidence due to control measures. This was illustrated by a 5.3% year-on-year decline in notifications due to bakers' asthma (due to flour and enzymes) from industries in the West Midlands, where it was argued that the number of exposed workers and control measures were unlikely to have changed considerably over the study period (Walters et al., 2013).

In Europe, Vandenplas et al. (2011) observed a downward trend in incidence using a negative binomial regression analysis of Belgian Workers' Compensation Board data from 1993-2002. Mean average annual incidence of occupational asthma was 29.4 new cases per million salaried workers (95% CI: 27.6 to 31.3) during the study period, and there was a significant decline in the overall incidence rate of occupational asthma from 35.5 new cases per million salaried workers in 1993 to 25.8 in 2002. Paris et al. (2012) assessed annual trends from notification data from the French national surveillance network, Réseau National de Vigilance et de Prévention des Pathologies Professionnelles (RNV3P), and demonstrated an overall decrease in incidence of work-related asthma over the period 2001-2009 (Kendall tau= -0.722; p=0.0006). A significant decrease was observed for some agents such as isocyanates (tau=-0.41; p<0.001), aldehydes (tau=-0.40; p<0.001) and latex (tau=-0.24; p=0.001), while a significant increase was observed for quaternary ammonium compounds (tau=0.34; p<0.001). Data from the Finnish Register of Occupational Diseases (1986-2002) indicated that notifications increased from 1986 to 1995, decreased markedly between 1995 and 1998, and remained stable thereafter. This sharp downward trend was attributed to a decrease in cases due to HMW agents (for example, cow dander, flour and grain, and storage mites), which was in turn related to a decrease in agricultural activities and improvements in dairy farming processes (Piipari and Keskinen, 2005).

#### 1.2.5 Under-recognition in primary care

The apparent decline in bakers' asthma in the West Midlands has provided some evidence for under-recognition by healthcare professionals. There is also evidence that general practitioners (GPs) do not identify potential cases and refer for specialist opinion. In a small cross-sectional study of adult asthmatics in one primary care practice in Oxfordshire, 32% of adult new-onset asthmatics were in jobs known to be at significant risk of occupational asthma, yet a potential link between occupation and symptoms had only been recorded in 18% of patients (de Bono and Hudsmith, 1999). 7 out of 182 (4%) of the patients with adult onset asthma had been given a diagnosis of occupational asthma, although in nearly half (3

out of 7) of these cases a GP had made the diagnosis without referral to a specialist. A cross-sectional study from Sheffield, UK revealed that 50% of patients diagnosed with occupational asthma have seen their GP more than 5 times with work-related asthma symptoms before specialist referral occurs (Fishwick et al., 2007). Furthermore Hoyle et al. (2010) reviewed cases of undetermined work-related respiratory illnesses that had been reported to The Health & Occupation Reporting network in General Practice (THOR-GP) at the University of Manchester, UK and found that only 26% were referred for specialist opinion and diagnostic clarification despite uncertainty over diagnosis: of those 85% were exposed to known asthmagens.

Lack of enquiry about work exposures has also been shown to delay the diagnosis of occupational asthma, and in studies from the UK and Canada mean delays of 3-4 years from symptom onset to diagnosis or referral to a specialist have been identified (Fishwick et al., 2007; Santos et al., 2007; Poonai et al., 2005). In a cross-sectional study using chart reviews and telephone interviews from Canada, the mean time from symptom onset to diagnosis was 4.9 years and patients waited 7.4 months before discussing the work-relation of symptoms with a doctor. The most commonly reported reason for this was lack of enquiry by the attending doctor (Poonai et al., 2005). In closed question responses from cross-sectional studies, GPs have cited insufficient time, lack of expertise and poor access to specialist services as barriers to the diagnosis of occupational asthma on their part (Parhar et al., 2011; Holness et al., 2007; Poonai et al., 2005). In response to such findings, BOHRF has published guidance for GPs and primary care nurses on assessment and management of patients with suspected occupational asthma (BOHRF, 2010a).

Workers are reluctant to consult healthcare professionals with new or worsening asthma symptoms, and asthmatic workers have been shown to endure work practices known to expose them to respiratory hazards, despite being aware of the health risks (Slater et al., 2000). Poonai et al. (2005) found that there was a mean delay of 8 months from symptom

onset before workers consulted a physician, where many workers (32%) had anticipated the likely diagnosis from the onset of symptoms. Fear of losing work time or employment, and lack of awareness of respiratory hazards at work have been cited as potential reasons for these phenomena (Poonai et al., 2005; Santos et al., 2007; Gordon et al., 1997). In a qualitative study of attitudes towards workplace health in symptomatic workers with occupational asthma from the UK, workers identified fear of losing work time and income, or fear of unemployment, as themes that hindered engagement with management and occupational health services on health issues (Bradshaw et al., 2007).

#### 1.2.6 Occupational risk factors

The occupational history should focus particularly on exposures occurring at the time that asthma symptoms started or worsened at work. The latency for LMW agents and a few HMW sensitizers (for example, laboratory animals) is typically within 2 years of ongoing exposure (Malo et al., 1992; Malo et al., 2001), whilst it is usually longer for most other HMW sensitizers: for example, there is an average symptom latency of 5 years for flour or latex after initial exposure (Allmers et al., 1996; Malo et al., 2001). There are >400 reported workplace sensitizers and many documented at-risk occupations. The most frequently reported agents include isocyanates, flour and grain dust, colophony and fluxes, latex, animals, aldehydes, adhesives, metals, resins and wood dust (Nicholson et al., 2010; Bakerly et al., 2008; Ameille et al., 2003; McDonald et al., 2005; Orriols et al., 2006; Toren and Blanc, 1999). The occupations most commonly reported to surveillance schemes of occupational asthma are animal handlers, bakers and pastry makers, chemical workers, food processing workers, hairdressers, paint sprayers, nurses and other health professionals, timber workers and welders (Bakerly et al., 2008; McDonald et al., 2005; Nicholson et al., 2010; Tarlo et al., 2008).

A number of population-based studies have also identified occupations with an increased risk of developing occupational asthma: in a pan-European cohort of 6837 workers aged 20-44

high risk occupations for developing asthma were nursing, cleaning, baking and spray painting (Kogevinas et al., 2007). In a case-control study by Jaakkola et al. (2003) asthma risk was increased for workers employed in a variety of occupations (illustrated in Table 1.2) when using professionals, clerks and administrative workers as the reference category.

Occupational group	Men		Women		Total	
	<sup>1</sup> OR	95% CI	<sup>1</sup> OR	95% CI	<sup>1</sup> OR	95% CI
Bakers and food processors	8.62	0.86, 86.5	0.83	0.22, 3.05	1.71	0.58, 5.10
Forestry and related workers	6.00	0.96, 37.5	-	-	-	-
Chemical industry workers	5.56	0.48, 64.7	5.69	0.58, 55.8	5.69	1.08, 29.8
Dentists and dental workers	-	-	4.74	0.48, 46.5		
Textile workers	4.70	0.29, 77.1	0.88	0.34, 2.26	1.19	0.49, 2.88
Metalworkers	4.52	2.35, 8.70	-	-	-	-
Waiters	-	-	3.03	1.10, 8.31	-	-
Rubber and plastic workers	2.70	0.58, 12.7	2.52	0.58, 10.9	2.61	0.92, 7.42
Electrical and electronic production workers	2.83	0.82, 6.93	0.61	0.11, 3.51	1.52	0.61, 3.80
Wood and paper workers	1.73	0.60, 4.99	2.03	0.33, 12.4	1.72	0.71, 4.17
Laboratory technicians	1.66	0.17, 16.6	0.44	0.05, 4.00	0.75	0.15, 3.74
Storage workers	1.57	0.40, 6.19	0.98	0.27, 3.60	1.21	0.47, 3.12
Cleaners	-	-	1.42	0.81, 2.48	-	-
Professionals, clerks, and administrative	1.00					

**Table 1.2. Adjusted odds ratios of asthma in different occupational groups from the Finnish Environment and Asthma Study, 1997-2000 (Jaakkola et al., 2003).**

**Professionals, clerks, and administrative workers were considered the reference category. For male workers, risk was increased for bakers and food processors, textile workers, electrical and electronic production workers, laboratory technicians and storage workers, forestry work, and metalwork. For women, asthma risk was increased for waiters, cleaners and dental workers. <sup>1</sup>Odds ratios were adjusted for age and smoking. OR=odds ratio; CI=confidence interval.**

The risk of sensitization is typically greater with higher and more frequent exposures, and a positive exposure-response relationship has been demonstrated for many agents, for example, organic acid anhydrides (Nielsen et al., 2001), bakery enzymes (Brant et al., 2005a), laboratory rat urine (Heederik et al., 1999), and platinum salts (Merget et al., 2000).

Respiratory protective devices may be used to reduce exposure to sensitizers but do not provide complete protection, as sensitization can still occur despite use or where measured air levels are extremely low or undetectable (Baur et al., 1998a). However, if exposure levels of some allergens fall short of determined limit values, they are not associated with an increased risk of occupational asthma: Baur et al. ascertained lower limit threshold values for a number of agents: wheat flour (1-2.4 mg/m<sup>3</sup>), fungal alpha-amylase (0.25 ng/m<sup>3</sup>), natural rubber latex (0.6 ng/m<sup>3</sup>), Western red cedar (0.4 mg/m<sup>3</sup>) and rat urine (0.7 microg/m<sup>3</sup>).

#### 1.2.7 Other risk factors

Atopy describes a tendency to hypersensitivity, and is demonstrated by the propensity to produce specific IgE to common aeroallergens, which can then be measured or demonstrated by skin prick testing. Atopy has been reported to increase the risk of occupational asthma and sensitization caused predominantly by high molecular weight agents such as animal dander (Krakowiak et al., 2007), flour and amylase (De Zotti and Bovenzi, 2000; Walusiak et al., 2004) and shellfish (Jeebhay et al., 2008; Cartier et al., 1984; Desjardins et al., 1995).

Rhinitis and rhino-conjunctivitis symptoms (runny nose, itchy nose, nasal blockage, and sneezing) occur frequently with occupational asthma (45-100% cases; Nicholson et al., 2010) and have been attributed to LMW agents such as organic acid anhydrides (Grammer et al., 2002; Nielsen et al., 2006), lasamide (Klusackova et al., 2007) and persulphates (Moscato et al., 2005), and to HMW agents such as wheat flour (Gautrin et al., 2002), snow crab (Cartier et al., 1984) and laboratory rats (Cullinan et al., 1999). However, rhinitis symptoms are more intense when associated with HMW agents (Malo et al., 1997) and, indeed, features of occupational rhinitis are more likely to precede IgE-associated occupational asthma (Castano

et al., 2009), where the risk of developing asthma is highest in the year after the onset of rhinitis symptoms (Cortona et al., 2001; Gautrin et al., 2001; Grammer et al., 2002; Gross, 1980; Karjalainen et al., 2003).

Cigarette smoking has been shown to increase the risk of sensitization to a number of agents, including platinum salts, seafood, flour and green coffee and castor bean (Nicholson et al., 2010), and increase the risk of occupational asthma for isocyanates, platinum salts and seafood, though its role is unclear for many agents. Venables et al. (1989a) showed a significant dose-response effect of cigarette smoking on the risk of sensitization, as demonstrated by skin-prick testing to platinum salts.

### 1.3 Diagnosis of occupational asthma

#### 1.3.1 Expert opinion

The symptoms of occupational asthma are on the whole indistinguishable from non-occupational asthma. Deterioration of asthma symptoms on workdays is poorly predictive for occupational asthma, with only 31-46% of those with clinical suspicion going on to have positive specific inhalation challenge (SIC) tests (Axon et al., 1995; Baur et al., 1998b, Koskela et al., 2003; Malo et al., 1991; Cote et al., 1990). Some factors may be useful in discriminating however: those with occupational asthma are less likely to report seasonal or allergic exacerbations, pet-related or stress triggers or a family history of asthma, than those with non-occupational asthma (Axon et al., 1995). Two authors have described high sensitivities (93-100%) for the general expert history in the diagnosis of occupational asthma (Cote et al., 1990; Ricciardi et al., 2003), and in a prospective study of 45 patients, Vandenplas et al. (2001) reported a high sensitivity (87%) and low specificity (14%) when compared with SIC as the gold standard.

### 1.3.2 Questionnaires

Several questionnaires have been validated for screening asthma in occupational settings: for office workers (Venables et al., 1993), laboratory animal and flour processing workers (McKinlay and Venables, 1993), healthcare workers (Delclos et al., 2006) and woodworkers (Schlunssen et al., 2004). However one such questionnaire had low sensitivity (28%) and specificity (73%) in detecting asthma measured by non-specific bronchial responsiveness (NSBR) in shipyard workers (Stenton et al., 1993), warranting caution in interpretation of such questionnaires in surveillance or specific workplace settings.

A few studies have looked at questionnaires to identify specifically occupational asthma. Malo et al. (1991) used an open questionnaire undertaken by interview with 162 workers. The overall sensitivity of the open questionnaire was 87%, but specificity was 27% when compared with SIC. When examining individual items concerning work effect on symptoms, improvement of symptoms away from work at the weekend or on holiday, had sensitivities of 77% and 88% respectively although specificities were low at 44% and 24%. More recently Vandenplas et al. (2005) undertook structured interviews and examined the performance of individual items on diagnosis, and found that 'wheeze at work' particularly, in addition to being sensitive (88%) [written in what appears to be an error in the Vandenplas et al. paper as 40%], was also 85% specific for occupational asthma, and was the symptom most associated with a positive diagnosis (OR=3.8; 95% CI: 1.7 to 8.6). The item 'symptoms worse at work' was sensitive (90%) but not specific (9%), and sensitivity was lower for 'symptoms at work everyday' (55%) and for 'symptoms progressively over the week' (15%). 'Improvement of symptoms at weekends' had a sensitivity of 76% and specificity of 54%, 'improvement during holidays' a sensitivity of 74% and specificity of 57%, and when both questions were asked together, overall sensitivity and specificity were 75% and 55% respectively. In a small study of 5 latex-exposed nurses with occupational asthma, sensitivity of 'symptoms better on days away from work' had a sensitivity of 100% (Vandenplas et al., 1995). Most recently, using a self-administered occupational asthma screening-questionnaire, Pralong et al. (2013)



described optimum sensitivities of 70-90% for a variety of asthma symptoms worse at work, and a sensitivity of 79% for 'improvement of symptoms away from work'. However specificity was very low 13-28% for symptoms at-work, and 24% for improvement away from work.

Thus describing a work effect on asthma symptoms has a high sensitivity but low specificity for identifying occupational asthma. A number of guidelines advocate asking workers about the work effect on their asthma symptoms (Nicholson et al., 2010; Fishwick et al., 2012; Baur et al., 2012), although all recommend further tests to validate a diagnosis of occupational asthma. Furthermore these questions can miss those workers with poor symptom perception, or possibly those with symptoms that worsen at the end of the shift and persist at home (Burge, 1979).

### 1.3.3 Serial peak-expiratory flow measurements

Peak expiratory flow (PEF) is the maximum flow achieved during an expiration delivered with maximum force, starting from the point of maximum lung inflation (Quanjer et al., 1997). It is quick to perform, inexpensive, and non-invasive using a PEF meter or portable spirometer (Moscato et al., 1995). Serial PEF measurements are more helpful in the diagnosis of asthma since one-off lung function measurements are often normal at presentation; indeed at least four times a day readings are required to accurately reproduce diurnal variation (DV) of peak flow in asthmatics (Gannon et al., 1998; D'Alonzo et al., 1995). Serial PEF recording at work and away from work has long been proposed to investigate the relationship between work exposure and lung function (Burge et al., 1979a; Burge et al., 1979b). Malo et al. (1993) compared hand reading of PEFs by three experts with SIC for unselected agents and achieved good concordance with expert opinion (83%), and a sensitivity of 72% and specificity of 78% when 2-hourly measurements were used. Four times daily measurements were almost as good, with a concordance of 77%, sensitivity of 70% and specificity of 78%, and were considered to be acceptable to the worker. Less

frequent readings produced higher specificity but low sensitivity. Anees et al. (2004) showed that the minimum criteria for optimal sensitivity/specificity using computerized interpretation were at least four readings a day, at least three consecutive workdays in each work period and at least three work-rest-work or rest-work-rest complexes (approximately three weeks), showing a sensitivity of 78% and specificity of 92% when tested. When data were collected less frequently, the sensitivity and specificity fell to 64% and 83% respectively. Beach et al. (2005) have undertaken a systematic review and meta-analysis of studies measuring performance of PEF measurements against SIC, and provided pooled estimates of sensitivity and specificity of 64% and 77% respectively, where both sensitivity and specificity were available.

Acceptable PEF records are returned in at least 52-87% of workers who are asked to complete them (Nicholson et al., 2010; Moore et al., 2009a; Huggins et al., 2005; Sauni et al., 2009; Malo et al., 1995) and data quality is also better when specific occupational PEF charts containing prompts about shift times and exposures are used (Huggins et al., 2005).

UK guidelines on the management of asthma (BTS/SIGN, 2012) suggest that all attending physicians, including GPs, attending should consider the diagnosis of occupational asthma in adult patients with new- or recurrent-onset asthma symptoms; additionally those in high-risk occupations should have a diagnosis of occupational asthma positively excluded, recommending serial PEF measurements with expert (including software) analysis, and referral to a specialist.

#### 1.3.4 Peak-expiratory flow analysis

Burge (1989) observed that most PEF records for occupational asthma were viewed qualitatively and subjectively. Perrin et al. (1992) demonstrated a sensitivity and specificity of 81% and 74% respectively for qualitative ‘visual analysis’ of PEF readings compared with SIC. Qualitative analysis requires inter-observer agreement to be high, and a systematic

review by Nicholson et al. (2010) reported 80% concordance between expert assessors, with kappa values of at least 0.6 in most studies reviewed (Baldwin et al., Venables et al., 1984; Winck et al., 2001; Perrin et al., 1992), with the exception of 2 studies where inter-observer agreement was lower (Chiry et al., 2007: kappa=0.27 to 0.7; Girard et al., 2004: kappa=0.4 to 0.6).

A number of quantitative methods have been utilized. Hayati et al. (2006) applied Shewart control charts and found that a lower control limit PEF at work (LCL(W)) of <60% of the personal best was 86% sensitive and 88% specific for identifying occupational asthma compared with SIC. Other authors have used an arbitrary DV in PEF >20% on single days or prolonged periods at work (Smith et al., 1987; Smith et al., 1989; Cartier et al., 1989b). Liss and Tarlo (1991) investigated performance of DV in PEF on days at work and days away from work, and that found a  $\geq 20\%$  fall in PEF, which happened more frequently or with greater variation on days at work than on days away, had a sensitivity of 72% and specificity of 53% for identifying occupational asthma. By excluding those with  $\geq 20\%$  variation in PEF on one day only, the sensitivity and specificity increased to 93% and 77% respectively. In a further study by Hayati et al. (2008), which used the ratio of DV in PEF at work compared with DV in PEF away from work, they found that a ratio of >15% was 94% sensitive and 61% specific for identifying occupational asthma.

Cote et al. (1993) compared qualitative serial PEF analysis (by agreement between 3 physicians that work PEF was lower than rest PEF) with 3 quantitative methods: (1) variation in PEF between periods at work and away from work being outside the 95% confidence interval for 15 non-work-related asthmatics, (2) variation between maximum PEF at weekends (away from work) and minimum PEF on days at work against non-asthmatic PEF variation, and (3) DV in PEF greater on work days compared to rest days. When compared with SIC, the qualitative analysis had a sensitivity of 87% and specificity of 90%. Of the quantitative analyses, variation in maximum PEF at weekends and minimum PEF during

workdays was the only analysis that had higher sensitivity and specificity than qualitative analysis (93% and 90% respectively). Anees et al. (2011) found that difference in mean PEF between rest and work periods best separated workers with occupational asthma from those with non-occupational asthma and normal subjects, and using this index, a difference in PEF of >16 l/min had a sensitivity of 70% in diagnosing occupational asthma. An increase in DV on workdays of >7% over rest days had a sensitivity of only 27% for the diagnosis of occupational asthma.

### 1.3.5 Software analysis of peak-expiratory flow measurements

A computer software-based PEF analysis tool called OASYS-2 has been developed in Birmingham, UK to overcome inter-observer variation in qualitative (visual) analysis (Gannon et al., 1996). Each work-rest-work and rest-work-rest complex from PEF measurements taken on days at work and away from work, was assigned a visual score by an expert, and a linear discriminant analysis identified the patterns associated with a positive record. The scores were categorized into 4 groups (1-4), where 1=no occupational asthma, 2=possible, 3=probable and 4=definite occupational asthma. A mean work effect index (WEI) or “OASYS score” was calculated for the record. When tested against gold standard cases of occupational asthma (ie. diagnosed with either SIC or immunological demonstration of sensitization) a WEI of >2.5 showed a sensitivity of 75% and specificity of 94% (Gannon et al., 1996; Burge et al., 1999). Sensitivity and specificity became 78% and 92% respectively, once at least three complexes of data (approximately 3 weeks, 3 consecutive workdays in any work period) and at least four readings per day were obtained (Anees et al., 2004). In practice, the sensitivity was 79% in a group of workers with occupational asthma due to detergent enzymes (Moore et al., 2009a), despite many not having three consecutive days in each work period, a requirement for optimal sensitivity.

A newly developed ABC score (area between the curves for PEF, at work and away from work) subsequently showed a sensitivity of 72% and specificity of 100% when a score of

15L/min/hour was used; this was applicable to all types of shift work (Moore et al., 2012). Elsewhere authors have been more critical: Girard et al. (2004) achieved a sensitivity of 35% and specificity of 65% in identifying occupational asthma from a WEI of >2.5, and Chiry et al. (2007) were unable to distinguish occupational asthma (mean WEI=2.42+/-1.0) from work-exacerbated asthma (mean WEI=2.06 +/- 1.4; p=0.4).

A number of factors may interfere with interpretation by any method (Gannon and Burge, 1997; Jares et al., 2012). These include:

1. Intermittent exposure to suspect agents at work
2. Exposure to irritants
3. Irregular administration of medications
4. Irregular timing of the first reading after waking
5. Respiratory tract infection
6. Variable patient technique for PEF measurement
7. Falsification of readings
8. Patient compliance

Falsification of readings has been considered as a limiting factor for interpretation of PEF measurements performed on hand-held manual meters with a linear scale. Malo et al. (1995) reviewed paper and electronic PEF readings for 21 subjects undertaking investigation for occupational asthma. Reported values corresponded precisely to stored values in 52% of readings, and 71% were recorded within 1 hour of the solicited time.

Compliance was less satisfactory in workers referred by a compensation board. However, electronic logging meters utilizing coiled springs, ultrasound or rotary turbine technologies are now in common usage, with automatic checking for reproducibility and quality criteria (Miller et al., 2005; Quanjer et al., 1993).

### 1.3.6 Cross-shift changes in peak expiratory flow and FEV<sub>1</sub>

PEF and FEV<sub>1</sub> are closely correlated in asthma (Troyanov et al., 1994) and have been shown to be as effective as each other in identifying early asthmatic responses to occupational agents (Weytjens et al., 1999). In the workplace, cross shift comparisons are made by subtracting the daily post-shift FEV<sub>1</sub> or PEF reading, made 1 hour after work, from the pre-shift reading, made 1 hour before work. Cross-shift changes in PEF and FEV<sub>1</sub> have often been used in epidemiological and clinical studies, though early studies failed to show discriminating changes in FEV<sub>1</sub> (Gandevia and Ritchie, 1966; Gandevia 1963; Peters et al., 1968; Peters et al., 1970). A single study by Park et al. (2009) has compared cross-shift changes in PEF with SIC testing. A 5l/min cross-shift fall in PEF was found to be 91% specific for the diagnosis of occupational asthma, but with only 50% sensitivity; this was shown to be inferior to serial PEF monitoring when tested.

Normal subjects and asthmatics exhibit diurnal variation in airway calibre that is acrophasic between 1400 and 1800 (the response is similar in phase but exaggerated in asthmatics) (Hetzel and Clark, 1980). Therefore all workers on day shifts would experience co-incident increases in PEF across the shift, thus reducing the effect on PEF of an immediate asthmatic reaction. A cross-shift change would also fail to identify a late asthmatic reaction where symptoms may not become evident for many hours after the start of the shift, or occur after the end of the shift. Additionally workers' symptoms may not improve overnight and can worsen progressively throughout the working week, which can reduce pre-shift readings (Burge et al., 1979a; Burge et al. 1979b; Burge et al., 1979c).

### 1.3.7 Specific inhalation challenge testing

Specific inhalation challenge (SIC) to occupational allergens was first described by Pepys and co-workers (Pepys and Hutchcroft, 1975; Pepys et al., 1972) and was based on the work of Colldahl on inhalation tests in allergy (Colldahl, 1967). The rationale for SIC is to assess airway responsiveness to specific single sensitizing agents, as opposed to non-specific stimuli

such as methacholine, cold air and exercise, after control measurements have been made without exposure in the workplace, on separate days. It can be carried out in two main ways: a controlled laboratory challenge in which suspected sensitizers are administered in controlled concentration by either inhalation chamber or closed-circuit (Vandenplas and Malo, 1997), or by realistic challenge - an attempt to reproduce actual work processes and exposures (Pepys and Hutchcroft, 1975; Aasen et al., 2013). When the expertise to undertake a SIC is not available, the agent is not clear, or there is a negative SIC but high index of suspicion, an inhalational challenge in the workplace may be carried out under the supervision of a technician, by measuring lung function before and after usual workplace exposures (Rioux et al., 2008). In the UK SICs to occupational agents are undertaken in a small number of specialist occupational lung disease centres (Ortega et al., 2002; Barber et al., 2008), and recent European Respiratory Society guidelines have attempted to standardize the methodology between centres (Vandenplas et al., 2014).

A positive test is generally considered to be a fall in  $FEV_1$  of >15-20% from baseline on >1 measurement (within 10-30 minutes for immediate asthmatic reactions and >1 hour for late reactions) (Cartier et al., 1989b). HMW agents generally cause immediate or dual asthmatic reactions and are on the whole simpler to administer, and dose-response curves are easier to generate (Rioux et al., 2008; Jares et al., 2012). With LMW agents the reaction is often late, typical of the pattern originally described as a cornerstone of asthma by Herxheimer (1952), but it can be immediate, dual or atypical, and is therefore more difficult to predict (Jares et al., 2012; Rioux et al., 2008; Talini et al., 2011). Few studies have examined the criteria for a positive test. Stenton et al. (1994) proposed criteria for detecting late asthmatic reactions from SICs, which they stated were not an all-or-none phenomena and could be identified by statistical method, by calculating pooled standard deviations for hourly  $FEV_1$  on at least 3 control days without exposure. A positive test required at least 2 consecutive measurements below the lower confidence limit for  $FEV_1$  following an active challenge (Burge et al. (2009) also applied this method to unsupervised workplace serial PEF measurements, and found it to

have a sensitivity of 67% and specificity of 99% against other confirmed methods of sensitization).

Although considered to be the gold standard for diagnosis of sensitizer-induced occupational asthma (Nicholson et al., 2010; Fishwick et al., 2012, Baur et al., 2012), SIC carries a small risk (3%) of precipitating a severe asthmatic reaction (Vandenplas et al., 2013b) and has a number of limitations which need to be considered in its interpretation:

1. SIC has been reported to produce false negative results, when compared with a combination of expert opinion, suggestive serial peak flow measurements and immunological tests (Moscato et al., 1991; Cartier et al., 1989a; Burge et al., 1979, Lin et al., 1995),
2. Once a worker is removed from exposure SIC tests can become negative, and this has been demonstrated after 2-6 years away from exposure (Lemière et al., 2000a; Paggiaro et al., 1994),
3. It is often difficult to reproduce workplace conditions where exposures are complex, such as with welding fume or metalworking fluid, or where processes involve high temperatures, explosives or flammable substances (Aasen et al., 2013). New methodology using closed-circuit devices has allowed control of level of exposure and increased safety of the procedure (Caron et al., 2010; Malo et al., 2004),
4. SIC can only be performed in relatively stable workers ( $FEV_1$  >60-70 percent of predicted and/or >2L) (Cartier et al., 1989b),
5. False positive results have occurred when workers are given large doses of allergen, beyond the usual occupational exposures that cause non-specific irritant reactions (Beach, 2005; Vandenplas and Malo, 1997), or when a worker has severe non-specific bronchial reactivity (Burge, 1982; Malo et al., 2004).



### 1.3.8 Non-specific bronchial reactivity

Inhalation challenge with methacholine is a measure of non-specific bronchial reactivity (NSBR) that can be performed before and/or after a period of work exposure. Hyper-responsiveness has been defined as the provocative concentration or dose of histamine or methacholine required to elicit a pre-determined change in FEV<sub>1</sub>, usually a 20% percent decline (PC<sub>20</sub> and PD<sub>20</sub>, respectively) (Beach et al., 2005). Beach et al. performed a meta-analysis of 61 studies and reported the sensitivity and/or specificity of a single measurement of NSBR with SIC testing in the diagnosis of occupational asthma. In most cases, PC<sub>20</sub> values <16 mg/mL were considered to reflect significant NSBR, however, cut-off values of <8 mg/mL and <32 mg/mL were also reported. The pooled estimate of sensitivity was 67% (95% CI: 58 to 74%) and specificity was 64% (95% CI: 56 to 71%), whereas the pooled estimate for studies that reported only sensitivity was higher (n=13; 77%; 95% CI: 59 to 88%). 10 studies reported sensitivity and specificity of NSBR compared with SIC to HMW agents. The pooled estimates for sensitivity and specificity were 79% (95% CI: 68 to 88%) and 51% (95% CI: 35 to 67%) respectively. 2 studies suggested that the sensitivity of a single NSBR test could be improved by shortening the latency and measuring NSBR within hours of exposure (Perrin et al., 1992; Vandenplas et al., 2001). The authors acknowledged that definitions of a positive SIC varied between studies, and different protocols were used to carry out SIC. They also made no attempt to evaluate the differing methodologies. Subsequent studies have produced broadly similar results, with variable sensitivities when tested against different agents: all agents (96-98%; Maghni et al., 2004; Yacoub et al., 2007), isocyanates (76%; Piirila et al., 2008) and persulphates (57%; Moscato et al., 2005). Therefore a single measurement of NSBR has only a moderate sensitivity and specificity for the diagnosis of occupational asthma.

### 1.3.9 Paired measurements of non-specific bronchial reactivity

A number of studies have attempted to measure PD<sub>20</sub> while exposed at work, and away from work. Cote et al. (1990) demonstrated that a two-fold change in NSBR after work exposure

to Western red cedar had a sensitivity and specificity of 62% and 78% when compared to positive SICs to plicatic acid. Tarlo and Border (1991) compared NSBR to methacholine at work, and after at least 10 days away from work, with SIC test for mixed agents. Paired methacholine changes had a sensitivity of 56% and specificity of 83% in identifying occupational asthma. Perrin et al. (1992) tested sensitivity and specificity of a 3.2-fold change in NSBR at work and 2-weeks away from work (where 3.2 was the 95% CI for NSBR test reproducibility), and demonstrated a sensitivity of 48% and specificity of 64%; reducing the required change to two-fold increased sensitivity to 67%, but reduced specificity to 54%. Therefore paired NSBR measurements only have a moderate sensitivity and specificity for identifying occupational asthma, and some authors cited difficulties for the worker in enabling enough time off work (2 weeks) to perform the test as limitations of its use.

#### 1.3.10 Sputum eosinophilia

The normal cellular profiles of induced sputum have been described by Spanavello et al. (2000), who demonstrated that in healthy volunteers induced sputum was rich in macrophages (69.2 +/- 13% of cells) and neutrophils (27.3 +/- 13%), and poor in eosinophils (0.6 +/- 0.8%), lymphocytes (1.0 +/- 1.2%), and epithelial cells (1.5 +/- 1.8%). Asthma is characterized by airway eosinophilic inflammation, and marked sputum eosinophilia (>2.2% of non-squamous cells) occurs in most subjects with asthma (Pizzichini et al., 1997). The cellular characteristics of induced sputum in occupational asthma are less well described however. LMW agents are associated predominantly with late asthmatic reactions (Obata et al., 1999; Maestrelli et al., 1995; Maestrelli et al., 2009), which in turn are associated with increased number of airway eosinophils (Maestrelli et al., 2009). Lemièrè et al (1999) showed that workers had a significant increase in eosinophils while exposed to a variety of predominantly LMW agents at work, but not away from work, and eosinophilia resolved after 3-weeks away from work. Indeed Girard et al. (2004) have proposed an increase of 1% in sputum eosinophils with work exposure as diagnostic for occupational asthma, but this only showed a sensitivity of 65% and specificity of 76%, and increasing the cut-off to 2% further

reduced sensitivity. Furthermore, Anees et al. (2002) showed that the majority of cases of occupational asthma due to LMW agents are of the non-eosinophilic (predominantly neutrophilic) variant, and only 14/38 (37%) of cases demonstrated sputum eosinophilia (>2.2%) while exposed at work. Indeed other studies have shown that some workers with occupational asthma have normal sputum eosinophil counts prior to SIC testing (Lemière et al., 2001, Obata et al., 1999).

Most studies that have investigated sputum eosinophilia after SIC have shown a median increase in percentage eosinophil levels after positive challenges with HMW and LMW agents (Lemière et al, 2000b; Lemière et al., 2001; Obata et al., 1999; Krakowiak et al., 2005; Alvarez et al., 2001). In the study by Lemière et al. (2001) the best cut-off for predicting a 20% fall in FEV<sub>1</sub> was an increase in sputum eosinophils of  $\geq 0.26 \times 10^6/\text{mL}$  compared with baseline values (sensitivity 82%; specificity 91.7%); sensitivity was increased to 96% when combined with a two-fold increase in NSBR. No accurate pooled estimates of sensitivity and specificity of sputum eosinophil counts have been made, and availability and resources required has limited their use to date.

#### 1.3.11 Fractional exhaled nitric oxide

Fractional exhaled nitric oxide (FE<sub>NO</sub>) is a simple non-invasive method of assessing airway inflammation (Dweik et al., 2011; Kharitonov et al., 1994), which correlates well with sputum eosinophilia (Jatakanon et al., 1998; Franklin et al., 2004; Langley et al., 2002). However, FE<sub>NO</sub> depends upon several other factors including inhaled corticosteroid (ICS) use, smoking, respiratory tract infections, atopy and height. ICS use results in a fall in FE<sub>NO</sub> levels in mild asthmatics (Kharitonov et al., 1996, Jatakanon et al., 1999; Silkoff et al., 2001), smoking lowers FE<sub>NO</sub> (Bommarito et al., 2008; Verleden et al., 1999) and respiratory tract infections raise FE<sub>NO</sub> (Kharitonov et al., 1995a; Proud 2005). Atopics have increased levels compared to non-atopics (Franklin et al., 2004; Ho et al., 2000; Olin et al., 2006).

Recent guidelines recommend the application of FE<sub>NO</sub> for monitoring airway inflammation in asthmatics and to support a diagnosis of asthma where further objective evidence is required (Dweik et al., 2011). Abnormal baseline FE<sub>NO</sub> measurements are considered to be  $\geq 22$ ppb in never- or ex-smokers and  $\geq 14$ ppb in active smokers (Moore et al., 2010; Anees et al., 2002), a level equivalent with a raised sputum eosinophil count of  $\geq 2.2\%$ . FE<sub>NO</sub> is in use in UK primary care practice as a complementary diagnostic tool in asthma, and as an aid to initiating or titrating asthma treatments (Price et al., 2013), but is currently not recommended as part of screening for occupational asthma (Fishwick et al., 2012).

The role of FE<sub>NO</sub> in occupational challenge testing remains unproven, though several prospective studies have investigated FE<sub>NO</sub> changes after SIC in this setting. Clinically relevant increases in FE<sub>NO</sub> have been seen 24-hours after positive SIC (Piipari et al., 2002; Baur and Barbinova, 2005) and also in symptomatic patients with negative SICs (Barbinova and Baur, 2006). Pedrosa et al. (2012) showed a significant 14ppb FE<sub>NO</sub> change 24-hours after positive SICs (n=21), when compared to negative SICs (n=13), and receiver-operator curve analysis achieved a sensitivity of 81% and specificity of 92% in identifying a positive result, from a 12% increase in FE<sub>NO</sub>. 3 other studies have demonstrated an increase in FE<sub>NO</sub> 24 hours after exposure to high and low molecular weight occupational agents, which correlated well with sputum eosinophil measurements (Lemière et al., 2010; Ferrazzoni et al., 2009; Swierczynska-Machura et al., 2008). However 2 studies reported that the predictive capacity of FE<sub>NO</sub> change after SIC was low (Walters et al., 2014; Sastre et al., 2013). FE<sub>NO</sub> remains elevated up to 72 hours after challenge and the maximal FE<sub>NO</sub> response is around 10-hours post-exposure (Kharitonov et al., 1995b; Paredi et al., 1999); studies in the occupational setting have used post-exposure measurements at 20-24 hours. The main limitation on most studies is the relatively small number of SICs available for analysis.

### 1.3.12 Specific IgE to occupational agents

Asthma is classically thought of as an IgE-mediated disease that is prevalent in atopic individuals (Beach, 2005). Therefore skin-prick allergy tests (SPTs) and serological measurements of specific-IgE are in common usage in supporting a diagnosis of asthma. Specific IgE is measured using either the Radioallergoabsorbant test (RAST) or Enzyme Linked ImmunoSorbent Assay (ELISA) (Goodwin and How, 1976; Glovsky, 2007), which provide a graded marker of the degree of sensitization, and are used to monitor response to exposure over time. A positive response to SPT is defined as a 3mm or greater wheal reaction to an allergen (Dreborg, 1991; Beach, 2005); this may be to common aeroallergens to signify atopy, or to specific occupational allergens where there is suspicion of sensitization.

In occupational asthma induced by HMW agents, specific-IgE is usually present. Several occupational allergens are commercially available for skin and serological testing and their sensitivity and specificity against SIC have been established. These include natural rubber latex in healthcare workers (Vandenplas et al., 2001) wheat and rye flours in bakers (Van Kampen et al., 2008), animal epithelium in farmers (Koskela et al., 2003), and egg protein in laboratory workers (Jones et al., 2013). Beach (2005) has undertaken a meta-analysis with pooled estimates of sensitivity and specificity for SPTs and serum-specific IgE against SIC. For SPTs to HMW agents, pooled sensitivity was 81% and specificity was 60%, from a total of 26 studies (9 of those reporting sensitivity only); for specific IgE the pooled sensitivity was 74% and specificity was 79%, from 9 studies reporting both.

Occupational asthma induced by LMW agents is generally not specific IgE-mediated, as LMW agents are too small to elicit an IgE-mediated response, and act as allergens by conjugation with carrier protein (haptens). Consequently SPTs or measurements of specific IgE in this situation are not usually helpful, and importantly, there are few immunological tests available for the majority of LMW agents. Some studies have assessed the performance of SPTs and specific-IgE for LMW agents with SIC however: for example, toluene

diisocyanate (TDI), MDI and HDI (Pezzini et al., 1984; Tee et al., 1998; Budnik et al., 2013), vinyl sulphone reactive dyes (Park et al., 2001), platinum salts (Merget et al., 1991) and anhydride dusts (Grammer et al., 1998; Baur and Czippon, 1995). Beach (2005) demonstrated a pooled sensitivity of 73% and specificity of 86% for SPTs to LMW agents from 5 studies, and a sensitivity 31% and specificity of 89% for specific IgE, from 11 studies.

The presence of specific IgE confirms sensitisation but does not necessarily indicate the cause of occupational asthma, as sensitization can exist in the absence of occupational asthma, ie. its specificity is low. The absence of commercially available allergens limits its use, particularly for LMW agents. Also it is of no value in the diagnosis of irritant induced asthma or work-aggravated asthma.

#### 1.3.13 Occupational health surveillance

The aim of health surveillance at work is to identify work-related disease at either pre-symptomatic or early symptomatic stage, with the intention of improving health outcomes for affected workers (Nicholson et al., 2010). Methods used in occupational asthma health surveillance are respiratory questionnaires, serial spirometry measurements, and detection of sensitization to a particular agent by either skin prick allergy tests or serum-specific IgE (Brant et al., 2005b, Gordon et al., 1997, Stenton et al. 1993). There are no studies comparing the effectiveness of one component of health surveillance with another or randomized controlled trials of the effectiveness of health surveillance in practice. However, a comparison between a health surveillance programme (using questionnaire and specific IgE to flour and fungal amylase) and an independent cross-sectional survey of employees was undertaken by Brant et al. (2005b) in supermarket bakery workers. One percent of bakery workers had work-related symptoms and specific IgE, compared with 4% of employees from the survey; thus surveillance was judged to have underestimated the burden of occupational asthma. Mackie et al., (2008) described a mean delay of 9 months between the detection of occupational asthma symptoms at health surveillance, and diagnosis; this contrasted with a

mean of delay of 4 years in a study of diagnostic delay in a UK multi-centre tertiary clinic population, where 86% of participants were not afforded health surveillance (Fishwick et al., 2007). In Ontario, Canada, where a health surveillance programme for isocyanate-exposed workers was introduced in 1983, a comparison was made between cases of occupational asthma due to isocyanates and cases due to other agents where no such programme existed (Tarlo et al., 2002). There was a shorter diagnostic latency (2.4 versus 3.4 years) and fewer hospital admissions in cases due to isocyanates when compared with non-isocyanate occupational asthma. However, there is potential for confounding in this study, as no attempt was made to account for the effects of engineering controls or industrial hygiene measures (ie. reduction in isocyanate exposure). Therefore there is some evidence that health surveillance for occupational asthma can shorten diagnostic latency and improve health outcomes, though health surveillance programmes have not been subjected to controlled trials.

#### 1.4 Outcomes of occupational asthma

##### 1.4.1. Health economics

The burden of disease of occupational asthma is associated with significant societal costs. Ayres et al. (2011) performed a desktop study using cost-of-illness method to define direct and indirect lifetime costs of occupational asthma for six scenarios - a male and a female worker each exposed to: isocyanates, latex and glutaraldehyde, or flour. 2003 SWORD incidence data were then used to calculate costs for 209 cases of occupational asthma. The cumulative lifetime costs were estimated to be £25.3-27.3 million (2004 prices), and when extrapolated for all causes in the UK in 2003, this came to £71.8-100.1 million. Using this model each subsequent year would see a new cohort of newly diagnosed occupational asthmatics identified and added, each costing up to £100 million per annum. There was a marked gender difference, with the costs for male workers twice that of female workers, reflecting the number of men exposed to common causative agents in the UK, as well as their higher income in at-risk occupations. About 49% of these costs were borne by the individual

worker, 48% by the state and 3% by the employer (although this estimate did not include costs of workplace modification, litigation or surveillance programmes). As the economic burden appeared to fall on the state and individual, Ayres et al. concluded that there is currently little incentive for the employer to act.

In the United States the total annual costs of occupational asthma have been estimated at \$1.6 billion (Leigh et al., 2002), comprising \$1.2 direct costs and \$0.4 billion indirect costs. To calculate costs Leigh et al. used the human capital method, which splits costs into direct categories such as medical expenses, as well as indirect categories such as lost earnings and lost home production. They concluded that the majority of costs are born by the taxpayer (through Medicare) and the individual worker and their families, with a minority through contributions to workers' compensation premiums.

#### 1.4.2. Socio-economic outcomes for the worker

A number of studies have followed-up workers diagnosed with occupational asthma and have noted that roughly one-third of workers (range 25-44%) were unemployed at 1-6 years after diagnosis (Gannon et al., 1993; Venables et al., 1989b; Ross et al., 1998; Vandenplas et al., 2002; Moscato et al., 1999; Ameille et al., 1997; Larbanois et al., 2002). Goe et al. (2004) found that workers with occupational asthma were twice as likely to have left their employer than those with work-exacerbated asthma (47% v 23%), though other authors found no significant difference in employment status at follow-up between the same groups (Cannon et al., 1995; Larbanois et al., 2002). Dimich-Ward et al. (2007) followed up 185 Western red cedar workers with occupational asthma who had remained employed after diagnosis, and found that the majority of subjects who were relocated to unexposed jobs within 1 year of diagnosis remained in their employment (74%) (with none quitting due to asthma), compared with 17% of workers who remained exposed leaving their employment because of their asthma.



62-85% of workers who leave employment (to either become unemployed or find alternative work) report suffering some loss of income (Vandenplas et al., 2002; Ameille et al., 1997; Moscato et al., 1999; Larbanois et al., 2002), and loss of employment has been associated with loss of income of 22-50% of baseline income (Moscato et al., 1999; Ameille et al., 1997; Larbanois et al., 2002), all significantly greater than if the workers remained exposed or were relocated within the same company. Financial loss may be greater for those with occupational asthma than those with non-occupational asthma (Santos et al., 2007); certainly workers with occupational asthma find employment more difficult to come by than non-occupational asthmatics (Cannon et al., 1995; Larbanois et al., 2002).

#### 1.4.3 Health outcomes for the worker

Patients with occupational asthma are significantly more likely to report poor physical activity, and poor physical and mental health, than patients with non-occupational asthma (Knoeller et al., 2013). The likelihood of improvement or of resolution of symptoms and lung function is greater in workers who avoid ongoing exposure to the causative agent (Nicholson et al., 2010; Tarlo et al., 2008; Beach et al., 2008; Chan-Yeung et al., 1982; Moscato et al., 1999), and continued exposure after diagnosis has been associated with a worsening of symptoms (Malo and Chan-Yeung, 2001; Ross and McDonald, 1998; Moscato et al., 1999) and lung function ( $FEV_1$ ) (Anees et al., 2006), even when workers are more heavily medicated (Marabini et al., 1993). Rachiotis et al. (2006) have undertaken a systematic review of 39 studies that evaluated the symptomatic and functional outcomes of occupational asthma after avoidance of exposures to HMW and LMW agents. Reported rates of symptomatic recovery varied from 0-100%, with a pooled estimate of 32% (95% CI: 26 to 38). These rates were lower with increased age and amongst clinic based populations. Patients with the shortest durations of employment (up to 76 months) had the highest rate of recovery (36%; 95% CI: 25 to 50), but this pattern was not sustained over longer durations; complete recovery was less common in those studies with an average duration of *symptomatic* exposure >26 months when compared to <26 months, though this was not statistically significant. In

most cases NSBR detected at diagnosis persisted and the pooled estimate of persistent NSBR was 73% (95% CI: 66 to 79). Patients with occupational asthma due to LMW agents were less likely to have persistent NSBR than those due to HMW agents, when age was accounted for. In between-study comparisons, the authors found no clear patterns of recovery by duration of follow-up, by a crude index of study nationality, or when comparing disease attributed to LMW or HMW agents. The majority of included studies recruited patients from specialist clinics, where there is considerable potential for bias, since patients with more severe disease may be referred more often for specialist assessment. There was also an absence of any standardized methodology for follow-up studies, and the quality of the included studies was not assessed formally.

The likelihood of resolution or improvement of symptoms is greater in workers who have had relatively normal lung function at diagnosis, and shorter duration of symptoms prior to diagnosis or removal from exposure, as seen for occupational asthma induced by isocyanates (Pisati et al., 1993; Pisati et al., 2007; Rosenberg et al., 1987; Park and Nahm, 1997; Piirila et al., 2000; Padoan et al., 2003), Western red cedar (Chan-Yeung et al., 1982), crab (Hudson et al., 1985) and reactive dyes (Park et al., 2006; Park et al., 2007). Descatha et al. (2007) looked at diagnosis of a variety of causal agents and found that the severity of occupational asthma at diagnosis was associated with the duration of symptoms before diagnosis, but not with the type of causal agent.

Reduction of exposure to sensitizing agents causing occupational asthma has been proposed as an alternative to total avoidance in order to minimize the adverse socio-economic impact of the condition. Vandenplas et al. (2011) performed a systematic review of 14 studies examining the symptomatic and physiological outcomes of workers with occupational asthma after reduction or cessation of exposure to the causal agents. The meta-analysis of pooled data showed that a reduction of exposure was associated with a lower likelihood of improvement (OR=0.16, 95% CI: 0.03 to 0.91) or resolution of asthma symptoms (OR=0.30, 95% CI: 0.11

to 0.84), and a higher risk of worsening symptoms (OR=10.23, 95% CI: 2.97 to 35.28) and more severe NSBR (OR=5.65, 95% CI: 1.11 to 28.82), compared with complete avoidance of exposure. There was potential for selection bias, since all studies were observational and the rationale for the intervention decision (reduction or avoidance of exposure) was largely unknown. In most studies involving LMW agents the proportion of workers who avoided exposure was 2.3 to 3.2-fold higher than those who reduced exposure. Paradoxically, reduced exposure was 1.2 to 5.0-fold higher than avoidance for workers with occupational asthma due to latex (Vandenplas et al., 2002) and platinum (Merget et al., 1999).

### 1.5 Conclusion

The burden of occupational asthma in the UK is substantial, with 1 in 6 cases of new-onset or reactivated adult asthma being caused by an agent at work. This currently costs the UK £1.1 billion each decade, with most of this cost borne by the taxpayer and the individual worker. As industries change and work practices evolve, new asthmagens are identified and certain hazardous exposures become more prevalent than others. In the past 3 decades there have been significant advances in the understanding of the patho-physiology of occupational asthma and in validated tools for screening and diagnosis, which have aided the management of the worker with asthma symptoms, although much work is still to be done on primary prevention and early detection of cases.

Early diagnosis is the key to good prognosis and can prevent avoidable cost to the patient in terms of ill health, work-loss and unemployment; consequently there is considerable benefit to workers, to the health service and to society in making an early diagnosis of occupational asthma. However occupational asthma is not well recognized by healthcare professionals in the UK, particularly in primary care populations where there appear to be as many undiagnosed cases of occupational asthma as diagnosed cases, and workers see their GP many times before referral to a specialist occurs. In order to address this problem, a clear understanding of the behaviour of workers with occupational asthma symptoms, and the

barriers to diagnosis for the healthcare professional, is required.

## CHAPTER 2: AIMS AND OBJECTIVES

### 2.1 Background

It is clear that occupational asthma is not well recognized by healthcare professionals, and that in addition to those workers who go undiagnosed, there is significant delay in diagnosis for many workers. This delay exists in spite of the availability of specialist care, validated screening and diagnostic tests, and occupational asthma guidelines for all healthcare professionals who attend working-age asthmatics. Under- and delayed diagnosis are associated with considerable health and financial costs to individual workers, who bear a significant proportion of those costs themselves. Therefore there is an urgent need to improve recognition of occupational asthma and reduce the delay in diagnosis in the UK. Data from the UK, Europe or North America concerning barriers to diagnosis for workers or healthcare professionals are sparse, and are currently limited to closed-question responses from cross-sectional studies. There have been no studies from the UK exploring barriers to the diagnosis of occupational asthma in detail.

### 2.2 Overall aim of the research

The over-arching aim of this research is to identify the barriers to diagnosis of occupational asthma on the part of the worker and of the healthcare professional, in order to determine a strategy for improving the diagnosis of occupational asthma in the UK.

### 2.3 Hypotheses

1. That poor recognition of occupational asthma in primary care is because primary healthcare professionals do not follow UK guidance for occupational asthma by asking working-age asthmatics about their work or the effect of work on their symptoms.

2. That delay in diagnosis of occupational asthma is partly due to multiple societal factors affecting whether an individual worker will disclose his or her occupational asthma symptoms to a healthcare professional or employer.
3. That healthcare professionals lack knowledge and experience in managing cases of occupational asthma, which prevents them from screening and diagnosing occupational asthma in working-age adults.
4. That there are a number of motivating factors for healthcare professionals (for example: perceived importance, resource constraints) which predispose to lack of consideration of the diagnosis of occupational asthma in adults.

#### 2.4 Specific Objectives

1. To evaluate current practice in assessing working-age asthmatics for occupational asthma in a primary care population, using UK guidelines for occupational asthma as a reference standard (Fishwick et al., 2012).

*Study design:* An audit of current practice in primary care practices in Birmingham, UK.

2. To explore and define the beliefs that influence health-seeking behaviour in workers with symptoms of occupational asthma, and to establish a framework for understanding how these factors lead to delays in diagnosis.

*Study design:* An inductive, phenomenological qualitative study of workers with occupational asthma, at a specialist occupational lung disease clinic in Birmingham, UK, generating data through semi-structured interviews.

3. To define the important barriers that prevent healthcare professionals from identifying cases of occupational asthma, and to see whether these differ between healthcare professions and medical specialties.

*Study design:* A cross-sectional questionnaire, distributed by e-mail to UK clinicians in the UK attending working-age asthmatic patients as part of their practice.

4. To evaluate the feasibility of initiating an electronic screening tool for primary healthcare professionals, its utility in collecting data on work-effect, and the willingness of individual healthcare workers to use it.

*Study design:* A prospective feasibility study in primary care practices in Birmingham, UK.

## CHAPTER 3: ASSESSMENT FOR OCCUPATIONAL ASTHMA IN PRIMARY CARE

### 3.1 Introduction

Occupational asthma is common, accounting for 1 in 6 cases of new-onset or reactivated adult asthma in the UK (Toren and Blanc, 2009; Nicholson et al., 2010; Baur et al., 2012).

However it is under-reported to surveillance schemes, and often goes unrecognized, particularly in primary care (de Bono and Hudsmith, 1999; Hoyle et al., 2010), where mean delays in diagnosis of 4 years have been described (Fishwick et al., 2007; Santos et al., 2007; Poonai et al., 2005). This is notwithstanding the publication of guidance for physicians, GPs and primary care nurses in the UK on the assessment and management of patients with suspected occupational asthma (Fishwick et al., 2012; Nicholson et al., 2010; BOHRF, 2010a; BOHRF, 2010b). These suggest that all working-age patients with new onset or re-activated asthma symptoms should be asked (1) about their occupation in order to highlight high-risk work, and (2) two simple work-effect questions (“Are your symptoms better on days away from work?” and “Are your symptoms better away from work on holiday?”) which have a high sensitivity (74-100%), but low specificity (24-57%) for identifying occupational asthma (Malo et al., 1991; Vandenplas et al., 1995; Vandenplas et al., 2005; Pralong et al., 2013b). Any worker with a work-effect on asthma symptoms should be referred to an occupational lung disease specialist (Fishwick et al., 2012).

In the UK occupational asthma is associated with a societal cost of £1.1 billion each decade due to poor health and employment outcomes; this is partially avoidable with early diagnosis and subsequent avoidance of exposure to causative agents. In order to remedy the delay in diagnosis, it is important to understand how working-age asthmatics (the ‘at-risk’ population) are currently assessed and managed in primary care. The hypothesis is that one important reason for delayed diagnosis may be a lack of enquiry about occupation, and the effect of work on asthma symptoms, by the primary healthcare worker (HCW).



### 3.2 Aim

The aim of this study was to evaluate current practice in assessing working-age asthmatics for occupational asthma in a primary care population.

### 3.3 Methods

#### 3.3.1 Study design

A case-note audit was performed at 4 National Institute for Health Research (NIHR) Primary Care Research Network (PCRN) practices in Birmingham, UK.

#### 3.3.2 Practice recruitment

Practices were recruited by the primary author (GW), through direct contact with the NIHR research nurse at each practice.

#### 3.3.3 Practice-level data

Practice-level data on list size, gender, number of working-age patients (age-band=16-64), number of patients with a Read-code of 'asthma' and 'occupational asthma' (Health and Social Care Information Centre, 2013), were gathered by searching the practice databases (Egton Medical Information Systems Ltd., Yeadon, Leeds, UK). Neighbourhood employment statistics were sought from Census data (ONS, 2011) and practice Index of Multiple Deprivation (IMD) scores and Townsend Index of deprivation scores were also obtained (Department for Communities and Local Government, 2007; Townsend et al., 1988).

#### 3.3.4 Sample data

A sample of 100 working-age adults (age 16-64) with a Read-code diagnosis of "asthma" was taken from each practice. All working-age adults were listed by their registration number (unrelated to age or date of registration) and records were sampled by taking separate blocks of 10 adjacent numbers. Using individual patients' electronic records, the following data

were collected where available: (i) demographics (age, gender, reported co-existing airways disease), (ii) date and age at onset of asthma (adult-onset ( $\geq 16$  year-old), prevalent childhood, reactivated childhood), (iii) date of last two asthma-reviews (urgent or routine), (iv) occupation, (v) work-effect (documented at all, at diagnosis, or within last two asthma-reviews), (vi) lung function (peak-expiratory flow or spirometry). Age was compared between samples by single-factor ANOVA, and the proportion of males in each sample was compared to the proportion of males from each practice population, by Chi-squared test with Yates' correction.

### 3.4 Results

#### 3.4.1 Practice-level data

The sample population was 27295 patients in total from 4 practices, and mean practice-list size was 6824 (SD+/-3846) of which 64% were of working age (Table 3.1). Practices used either the Telnet (EMIS LV 5.2) or web-based (EMIS Windows 3.1.7.11) versions of the EMIS electronic database. The prevalence of asthma in working-age adults based on Read code was 12% (range 8-15%) of which 47% were male, and the prevalence of occupational asthma in working-age asthmatics was 0.3% (range 0-0.8%). All 4 practices were situated in electoral wards where the most frequently reported industries of employment are associated with an increased risk of occupational asthma (detailed in Table 3.2). Additionally all practices were located in the top 36% most deprived neighbourhoods in England, based on IMD ranking, and in the 5<sup>th</sup> quintile for Townsend Index of deprivation scores.

	<b>Practice 1</b>	<b>Practice 2</b>	<b>Practice 3</b>	<b>Practice 4</b>
Practice list size	7142	5581	11885	2687
Electronic database used	EMIS LV 5.2	EMIS LV 5.2	EMIS Windows 3.1.7.11	EMIS LV 5.2
Number of patients of working-age <sup>1</sup> <i>n (%)</i>	4452 (62)	3767 (68)	7599 (64)	1746 (65)
Prevalence of asthma <sup>2</sup> in working-age adults; number (%)	609 (14)	575 (15)	601 (8)	260 (15)
Number of male working-age patients with asthma <i>n (%)</i>	300 (49)	265 (46)	261 (44)	136 (52)
Prevalence of occupational asthma <sup>3</sup> in working-age asthmatics (%)	0.2	0	0.8	0

**Table 3.1. Summary data of patients with asthma and occupational asthma, by practice.**  
**EMIS=Egton Medical Information Systems. <sup>1</sup>16-64 years of age; <sup>2</sup>patients who have a Read-coded diagnosis of “asthma”; <sup>3</sup>patients who have a Read-coded diagnosis of “occupational asthma”.**

	Practice 1	Practice 2	Practice 3	Practice 4
Electoral ward	Billesley	Bournville	Aston	King's Norton
Most frequently occurring industries of employment <sup>1</sup>	<ul style="list-style-type: none"> <li>• Manufacturing</li> <li>• Wholesale/retail trade</li> </ul>	<ul style="list-style-type: none"> <li>• Health and social work</li> <li>• Education</li> <li>• Real estate/business</li> </ul>	<ul style="list-style-type: none"> <li>• Manufacturing</li> <li>• Wholesale/retail trade</li> </ul>	<ul style="list-style-type: none"> <li>• Manufacturing</li> <li>• Wholesale/retail trade</li> <li>• Health and social care</li> </ul>
Proportion of local population in skilled trades <sup>2</sup> (%)	14	9	12	13
Proportion of local population in plant / machine operations <sup>3</sup> (%)	11	9	15	12
Weekly Household Total Gross Income <sup>4</sup> (£/week)	410	470	280	400
Townsend Index score <sup>5</sup> (quintile)	3.8 (5 <sup>th</sup> )	2.3 (5 <sup>th</sup> )	16.3 (5 <sup>th</sup> )	5.0 (5 <sup>th</sup> )
IMD score <sup>6</sup>	2874	4741	1009	11731
IMD score rank (% top most deprived in England)	9	15	3	36

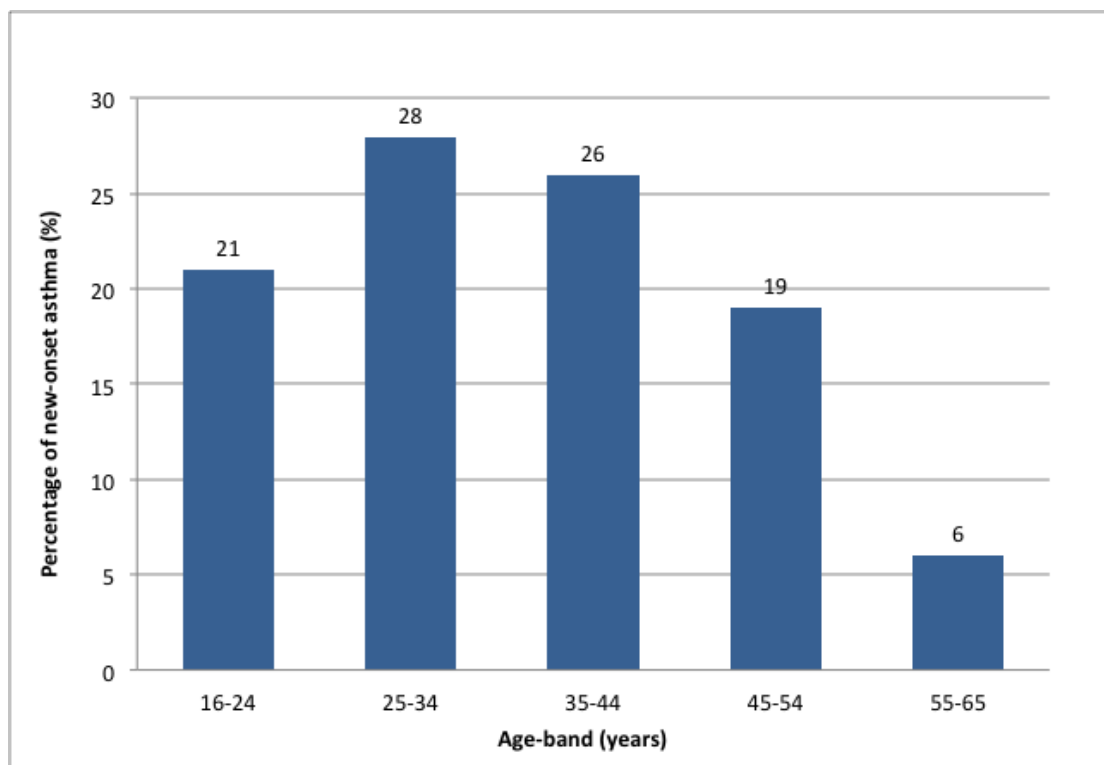
**Table 3.2. Summary of socio-economic data by practice and electoral ward.**

**IMD=Index of Multiple Deprivation. <sup>1</sup>Data taken from ONS, 2011. <sup>2</sup>Average for England is 12%; <sup>3</sup>average for England is 8%; <sup>4</sup>average for West Midlands is £470 per week (ONS, 2011). <sup>5</sup>Townsend Index is based on electoral ward and <sup>6</sup>IMD is based on the postcode where practice is located (Department for Communities and Local Government, 2007).**

### 3.4.2 Sample data

There were 396 working-age asthmatics in the sample (Table 3.3; 96-100 per-practice; 49% male; mean-age 39 (SD+/-14). Single factor ANOVA test revealed no significant differences between the 4 samples on the basis of age ( $p=0.06$ ). Additionally there were no significant differences between the proportion of males in each sample and its parent practice population of working-age asthmatics (Practice 1: 59% audit sample versus 49% practice population ( $p=0.09$ ;  $\chi^2=2.9$ ); Practice 2: 45% versus 46% ( $p=0.9$ ;  $\chi^2=0.02$ ); Practice 3: 42% versus 44%

( $p=0.87$ ;  $\chi^2=0.03$ ); Practice 4: 49% versus 52% ( $p=0.66$ ;  $\chi^2=0.2$ ). There were no Read-coded cases of “occupational asthma” (one non-coded case of diagnosed occupational asthma was identified during data-gathering). 18 patients (2%) had co-existing diagnoses of additional airways diseases (COPD or bronchiectasis). The onset of asthma in 234 patients (59%) was during working-age ( $\geq 16$  years old), 95% having new-onset rather than reactivated childhood asthma. Onset of asthma was most likely to occur between 25 and 34 years of age (66 cases; 28%) and least likely between 55 and 65 years of age (14 cases; 6%); see Figure 3.1.



**Figure 3.1.** All adult-onset asthma cases from the sample, by age at onset (n=234).

### 3.4.3 Recording occupation and work-effect

Occupation was recorded in 55 out of 396 (14%) cases; of these 2 out of 55 were in an asthma review template, 17 out of 55 in free-text, and 36 were found incidentally, elsewhere in the patient record. 13 out of 55 (24%) patients were both in high-risk occupations for occupational asthma and experienced the onset of asthma in adult life (Table 3.3).

		Practice 1	Practice 2 <sup>1</sup>	Practice 3	Practice 4	Total
Number of working-age asthmatics in sample		100	96	100	100	<b>396</b>
Number of male patients <i>n</i> ( %)		59 (59)	43 (45)	42 (42)	49 (49)	<b>193 (49)</b>
Mean age (+/-SD)		36 (15)	40 (14)	41 (12)	37 (15)	<b>39 (14)</b>
Co-existing COPD / bronchiectasis		7 / 2	2 / 0	6 / 0	3 / 0	<b>18 / 2</b>
Onset of asthma	Number with adult-onset of asthma <sup>2</sup> <i>n</i> ( % of sample)	48 (48)	56 (58)	76 (76)	54 (54)	<b>234 (59)</b>
	Number with new-onset, rather than reactivated childhood asthma <i>n</i> ( % of adult-onset asthmatics)	44 (92)	53 (95)	74 (97)	52 (96)	<b>223 (95)</b>
Occupational enquiry	Occupation recorded <i>n</i> ( %)	6 (6)	20 (21)	11 (11)	18 (18)	<b>55 (14)</b>
	Occupation recorded at an asthma review <i>n</i> ( %)	5 (5)	6 (6)	7 (7)	1 (1)	<b>19 (5)</b>
	Number in high-risk occupations and with adult-onset asthma	1 • Automotive assembly	4 • Soldering • Hairdressing • Glassware manufacturing • Animal husbandry	5 • HCW x3 • Cleaning • Food processing	3 • Welding • HCW • Hairdressing	<b>13</b>
Work-effect enquiry	At diagnosis <sup>3</sup> <i>n</i> ( %)	3/26 (12)	0/22 (0)	1/47 (2)	0/22 (0)	<b>4/117 (3)</b>
	At most recent two asthma-reviews <sup>4</sup> <i>n</i> ( % of those having asthma-reviews)	2/72 (3)	3/66 (5)	1/92 (1)	0/71 (0)	<b>6/301 (2)</b>

**Table 3.3. Summary data of working-age asthmatics by practice sample. HCW=health care worker. <sup>1</sup>4 patients from the sample were incorrectly coded as being asthmatic, thus n=96 rather than 100; <sup>2</sup>age 16 or older; <sup>3</sup>only 117/396 patients had visible records at diagnosis (otherwise (i) the diagnosis was in childhood, (ii) the patient had registered with practice after diagnosis, or (iii) the diagnosis was made before the introduction of electronic patient records); <sup>4</sup>301/396 patients had undertaken at least two asthma-reviews.**

In 301 out of 396 (76%) patients, at least two asthma reviews had been carried out, either by a nurse or a GP, and from documentation of the most recent attendance, 27% were urgent appointments and 73% were routine asthma reviews. In 9 out of 396 (2%) patients, work-effect was recorded, 7 out of 9 at the time when occupation was documented. 117 records had information available since the date of diagnosis, at which point only 4 (3%) had had a work-effect enquiry. In 6 out of 301 (2%), work-effect was enquired about at one of the two most recent asthma reviews, of which 3 had positive responses for which no demonstrable action was taken. 303 out of 396 (77%) patients had single peak-expiratory flow measurements, and 70 out of 396 (18%) patients had spirometry measurement results documented since diagnosis. 39 (10%) patients had been Read-coded as 'asthma-resolved'.

### 3.5 Discussion

#### 3.5.1 Summary

This study showed that the recorded prevalence of asthma in working-age adults (age 16-64) was 12% (8-15%) and the recorded prevalence of occupational asthma in working-age asthmatics was 0.3% (0-0.8%). Occupation was recorded in only 55 out of 396 (14%) working-age asthmatics, and 13 out of those 55 were in high-risk occupations and had experienced the onset of asthma during working-age. Only 9 out of 396 (2%) had any enquiry about work-effect on their asthma symptoms.

#### 3.5.2 Limitations

This was a retrospective study, which is an appropriate study design for evaluating current and historical clinical practice. It should be acknowledged that the sample size in this study was small with only 4 practices recruited, although these were broadly representative of UK practice populations. Practices of varying size were recruited with a mean list size of 6824 (range: 2687-11885); this equates to a mean list size in England of 6487 (Health and Social Care Information Centre, 2008). 64% of registered patients at the recruited practices were of working age, which is comparable to the national estimate of 62% for the proportion of

working-age adults registered in primary care (Office for National Statistics, 2010). The point prevalence of Read-coded asthma was 8-15% in recruited practices, with a pooled figure of 12%. This figure is higher than recent UK data, which estimate the prevalence of active asthma in all adults, that is, asthma requiring current treatment, as 5.9-8.7% (National Health Service Information Centre, 2011; Lung and Asthma Information Agency, 2011; Asthma UK, 2004). This may be explained by over-representation in working-age adults under the age of 65, and also by historical diagnoses made in childhood where asthma may have resolved but coding had not been changed accordingly.

All 4 primary care practices served urban populations in Birmingham, UK, each with low socio-economic status within the national distribution, and associated particularly with employment in manufacturing industries. Indeed UK labour force figures revealed that the sample population slightly over-represented employment in skilled manual work and plant operations. Therefore one might expect that the risk of developing occupational asthma in these areas would be higher than in more rural areas. This is notwithstanding the risk to rural dwellers in high-risk occupations such as farming and saw milling, or those who commute to urban workplaces.

The samples of 96-100 working-age asthmatics from each practice were shown to be similar to their parent practice populations in terms of gender representation. Four patients from Practice 2 who were identified prior to analysis as being incorrectly coded as asthmatic were removed from the analysis. Beyond this, the study did not adjust for any coding errors; indeed no objective demonstration of asthma, such as peak expiratory flow or spirometry measurements, were required for inclusion in the sample. However each electronic patient record was searched comprehensively for all data from asthma-related consultations, and furthermore Read-coding has been demonstrated to accurately represent morbidity from a number of chronic diseases, including asthma, in primary care (Pearson et al., 1996).



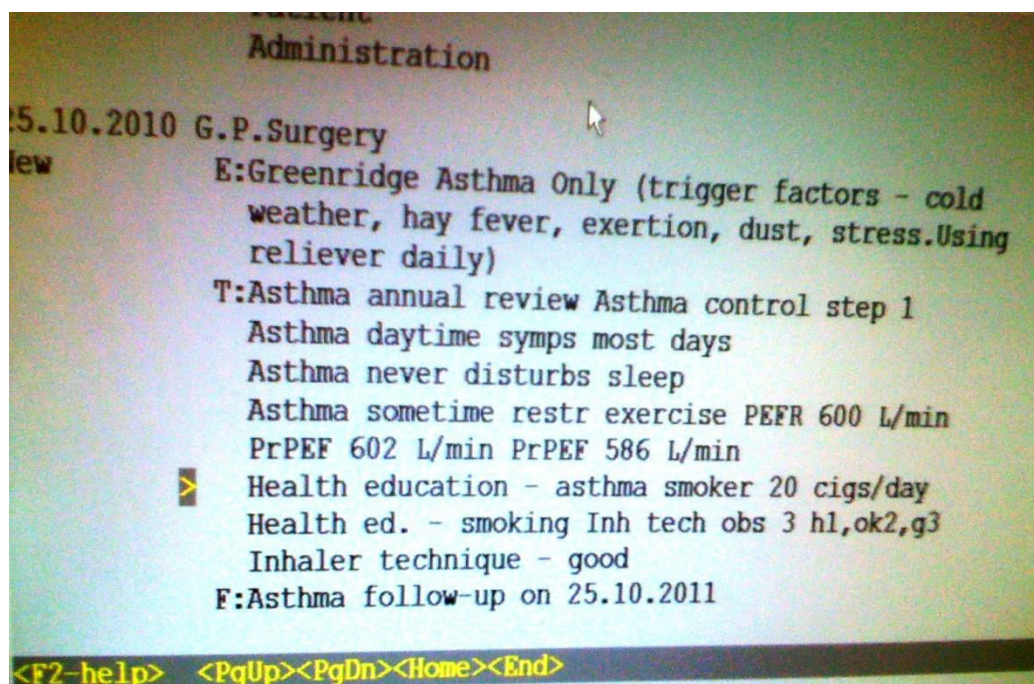
### 3.5.3 Prevalence of occupational asthma

The pooled prevalence of occupational asthma from the study sample of 0.3% of working-age asthmatics is a significant under-estimate of the true prevalence in primary care asthmatics. Estimates were low (0-0.8%) in all 4 practices. 59% of working-age asthmatics from the sample experienced onset of their asthma in adult life, and using a conservative estimate of 1 in 10 cases of adult-onset asthma being due to a work-related cause (Nicholson et al., 2010), the expected prevalence should have been in the region of 6% of working-age asthmatics. One case of diagnosed but non-coded occupational asthma was found incidentally during the study, suggesting that under-reporting by lack of coding may contribute, but the degree of under-estimation in all 4 practices suggests that at least 5-fold under-recognition exists in primary care.

### 3.5.4 Recording occupation

A significant number of working-age asthmatics experienced the onset of asthma in adult life (59%) and the majority of these (95%) had new-onset disease rather than re-activated childhood disease. The onset of asthma most frequently occurred in the 25-34 year age-band, although adult-onset disease was represented across all ages. Despite the frequency of occurrence of incident asthma, there was sparse documentation of occupation (14% of cases). All 4 practices used their own electronic asthma review templates to record data on symptom severity and control, specific asthma triggers, treatment and peak-expiratory flow. Occupation was rarely documented within the template, and did not feature as a specific prompt; indeed the majority (65%) of occupational documentations were found elsewhere in the patient record, unrelated to asthma symptoms. Where occupation was recorded, a significant number of those patients (13 out of 55; 24%) were in high-risk occupations for occupational asthma. Only 3 of those had any enquiry about work-effect on symptoms, of whom 1, a circuit-board solderer was investigated further for occupational asthma by his occupational health department; the implication being that where risk is identified, it is not appreciated or action is not taken by the primary HCW. The British Thoracic Society

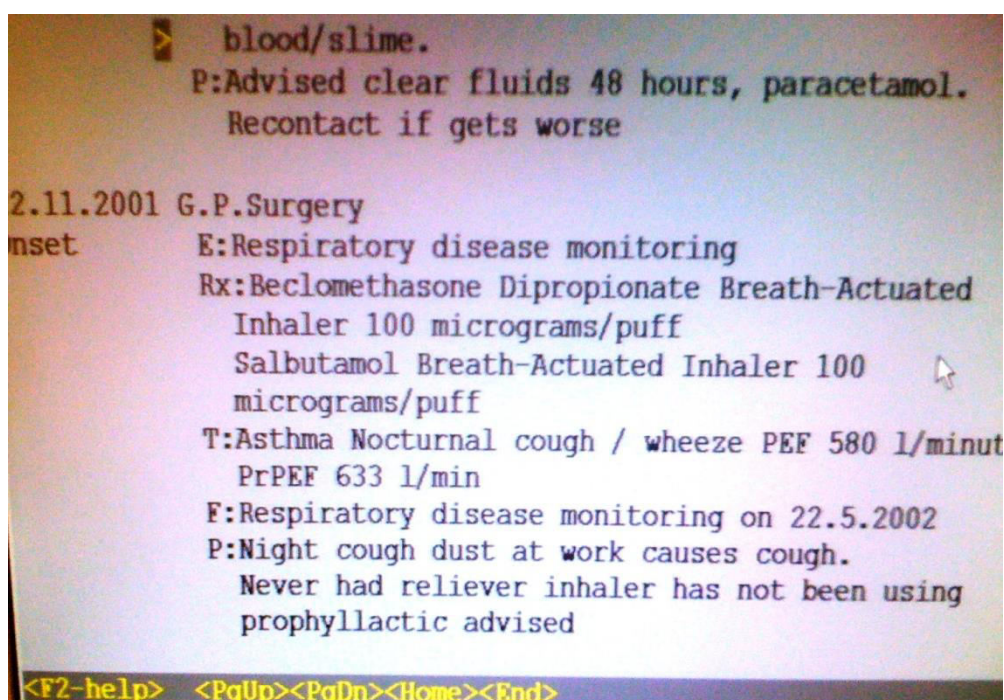
guidelines (Fishwick et al., 2012) suggest that all adult onset asthmatics should be asked ‘*What is your current job?*’ by their attending healthcare professional. Furthermore, the British Occupational Health Research Foundation (BOHRF) guidance for general practitioners and practice nurses (BOHRF, 2010a) lists high-risk occupations in the aetiology of occupational asthma, and provides an *aide memoire* for health professionals, as part of the algorithm for assessing work-relatedness of symptoms. The occupations listed are baking and pastry-making, chemical processing, farming, food processing, hairdressing, health care and dental care, laboratory animal work, spray painting, textile, plastics and rubber manufacture, welding, soldering and metalwork, woodwork, and other jobs with exposure to dusts and fumes. There was evidence that other symptom triggers and allergens are routinely considered as part of an asthma review, as is illustrated in a typical asthma review template screenshot in Figure 3.2.



**Figure 3.2.** A screenshot from an asthma review template from a study patient from Practice 1. Asthma triggers are listed, but do not include occupation, and there is no reference to work effect on symptoms. The database used is Egton Medical Systems Ltd. (EMIS) LV 5.2.

### 3.5.5 Recording work-effect

Only 2% of patients had been asked any questions about work-related asthma symptoms, at any point since diagnosis. However data from the point of diagnosis was only available in 117 patients; for the other 279, the diagnosis was made in childhood, the patient had registered at the practice after diagnosis, or the diagnosis was made before the introduction of electronic patient records. Thus from those with visible records only 4 out of 117 patients (3%) were asked work-effect questions at diagnosis. In 301 patients who had undertaken at least 2 asthma reviews, only 6 (2%) had work-effect enquiry documented recently, and 3 of those elicited a positive work effect (see Figure 3.3). There was evidence of further investigation in only 1 of those patients, the circuit-board solderer described earlier.



**Figure 3.3.** A screenshot from an asthma review template from a study patient from Practice 2. There is a reference to work effect on symptoms and more medication is prescribed, though no further action is taken to investigate the work effect. The database used is Egton Medical Systems Ltd. (EMIS) LV 5.2.

### 3.5.6 Context of findings

These findings of a lack of appreciation of work-related symptoms and occupational risk in primary care validate the results from a cross-sectional study by Fishwick et al. (2007), which revealed that 50% of patients diagnosed with occupational asthma have seen their GP at least 5 times with work-related asthma symptoms before specialist referral occurs. The findings also complement those of two other small-scale studies, including a previous cross-sectional study in one primary care practice in Oxfordshire, UK, which aimed to assess the prevalence of occupational asthma in the community (de Bono and Hudsmith, 1999). 32% of adult new-onset asthmatics were in jobs known to be at significant risk of occupational asthma, yet a potential link between occupation and symptoms had only been recorded in 18% of patients. Hoyle et al. (2010) reviewed cases of undetermined work-related respiratory illnesses that had

been reported to The Health & Occupation Reporting network in General Practice (THOR-GP) at the University of Manchester, UK and found that only 26% were referred for specialist opinion and diagnostic clarification despite uncertainty over diagnosis, of those 85% were exposed to known asthmagens.

Further study is required to establish whether or not the main barrier for healthcare professionals in identifying an occupational aetiology for asthma symptoms, is a lack of knowledge and clinical experience. Barber et al. (2010) have already created an online learning module for occupational asthma, which was associated with positive feedback from primary care professionals, and improved knowledge and self-reported use of occupational asthma guidelines.

### 3.6 Conclusion

The recorded prevalence of occupational asthma in primary care is much lower than expected, which suggests that significant under-recognition exists, with some under-reporting also likely. There is poor enquiry regarding occupation and work-effect of asthma symptoms in primary care, despite several national guidelines for identifying occupational asthma. A further study is required to establish the reasons why healthcare professionals do not ask workers with asthma symptoms about their occupation and the relationship between their symptoms and work. Furthermore, existing electronic templates for managing adult asthmatic patients could be modified to incorporate data entry and prompts for identifying occupational asthma.

## CHAPTER 4: UNDERSTANDING HEALTH BELIEFS AND BEHAVIOUR IN WORKERS WITH OCCUPATIONAL ASTHMA

### 4.1 Introduction

Occupational asthma is a disease characterized by variable airflow limitation and/or hyper-responsiveness, and caused by inhalation of an agent in the workplace (Baur et al., 2012).

Most cases of occupational asthma have an allergic mechanism, where there is sensitization to an agent after a latent period of exposure. In western industrialized populations occupational asthma is the most frequently reported occupational respiratory disorder (McDonald, 2000), and in the UK represents 1 in 6 cases of new-onset adult asthma (Nicholson et al., 2010).

Occupational asthma costs the UK £1.1 billion each decade (Ayres et al., 2011). This cost is to some extent avoidable, since individuals with a short latency between symptom onset and diagnosis or removal from exposure to a sensitizing agent, have a better prognosis when considering lung function and quality of life (Nicholson et al., 2010). However, in cohort studies from the UK and from Canada, mean delays of 3-4 years between symptom onset and diagnosis or referral to a specialist, have been identified (Fishwick et al., 2007; Santos et al., 2007; Poonai et al., 2005).

One fundamental reason for the delay in diagnosis of occupational asthma is a reluctance by the worker to report asthma symptoms (Poonai et al., 2005). Fear of losing work time, income or employment, and a lack of awareness of respiratory hazards at work have been cited as potential reasons for this (Bradshaw et al., 2007; Poonai et al., 2005; Santos et al., 2007; Gordon et al., 1997). In addition, lay perceptions of health vary according to patients' immediate cultural and social circumstances (Stacey, 1988). Such lay health beliefs may be particular to workers with occupational asthma, a group that comprises predominantly, but not exclusively, skilled and unskilled manual workers.

## 4.2 Aim

Qualitative research was considered particularly important, since there has been no in-depth study of health beliefs in workers with occupational asthma, and reports which cite barriers to diagnosis have been limited to closed-question data from cross-sectional studies. Therefore the specific aim of this study was to explore in depth the health beliefs of workers with occupational asthma symptoms, and establish a theoretical framework for understanding workers' beliefs and behaviour.

## 4.3 Qualitative research

It is more helpful to describe qualitative research by the overall aims of the study, rather than by the methods used to produce its data, since these methods do not distinguish it from quantitative research. For example, although qualitative research is usually associated with language data, and quantitative research with numerical data, there are instances of qualitative studies using frequency counts, and quantitative studies using language data. Thus a qualitative methodology is appropriate if the research aim is to understand the perspective of participants, explore meanings of a phenomenon or observe a process in-depth. There are some concepts common to much qualitative research; these are a commitment to (i) *naturalism*, that is, studying phenomena in their natural environment, (ii) *reflexivity*, that is, subjecting researchers' own values and practice to the same analysis as the topic of interest itself, (iii) a *focus on meaning and understanding*, and (iv) *flexibility of research strategy* (Green and Thorogood, 2014:p22-27).

## 4.4 Methods

The consolidated criteria for reporting qualitative research (COREQ) (Tong et al., 2007) were followed for structure and reporting of the methods section.

#### 4.4.1 Study design

An inductive, phenomenological qualitative methodology was selected as the most appropriate, given the study aims. Data were generated through semi-structured interviews.

#### 4.4.2 Setting

Workers were recruited from the Heart of England NHS Foundation Trust Occupational Lung Disease Unit, a tertiary referral unit based at the Birmingham Chest Clinic, Great Charles Street, Birmingham, UK. Within this unit 3 occupational lung disease specialist physicians (including the primary author GW) have clinical responsibility for 50-70 new outpatient referrals per annum with suspected occupational asthma. New referrals are taken from primary care, secondary care and occupational health services throughout the West Midlands.

#### 4.4.3 Eligibility criteria

The eligible sample population included any adult of working age who had received a diagnosis of, or was undergoing confirmatory investigations for, occupational asthma. Workers whose first language was not English were eligible.

#### 4.4.4 Sample selection

A purposive sample was chosen (Green and Thorogood, 2014), to include male and female workers of any working-age and ethnicity, with a variety of occupations, based on common exposures associated with occupational asthma (Diar Bakerly et al., 2008; BOHRF, 2010a).

#### 4.4.5 Participant recruitment

Workers meeting the eligibility criteria for the study were approached directly by their attending clinician and referred to the primary author for recruitment and to provide written informed consent. There were no monetary incentives for participants, therefore the consent process and interviews were undertaken at the same visit as the clinic appointment. Only one invited worker declined to participate, as he needed to leave quickly following his clinic



appointment. The patient information letter and consent form are shown in Appendix 1 and Appendix 2 respectively).

#### 4.4.6 Data collection

Each participant underwent one face-to-face semi-structured interview with the primary author lasting between 10 and 30 minutes: all interviews took place in a dedicated clinic room at the unit. A recording of each interview was made using a portable digital audio recorder, and field notes were taken during each interview to document any pertinent non-verbal responses. The initial questions confirmed basic demographic data (age, duration of employment) and thereafter the interview schedule contained five domains: (1) *the patient's understanding of their symptoms*, (2) *initial response to symptoms*, (3) *the general practitioner and occupational asthma*, (4) *social structure and occupational asthma*, (5) *the employer and occupational asthma*. Questions were generated from prevailing ideas within the background literature (Fishwick et al., 2007; Santos et al., 2007; Poonai et al., 2005; Gordon et al., 1997; Slater et al., 2000; Bradshaw et al., 2007), and through discussion with qualitative research and subject specialists (AS, SB, JA).

#### 4.4.7 Data transcription

All interviews were transcribed by hand into Microsoft Word: Mac 2011 Documents (Mac version 14.1.4; Microsoft Corporation, Redmond, WA, USA) by the primary author. Grammar was left unchanged and spelling conventions of Standard UK written English were used. The purpose of this was to aid the readability where there was linguistic variation, particularly where West Midlands' English dialects were used. The meanings of words are shaped by the manner in which they are spoken (Bailey, 2008; Lapadat and Lindsay, 1999), therefore utterances such as 'um' or 'er', sighs and in-/out-breaths, emphasized words, laughing and coughing, and encouraging noises (such as 'mm'), as well as verbal interactions such as false starts, repetitions, pauses and overlapping speech were included (Collins and Britten, 2010:p43-52; Bailey, 2008). Non-verbal features were represented only when cited

in field notes, where it was felt that they aided the interpretation of accompanying speech. Interruptions were acknowledged but their content not transcribed, and social talk before and after each interview was excluded. The transcription conventions are shown in Table 4.1.

Transcription conventions	
(?)	Talk too obscure to transcribe
Hhhhh	Audible out-breath
.hhh	Audible in-breath
[	Overlapping talk begins
]	Overlapping talk ends
(.)	Silence, less than half a second
(..)	Silence, less than one second
(2.8)	Silence measured in 10ths of a second
:::	Lengthening of a sound
becau-	Cut off, interruption of a sound
<u>he</u> says	Emphasis
=	No silence between sounds
LOUD	Loud sounds
?	Rising intonation
(left hand on neck)	Body conduct
[notes, comments]	

**Table 4.1. Transcription conventions. Taken from Bailey (2008), adapted from Jefferson (1984).**

#### 4.4.8 Thematic analysis

Thematic analysis is a common approach for qualitative data analysis that summarizes participants' accounts, provides a map of the content across the dataset, including both variations and regularities (Pope and Mays, 2006:p69-70). Themes are "*recurrent concepts which can be used to summarize and organize the range of topics, views, experiences or beliefs voiced by participants*" (Green and Thorogood, 2014:p210). Analysis begins by identifying and coding themes in the data, and then examining the whole dataset to ensure

that all manifestations of each theme have been accounted for. Ryan and Bernard (2003) suggested that identifying repetition, metaphors, analogies, similarities or differences are all strategies that can be utilized. These may be developed deductively at the outset of the study, or inductively by beginning analysis early on during data collection and then applying the coding scheme to the remaining dataset. Once coded, the dataset can be viewed horizontally, by theme, in order to provide a rich and detailed understanding of the data. Subsequently by moving the analysis beyond a descriptive exercise the researcher may try to identify relationships between the themes, developing classifications, taxonomies or models to express these connections (Pope and Mays, 2006;p69-70).

Thematic analysis was undertaken by the primary author, with a co-investigator (AS) acting as a critical friend (Melia, 2000; Soundy et al., 2012). Analysis commenced once data collection was underway, using empirical data driven codes in an inductive process. An interim coding scheme was developed after 10 interviews and the semi-structured interview schedule was adapted to pertinent themes (Appendix 3 contains final interview schedule). Further interviews and analysis were undertaken simultaneously until no further new themes were identified (audit trail is available). Typicality of response and deviant case analysis, as well as the role of the researcher in the research process (reflexivity) were all considered, in order to maximize trustworthiness (Green and Thorogood, 2014).

#### 4.4.9 Reflexivity

The primary author (GW) is a 36-year old male White British specialist doctor in occupational lung diseases, who did not attend any of the participants as a clinician. It was however necessary to quickly establish a rapport with interview participants, in order to encourage openness in response to sensitive questions about working and healthcare relationships, when workers feared that any criticism voiced might lead to negative employment consequences. The interview site was an empty clinic room in an NHS department, chosen in order that workers could undertake consent and interview in one visit.

The primary author explained that clinical judgments were not being made on the basis of participants' responses, however the interview location can impact on data generation (Pope and Mays, 2006; Green and Thorogood, 2014): interviewing somebody in a public space, such as a hospital room, may stress different aspects of their identity rather than in their own home. Although confidentiality was assured, effort was required to create a relaxed atmosphere and develop a rapport, by being non-judgmental and interested.

#### 4.4.10 Ethical approval

Ethical approval for the study was granted by the North West (Haydock) National Research Ethics Service (NRES) Committee (REC reference: 13/NW/0035) and sponsorship was provided by Heart of England NHS Foundation Trust (R&D Code: 2012158RM).

### 4.5 Results

#### 4.5.1 Descriptive analysis

Twenty workers participated in the study (14/20; 70% male) and the median age was 52 (interquartile range=49-57). Sixteen out of 20 (80%) of workers were White British and the other 4 were Pakistani, British Asian (of Pakistani origin), Black Caribbean and White Eastern European (Polish). Workers' occupations and exposures were varied and these are shown in Table 4.2. Five participants worked in the healthcare industry, and the most commonly encountered causative agents were isocyanates (n=4), wood dust (n=2), metalworking fluids (n=2) and solder flux (n=2).

<i>Patient number</i>	<i>Age</i>	<i>Gender</i>	<i>Ethnicity</i>	<i>Occupation</i>	<i>Exposure</i>
P1	64	Male	White British	Wood worker	Wood dust
P2	49	Male	Pakistani	Automotive assembly	Metalworking fluid
P3	49	Male	White British	Plumbing lecturer	Acidic solder flux
P4	52	Female	White British	Hospital cleaner	Cleaning agents
P5	36	Male	White (Polish)	Circuit-board repairer	Colophony solder flux
P6	57	Male	White British	Swimming pool supervisor	Chloramines
P7	55	Female	White British	Plastics moulder	Polymer fume
P8	48	Male	Asian British	Metalworker	Metalworking fluid
P9	48	Male	White British	Chemical engineer	Vitamin E
P10	52	Male	White British	French polisher	Wood dust
P11	55	Male	White British	Orthopaedic practitioner	Methyl methacrylate
P12	43	Male	White British	Metalworker	Chromium
P13	22	Female	White British	Anaesthetic nurse practitioner	Anaesthetic gases
P14	63	Male	White British	Adhesive development chemist	Isocyanates
P15	55	Male	White British	Car headliner assembler	Isocyanates
P16	58	Female	White British	Operating theatre nurse	Diathermy
P17	59	Female	Black Caribbean	Hospital ward nurse	Latex
P18	52	Female	White British	Curtain blind manufacturer	Isocyanates
P19	64	Male	White British	Security van driver	Diesel exhaust
P20	50	Male	White British	Spray painter	Isocyanates

**Table 4.2. Summary of all workers undertaking semi-structured interviews, showing demographics, occupations and causative agents.**

#### 4.5.2 Qualitative analysis

Four themes concerning health beliefs and health-seeking behavior were identified: (1) the worker's understanding of his/her symptoms, (2) working relationships, (3) the worker's initial course of action, and (4) the worker's negotiation of healthcare encounters. Themes, sub-themes and codes are summarised in Table 4.3.

Theme	Sub-theme	Code
<u>Theme 1</u> : The worker's understanding of his/her symptoms	Lack of acceptance of symptoms as an illness	Poor insight into onset
		Considered to be normal
		Ignoring symptoms
	Lack of association of symptoms with work	Inability to see a pattern
		Noticed pattern but over-simplified
		Only makes link after significant event
	Failure to attribute symptoms to asthma	Accepts something is wrong but no appreciation it may represent asthma
		Misattribution to another illness
		No knowledge of asthma as a disease
<u>Theme 2</u> : Working relationships	Relationships with colleagues	Fear of talking between colleagues
		No culture of discussing health
	Relationships with management	Company structure (the artificial person)
		Ineffective management
		Ineffective individuals
		Focus on productivity over health
		Intimidation
		Attention only in light of authority advice
		Fear of job and financial loss
<u>Theme 3</u> : The worker's initial course of action	Proactive person response	Goes to the GP
		Goes to another authority
		Prompted to act by family members
		Becomes the "go-to" person
	Passive person response	Absenteeism
		Presenteeism
<u>Theme 4</u> : The worker's negotiation of healthcare encounters	Failure to initiate action by the GP	Failure to enquire about work at all
		Reliant on patient to offer work aspect
		Dismiss work effect on symptoms
		Oversimplify illness
		Misattribution of symptoms to another disease
	Proactive negotiation by the worker	Makes link with work-aspect themselves
		Requests a second opinion
	Passive acceptance of authority by the worker	Initial misunderstanding fuelled
		Initial understanding challenged

**Table 4.3. A summary of themes relating to the health beliefs and behaviours of workers with occupational asthma symptoms.**

#### 4.5.2.1 Theme 1: The worker's understanding of his/her symptoms

Five out of 20 (25%) workers lacked insight into the onset of their symptoms, with one worker [P5] suggesting that this was due to the intensity of shift work:

[P5] *"I don't speak with anybody before with this problem [asthma symptoms] because I don't see the problem... After eight hours I think it is a little bit hard to check on these things because you are working, you are tired"*.

Nine out of 20 (45%) workers considered asthma symptoms to be normal while working, that is, expected from working with dust or chemicals, normal for undertaking shift work, or part of ageing or deconditioning (Table 4.4). Eight out of 20 (40%) workers appreciated their symptoms but ignored them, either because they appeared to be short self-limiting illnesses, or they were not perceived as serious enough to warrant further attention. Indeed one worker [P12] sought medical assistance only when his symptoms began to impact on him: *"I was leaving my shift early, so obviously I weren't earning my normal wages, so it was starting to impact on my lifestyle, my job and my earnings"*. Six out of 20 (30%) workers initially failed to recognize the work-related pattern typical for occupational asthma symptoms, due to their gradual or indistinct onset. In one case [P17] the reason for this was identified as the worker's symptoms being masked by a hectic lifestyle. Five out of 20 (25%) workers did recognize the work-related pattern but dismissed work as the cause because environments were not dusty, no colleagues were affected, or because there was a long latent exposure before symptom onset (Table 4.4).

For 7/20 (35%) workers, suspicion of work causation was triggered only by a significant event such as a serious illness (pneumonia, hospital attendance) (3/20; 15%) or a discussion with a colleague regarding the meaning of their symptoms (4/20; 20%).

[P11] *“There was a person at work who had to retire um: for the same reason, retired very early, working in the same theatre, and she said “this is what happened to me, I think it may be happening to you as well”.*

Ten out of 20 (50%) workers recognized their symptoms but misattributed them to recurrent self-limiting respiratory tract infections (4/20; 20%) or other serious illnesses (cancer (3/20; 10%), tuberculosis (1/20; 5%), heart disease (2/20; 10%)); this was often based on previous or family experiences (Table 4.4). Importantly 3/20 (15%) workers were unaware of asthma as a disease entity, either with no previous exposure (2/20; 10%) or believing it to be a childhood disease (1/20; 5%). Conversely, one worker [P12] had previously suffered from allergic diseases which enabled him to recognize the symptoms rapidly: *“Yeah well I’d had mild asthma as a child which had gone, I’ve also had, as a child, hay fever- sort of allergy type things so- no I was aware it was asthma- my partner’s got asthma as well=”.*



<b>Normalising symptoms</b>	
P17	<i>"I think I just thought the chemicals smelled strong (.) and I thought oh I'm just coughing because of the chemicals (.) I didn't put it that I was coughing because something was wrong with <u>my lungs</u>, I just put it that I was reacting to a smell"</i>
P10	<i>"I'd start feeling a little bit breathless and stuff but I was putting it down to maybe it's the patterns, because I hadn't done nights for a long time"</i>
P14	<i>"I didn't sort of feel (.) that I was ill and therefore that I'd got anything to talk about with the doctor, you know (.) I just thought I was unfit, compared with those eighty year olds running round"</i>
<b>Ignoring symptoms</b>	
P7	<i>"I just thought oh I've got a cough, I've got a cold, and then when I thought I'll go and see the doctor, it disappeared. So I thought oh I'm alright",</i>
P16	<i>"Well I think when you've got progressive things like weight loss or other things going on, then you get concerned, but when it's a cough you tend to ignore it=".</i>
<b>Not linking work-related symptoms with workplace hazards</b>	
P1	<i>"I just thought it can't be [work-related], because everybody would have it, that was in my head. There's people there who've been there as long as I've been there, twenty odd years and they're not even affected",</i>
P1	<i>"I didn't think it was work I didn't... 'cos if you went down to where I worked and looked (.) you wouldn't see that much dust"</i>
P6	<i>"That was it, thinking why should- you know why should work be a problem, it hasn't been a problem up to now? Why should it have sort of changed?"</i>
<b>Failure to attribute symptoms to asthma</b>	
P16	<i>"I thought I'd got a chest infection... Or just (.) a cold or something so I sort of ignored it"</i>
P9	<i>"I've had cancer before, a tumour on the back, so I was just worried if I'd got cancer on the chest"</i>
P7	<i>"Well I just wondered what it was, didn't know what it was, and because all my family have heart problems I just went and said to the doctor, I've got this horrendous (.) pain in the middle of my chest and in the middle of my back"</i>
P3	<i>"(.) That's when the adverts- campaign started, if you've had a cough for more than three weeks, get it checked out, it could be the start of [lung] cancer, so it did worry me to be honest"</i>
P8	<i>"I didn't know asthma (.) and just the fact that I had- (.) couldn't breathe, that's what it was. I don't know, I've got no mates with asthma, no one that I can compare notes with or anything like that".</i>

**Table 4.4. Workers' responses showing examples of the variation in perception of occupational asthma symptoms.**

#### 4.5.2.2 Theme 2: Working relationships

Workers frequently did not discuss their symptoms with work colleagues (see Table 4.5). Five out of 20 (25%) workers feared that “careless talk” about their ill health would reach management and have a detrimental effect on their employment, through which they might suffer financially. Six out of 20 (30%) workers (all male) stated that health matters were never discussed at work, either because there was no prevailing culture for open discussion, because colleagues were not able to provide a solution, or if a worker was perceived as unfit by his/her colleagues this might cause antipathy.

Seventeen out of 20 (85%) workers described poor relationships with their employers over health matters. Many of those workers felt that management would not acknowledge their concerns (Table 4.5): this was either due to inaction from ignorance of workplace health and safety, or through mistrust of workers’ complaints of ill health. In fact 6 workers suggested that their managements only took their concerns seriously once authoritative information was available, such as a report from occupational health or an occupational lung disease specialist. Four workers identified individual managers who they felt were unsympathetic or ineffectual in dealing with their concerns. Twelve workers believed that company productivity was considered more important than the health and safety of employees, and 2 workers felt they were being exposed to an avoidable risk of asthma because their employer would not pay for process control measures, such as local exhaust ventilation on soldering stations [P5]. One worker [P4] was intimidated after raising concerns:

[P4] *“I got warned at work for telling the other ward hostesses who do the same job as me (..) because I seen them at break and they had me in the office and they told me to stay (.) don’t come over the other side of the hospital”.*

Therefore many workers were dissuaded from discussing their health issues with managers, mainly for fear of being seen as unfit for work and being dismissed, but also because they could not see that it would effect a workplace solution.

However, 2/20 (10%) workers had more positive relationships with their employers; one worker [P2] found his employer to be both communicative and supportive due to trust developed through long service: *“I’ve been with the company twenty-three years, and I’m quite happy to work there and they have supported me... I’m quite happy that the management know about my issues, and the job they’re giving me is suitable”*; the other worker [P11] felt that his manager was supportive and proactive in finding a solution, and was almost being too cautious in redeploying him: *“The theatre manager at the time was very concerned, he wanted to redeploy me to another department and take me out of theatres completely”*.

<b>Discussion with colleagues</b>	
P19	<i>"If you talked about anything like that you got the sack (..) 'cos Company X [security company] in them days were a bullying company, and you daren't talk about anything".</i>
P7	<i>"But everybody's [work colleagues] frightened of losing their job aren't they? And because I'm a bit more- well I'll tell the management what I think, and they're [colleagues] a bit oh God no, we'll do it, you know, behind their back"</i>
P12	<i>"Yeah well there was er:: a group- there was possibly half a dozen of us that were all experiencing the problems and we were talking within ourselves, but you tend- in the industry, in the current climate, you feel sort of quite scared to say too much because you don't want it to impact on your future"</i>
P8	<i>"They couldn't give me a fix or anything, so it wasn't worth asking the question".</i>
P1	<i>"Then you get the resentment from off the peers that you are working with, because some of them are covering your work, you know".</i>
<b>Discussion with management</b>	
P8	<i>"They weren't helpful at all, because they don't think that they could inflict asthma or anybody or nothing",</i>
P1	<i>"One of the managers (..) ended up with the same type of thing as what I got (..) and... it wasn't until he got it himself he started to say "I didn't know it was like this, you know""</i>
P3	<i>"My manager's an absolute smashing bloke (..) but he's not a manager. He was put in place as a 'yes man'. He cannot make a decision without his manager telling him what to do basically"</i>
P20	<i>"They're totally unsympathetic, they're interested in making a buck and that's it... We live in a culture at the moment where we sue for this, that, and the other, they just wouldn't want you on the premises any more... There would be no sympathy... I've never been offered light duties in all my life, they're not going to say well we'll get him to do this, that and the other, it's just we'll find someone else"</i>
P18	<i>"I mean I had a heart attack and they didn't really- (..) they were concerned, but it was more for the business side I think, than my personal (..) [health and safety]"</i>
P4	<i>"You feel as if you um (..) if you don't come into work when you are ill (..) you get penalized, you know"</i>
P19	<i>"I mean the company pays for their own particular medical people, who'd say "that bloke is unfit for work, get rid of him" (..) just so I wouldn't take it any further (..)"</i>

**Table 4.5. Workers' responses showing examples of reluctance to talk with colleagues and management about their health problems.**

#### 4.5.2.3 Theme 3: The worker's initial course of action

Eight out of 20 (40%) workers responded proactively to seek an explanation or solution once they had acknowledged their symptoms. Most workers consulted their GP, although one worker [P2] self-referred to his employer's occupational health. Eight out of 20 (40%) workers were prompted to take action by a family member, particularly when short-lived symptoms had recurred or become permanent: [P2] *"They [patient's family] thought that this coughing is getting regular and, you know, it needs sorting, you need to have a word with the doctor about it"*. Conversely 2 workers felt that their families gave no encouragement to them to seek help, due to lack of insight into work processes and the nature of asthma. Additionally one worker [P3] felt stigmatized by his asthma symptoms, which he felt were comparable with those of depression, and would be seen as unfit for work by his family. Two workers described becoming a source of support or authoritative advice for their colleagues:

[P4] *"The girls [colleagues] were asking me how did I get on? You know with tests and that, and I said that er (.) .hhh you know, I said they ought to be careful and- and just watch out for symptoms"*

Six out of 20 (30%) workers sought little in the way of an explanation or a permanent solution. This was manifested by repeated episodes of sickness absence: [P8] *"I was finding that it was hard- I couldn't do certain things... I was having a lot of time off as well, gave me a lot of hassle at work as well... if I feel that something's not right in there I'll go home"*, or by presenteeism: [P13] *"I've never liked taking time off work, so even when like I've struggled, I've kind of just like persisted and just carried on"*. A variety of reasons for persisting at work while unwell were given by workers, such as fear of financial loss: [P18] *"I mean I could've really come out- really done with coming out of it, but (.) everybody needs to earn a living"*, fear of being penalised: [P8] *"If you have time off they still chuck the rulebook at you. I was feeling ill right but I still had to go to work"*, and a sense of responsibility to the employer: [P12] *"I'm a [shift] manager, so I couldn't really lose much*

*time, you know... I need to be there to run the show really, not sounding silly, you know but- they do rely on some of us”.*

#### *4.5.2.4 Theme 4: The worker’s negotiation of healthcare encounters*

Sixteen out of 20 (80%) workers believed that their initial encounter with a healthcare professional, almost exclusively an appointment with their GP, was unhelpful, with inadequate action being taken to explain their symptoms or make a diagnosis. Seven workers believed that the GP failed to enquire about their occupation or whether their symptoms were work-related, indeed 3 workers stated that they had to prompt the GP in order to enable discussion about risk associated with their job, or the implication of a work-related symptoms: [P20] *“It was me that mentioned work- it was me that said I’m a painter I can’t just say I got asthma, that’s me I can’t work anymore”*. Many workers felt that they were given an inadequate explanation for the work aspects of their symptoms either by being dismissed without further exploration or by over-simplifying the pathology: [P6] *“All the time I’d be sort of struggling with this sort of wheezy sort of chesty cough sort of thing... and they [GPs] sort of went “oh post nasal drip””*. One worker [P15] believed that his GP dismissed his concerns about the work aspect of his symptoms because he thought that he was exaggerating for financial gain. Seven workers believed that their GP initially followed the wrong diagnostic line: [P7] *“I just had what I call pain in the middle of my chest... so they [GP] always said “it’s your heart, it’s your heart” ‘cos it- at that time I wasn’t coughing constantly”*; for some this exploration was understandable, though for others this was an unacceptable cause of delay.

Many workers were proactive in negotiating the medical management of their illnesses, by questioning the explanations they were given, or simply asking for a second opinion. Four workers recognized the limitations of their GP consultations and identified the work link themselves, through Internet research or by evolution of the symptom pattern:

[P20] “...and then she [practice nurse] just said “you got asthma” (..) and I said well (.) I’m not happy with you just telling me I’ve got asthma, I paint cars for a living (.) if I go in somewhere and I’ve got asthma, then that puts me out of work (.) er:: so (.) what sort of asthma have I got, you need to be a bit more specific than that?”

[P3] “I started to think, it’s actually at its worst (.) in the first few weeks of the course [plumbing course] and then sort of improves, and then it starts off again... when all the soldering were taking place... So then I start to put two and two together”,

[P7] “I kept saying there’s no ventilation, there’s no extractor fan. I looked it all up, found out it could cause different types of cancer and that, and said I’m sure it’s works related”.

Five workers accepted an initial inaccurate explanation for symptoms by their GP, or in one case by a respiratory physician, because they were seen as an authority. One worker [P3] had his own misunderstandings fuelled:

[P3] “I genuinely thought I’d got a chest infection um: (..) Then as my GP sort of- (..) confirmed that (.) I believed him and I took the antibiotics and I took stronger antibiotics... and then I’m told there are six thousand viruses out there and- so you believe them, they’re the medics you know”

## 4.6 Discussion

### 4.6.1 Summary

This study used a qualitative approach to define the health beliefs and behaviour of working-age adults with occupational asthma symptoms through semi-structured interviews and thematic analysis. Four major themes were identified that were important in how workers behaved with regard to their asthma symptoms before diagnosis. There was a variation in how workers perceived the onset, the timing and the seriousness of their symptoms, and the evolution of their understanding was heavily dependent on how actively they pursued advice or a solution for their symptoms.

### 4.6.2 Understanding symptoms

Understanding of symptoms varied between individuals from a lack of insight into the onset, pattern and nature of symptoms, through to misunderstanding of what they represented, or ignorance of the existence of asthma as a disease entity; indeed most workers who were interviewed failed to suspect or identify asthma as the cause for their symptoms initially. This is expected, since there is a variation in perception of asthma symptoms amongst non-occupational asthmatics, with poor insight and under-appreciation of severity of symptoms described in male and female adults of all ages and socio-economic backgrounds (Smith et al., 2009; Scarno and Stendardi, 2006; Haughney et al., 2004). This may account for significant delays in diagnosis in the occupational setting, where latencies of 8-months or more between the onset of asthma symptoms and consulting a physician have been reported (Poonai et al., 2005). Many workers in the present study required a cue to action to change their health seeking behaviour, such as an illness event, a prompt by a family member or colleague, or a decline in physical function affecting their ability to work. However, despite evidence that UK Health and Safety Executive educational campaigns have reduced the incidence of occupational asthma related to certain individual exposures like isocyanates in motor-vehicle repair (HSE, 2007a; Stocks et al., 2013), no worker in the present study



indicated that a workplace educational intervention had changed their understanding of their own symptoms.

#### 4.6.3 Working relationships

Employers were perceived as ignorant of health matters, and ineffective or intimidating when dealing with workers' concerns, often because workers believed that their focus was on the financial cost of asthma (loss of productivity) rather than the human cost of ill health. Indeed workers feared being seen as unfit for work, losing their employment and suffering financially; for many it was understandably more preferable to carry on in a job exposed to respiratory hazards, than to face unemployment or difficulties finding equivalent work. In the study by Bradshaw et al. (2007), a significant proportion of workers with occupational asthma continued working with asthmagens that caused ongoing symptoms, since the fear of financial loss through job loss was greater than the concern for their own respiratory health. This is understandable, since 85% of workers who leave their employment (either become unemployed or find alternative work) suffer a loss of income of 22-50% (Vandenplas et al., 2002; Moscato et al., 1999; Ameille et al., 1997; Larbanoir et al., 2002). Additionally, a large proportion of the costs of occupational asthma are borne by the individual worker (49%) rather than the employer (3%), who therefore has little incentive to act (Ayres et al., 2011).

#### 4.6.4 Action, inaction and negotiation

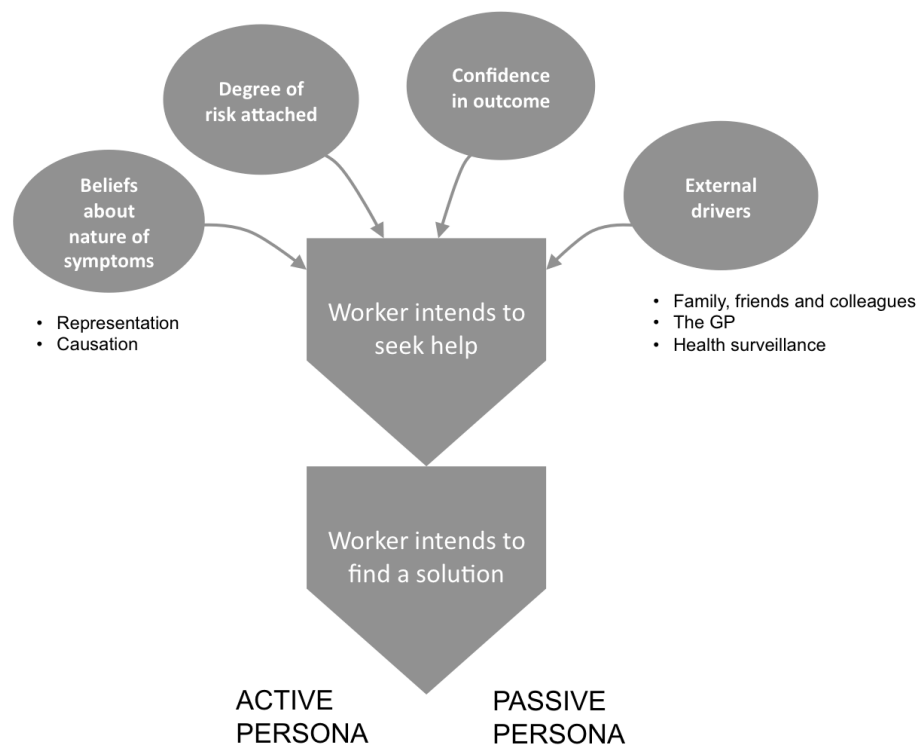
The current results highlighted that the evolution of a worker's understanding of their symptoms depended upon how motivated they were to define them, or seek a solution. Proactive workers would seek help from an accessible authority, who was usually the GP; some workers later became a source of authority themselves for other affected workers (the 'go-to' person). Motivations for seeking medical help were from enhanced internal foci on symptoms as they worsened or recurred, from the inability to work and earn money while experiencing symptoms, or through pressure from family members. Passive workers either took repeated episodes of sickness absence without further exploration of causation, or

persisted at work with symptoms through fear of losing their job, a sense of responsibility to colleagues or their employer, or simply because they felt that the symptoms they were experiencing did not impact on their function. Indeed there is evidence that workers persist in work environments despite knowing they are being exposed to respiratory hazards, which in some cases actually cause them harm (Slater et al., 2000).

Although there was more than one pathway for accessing healthcare (some routes were closed off to several workers if an employer had no formal occupational health provision, or there was no Trades Union representation) most workers saw their GP in the first instance.

However, it was common for individuals to reflect on poor experiences, mainly generated by missed opportunities to identify work causation. The inability to identify causation was attributed to the GP's lack of enquiry, dismissing work-related information, oversimplifying symptoms or misdiagnosing. This is supported by data that show GPs, who have an important role in screening for occupational asthma, fail to enquire about occupational exposure and the effect of work on asthma symptoms (Walters et al., 2012) despite recent guidance (BOHRF, 2010). In cross-sectional studies, GPs have cited insufficient time, lack of expertise and poor access to specialist services as barriers to diagnosis (Parhar et al., 2011; Holness et al., 2007; Poonai et al., 2005).

Proactive workers negotiated ineffective encounters by questioning poor explanations; however, passive workers repeatedly accepted poor explanations, even when their understanding of the work-symptom relationship was accurate. The health belief model explains differences in behaviour by variations in workers' health beliefs (Rosenstock, 1966; Rosenstock et al., 1988): these are core beliefs related to the perceived seriousness of, and susceptibility to a disease, personal costs and benefits of changing behaviour, and the presence of specific cues to action such as illness events. Indeed, in this study there appear to be key influences motivating a worker to seek an explanation for their symptoms or a definitive solution (see Figure 4.1).



**Figure 4.1. The major influences on workers' health-seeking behaviour: (1) the belief that symptoms may represent asthma and the chronicity and pattern of symptoms is related to ongoing exposure at work, (2) whether disclosure of symptoms to colleagues or employers is likely to effect any change in the workplace or workplace procedures, or conversely result in financial loss, (3) a worker's confidence in their GP (or other healthcare professional) that a consultation will result in an adequate diagnosis or solution, and (4) whether there are external drivers, such as a prompt by a concerned colleague or family member, occupational health surveillance, or an effective healthcare consultation. The activity or passivity of workers' health-seeking behaviour is determined by the nature and extent of their prevailing beliefs.**

#### 4.6.6 Limitations

Participants had a variety of causative exposures, and were broadly representative of workers with occupational asthma in the West Midlands, UK (Diar Bakerly et al., 2008). 80% of workers interviewed were British males, the rest were English-speaking ethnic minority workers. However some locally represented ethnicities, such as Irish, Somalian or Black African were not accounted for in the sample. The sampling strategy accommodated workers already diagnosed with occupational asthma and undergoing clinical follow-up, which may have introduced recall bias, and excluded those workers who had not yet sought, or had no intention of seeking healthcare, who may have different health beliefs. These biases limit generalizing the findings to all workers with work-related respiratory symptoms.

Additionally data collection was not a purely inductive exercise because the interview schedule was determined beforehand, albeit loosely; a compromise was required to ensure that data collection was reasonably practicable. In order to increase truthfulness and reduce subjectivity, strategies such as deviant case analysis and triangulation via a critical friend, were employed.

#### 4.7 Conclusion

This study aimed to gain an in depth understanding of the beliefs and behaviour of workers with occupational asthma symptoms, and define the major barriers to diagnosis on the part of the worker. Perceptions of asthma symptoms in the workplace varied, with some aspects such as lack of insight into symptom onset, and poor awareness of work-effect or the nature of chronic asthma, likely to predispose to a significant delay in diagnosis for many workers. These are basic insights that should be accounted for when designing workplace interventions, and should be considered in addition to the educational goals associated with work hazards and risk. Many symptomatic workers were discouraged from taking action through a fear of financial loss, or a lack of confidence that disclosure of symptoms would effect either a healthcare or workplace solution. Equipping workers with guidance on the diagnostic process and consequences of occupational asthma, along with strategies for

negotiating solutions with healthcare professionals and employers, could empower workers to make more informed choices about their health, at an earlier stage in the disease process.

## CHAPTER 5: BARRIERS TO IDENTIFYING OCCUPATIONAL ASTHMA FOR HEALTHCARE PROFESSIONALS

### 5.1 Introduction

Mean delays in the diagnosis of occupational asthma of 3-4 years have been reported in the UK and elsewhere (Fishwick et al., 2007; Poonai et al., 2005; Santos et al., 2007), which lead to avoidable societal cost (Ayres et al., 2011) and worse health outcomes and quality of life for affected workers (Nicholson et al., 2010). One fundamental reason for this delay is lack of enquiry about an occupational cause for asthma symptoms by healthcare professionals; this is notwithstanding publication of guidelines for healthcare professionals on the management of workers with suspected occupational asthma (BTS/SIGN, 2012; Fishwick et al., 2012; BOHRF, 2010a), which suggest that adult workers with new onset or reactivated asthma symptoms should be asked about the nature of their work and whether their asthma symptoms are better away from work. It is clear from the study in Chapter 3 and from previously published research that when consulting working-age asthmatics, GPs and practice nurses in the UK rarely enquire about the nature of work, or the effect of work on symptoms, even when the asthma is of new-onset (de Bono and Hudsmith, 1999; Fishwick et al., 2007; Walters et al., 2012). In cross-sectional studies from elsewhere, GPs have cited insufficient time, lack of expertise and poor access to specialist services as obstacles to diagnosis (Parhar et al., 2011; Holness et al., 2007; Poonai et al., 2005); however, the nature of the barriers to enquiry by UK healthcare professionals have not been explored in any detail. This specifically requires urgent investigation in order to inform the best strategy for improving identification of incident cases of occupational asthma.

### 5.2 Aim

The aims of this study were to define the important barriers that prevent healthcare professionals from identifying potential cases of occupational asthma, and to see whether these differ between healthcare professions and medical specialties.

## 5.3 Methods

### 5.3.1 Methodology

A cross-sectional questionnaire design was used to address the research aims of the study. The questionnaire was distributed by e-mail to clinicians in the UK attending working-age asthmatic patients as part of their practice.

### 5.3.2 Questionnaire development

A matrix of beliefs and attitudes hypothesized to delay identification of occupational asthma was developed and is shown in Appendix 4. This was informed by focused discussion with 2 occupational lung disease specialists, by reference to previously cited barriers to diagnosis (Poonai et al., 2005; Parhar et al., 2011; Barber et al., 2010; Lugtenburg et al., 2011), and from data gathered from the qualitative study in Chapter 4. Using the matrix, simple and specific questionnaire items were written (Williams, 2003) and 5-point Likert ordinal scales (strongly agree, agree, neither agree nor disagree, disagree, strongly disagree) used for closed responses (Bowling, 1997): an opportunity for open comment was also included. Questions asked whether participants enquired about the nature of work, the effect of work on asthma symptoms, and whether they followed guidelines, as well as a number of potential barriers to doing so (for example: motivations, perceived importance, experience). Demographic questions referring to number of years' experience, medical specialty (occupational health, general practice, general hospital medicine, respiratory medicine, occupational lung disease), role (doctor, nurse) and level of training (for doctors: trainee, consultant/GP) were also included.

A case study-based section was then developed to follow the first set of questions, for which participation was optional. A questionnaire designed and validated for assessing health-care professionals' perceptions on reaching judgments about work-causation of asthma and musculoskeletal disease was modified with permission (Beach et al., 2012), in order to utilize the asthma case. This consisted of 4 workplace scenarios based on a bakery worker with

asthma symptoms, with different combinations of strong or weak work- and non-work histories:

- Type SW—strong work, weak non-work history/features (occupational asthma likely);
- Type SS—strong work, strong non-work history/features (grey case);
- Type WW—weak work, weak non-work history/features (grey case);
- Type WS—weak work, strong non-work history/features (occupational asthma unlikely).

Participants were randomized automatically by the online distribution tool to read one of the 4 scenarios, in order that a similar number of each was distributed. They were then given closed questions with 5-point Likert responses that measured confidence in allocation of causation. An opportunity for open comment was included at the end. The final version of the questionnaire is shown in detail in Appendix 5. The questionnaire was then administered through the online cloud-based forms creator [www.surveymonkey.com](http://www.surveymonkey.com) (Palo Alto, California, USA).

### 5.3.3 Pilot phase

The questionnaire was circulated to 10 healthcare professionals with clinical experience of managing working-age asthmatics, construct validity was tested by identification of any redundant or ambiguous items, and the questionnaire was modified accordingly. The questionnaire (non-case study section only) was then completed by 17 healthcare professionals (7 hospital physicians, 4 general practitioners, 6 specialist respiratory nurses) on 2 occasions, 3-weeks apart, and test-retest reliability of each item was assessed using Wilcoxon rank-sum tests and Cohen kappa statistics (Williams, 2003; Field, 2004). Strength



of agreement by kappa statistic was rated as poor ( $<0.20$ ), fair (0.21-0.40), moderate (0.41-0.60), good (0.61-0.80), or very good (0.81-1.00) (after Landis and Koch, 1977).

#### 5.3.4 Study phase

UK-registered nurses and doctors with clinical responsibility for working-age asthmatics, who were working in occupational health, general practice and general hospital medicine were targeted for the study through their professional organisations. Estimates of the numbers of practising nurses and doctors from each specialty are shown in Table 5.1. Professional organisations were invited to sponsor the study via their national or regional offices by e-mail communication: those agreeing to do so were e-mailed a copy of the ethical approval letter, participant invitation letter (shown in Appendix 6) and a text insert with a hyperlink to the questionnaire, appropriate for an email or bulletin (Appendix 7). Sponsoring organisations passed the invitation to participate to their members, either by e-mailing them directly or by inclusion in a bulletin, and subsequently sent out reminders 4-weeks after the initial contact to increase response rate. Membership list sizes for sponsoring organisations are shown in Table 5.1. The online questionnaire remained open for 10-weeks, at which point no further responses were collected. There was no monetary incentive for organisations or their members to take part.

	Health professionals			Professional organisations		
	Professional group	Estimated number of UK practitioners		Representative professional organisation	Number of members contacted	Number of responses (%)
		Consultant (or GP) level	Specialist trainee level			
Doctors	Occupational lung disease specialists	19	n/a	• Group of Occupational Respiratory Disease Specialists (GORDS)	19	17 (89)
	General hospital physicians	11,225 <sup>1</sup> Of which 4367 are involved in acute medical take	7049 <sup>1</sup> Of which 5,382 in acute specialties / traditionally involved in acute take	• Royal College of Physicians (RCP) East Midlands (North and South); • Postgraduate deaneries (West Midlands, East of England, Yorkshire and North West)	1508	247 (16)
	Occupational medicine physicians	1560 <sup>2</sup>	92 <sup>2</sup>	• Faculty of Occupational Medicine (FOM); • Society of Occupational Medicine (SOM)	1268	134 (11)
	General practitioners	35,774 <sup>3</sup>	8100 <sup>3</sup>	• Royal College of General Practitioners (RCGP) faculties (North West Wales, North West England, Cumbria, Mersey, Humber, Yorkshire)	7893	205 (3)
Nurses	GP practice nurses	21,634 <sup>4</sup>	n/a	• UK Association of Respiratory Nurse Specialists (ARNS); • Primary Care Respiratory Society UK (PCRS-UK); • Society of Occupational Medicine (SOM)	2270	209 (9)
	Occupational health nurses	3,332 <sup>5</sup>	n/a			
	Clinical nurse specialists	11,790 <sup>6</sup>	n/a			

**Table 5.1. Overview of health professional groups with clinical responsibility for working-age asthmatics patients in the UK, showing response rates from sampled organisations.** <sup>1</sup>Royal College of Physicians, 2011; <sup>2</sup>Faculty of Occupational Medicine, 2011; <sup>3</sup>Centre for Workforce Intelligence, 2011; <sup>4</sup>Health and Social Care Information Centre, 2012; <sup>5</sup>Nursing and Midwifery Council, 2010; <sup>6</sup>Royal College of Nursing, 2012: this number refers to all clinical nurse specialists from any specialty, not just respiratory specialist nurses.

### 5.3.5 Data analysis

Descriptive analysis was undertaken on the whole dataset in order to describe the range of responses to each variable and examine the data for skewness. Data were categorized by role (nurse, doctor) and specialty (occupational health, general practice, general hospital medicine, occupational lung disease specialist) and cross-tabulated with response variables to identify any associations. Analysis of variance was undertaken using Kruskal-Wallis tests and significance testing was undertaken using chi-squared tests for binary data and Mann-Whitney U-tests for ordinal data. All analyses were considered significant at the 5% level and were reported with 95% confidence intervals.

Subsequent multivariate ordinal regression analyses were performed and examined for goodness of fit, where the outcome was considered to be participants' responses to the following question: *"I ask them about the effects of work on their asthma symptoms?"* Explanatory variables considered were specialty, role grade of doctor, self-reported motivation, perceived importance of a diagnosis, confidence in diagnosis of occupational asthma, experience managing cases, background knowledge, and awareness of guidelines.

For the optional case study descriptive analyses of doctors' responses were undertaken as well as cross-tabulations to test differences in responses between medical specialties. For responses to "grey cases" (type SS and type WW) a logistic regression analysis was performed with responses to the question *"Do you think that the diagnosis is occupational asthma?"* as the dependent variable. Explanatory variables examined were medical specialty, grade of doctor, work factors (strong or weak work history, strong or weak non-work history), confidence, knowledge and experience. All statistical analyses were undertaken using S.P.S.S. version 21 (IBM, Armonk, New York).

### 5.3.6 Ethical approval

Ethical approval for the study was granted by Heart of England NHS Foundation Trust (R&D Code: 2013013RM).

## 5.4 Results

### 5.4.1 Pilot phase

On test-retest reliability testing of 14 questionnaire items by 17 raters, there was categorical agreement in 8-16 cases (Table 5.2) with a mean percentage agreement of 74% (range 47-94). Question 9 (*“I follow occupational asthma guidelines when assessing a working-age asthmatic?”*) required a binary response (yes/no), all other questions required ordinal responses by Likert scale (strongly agree, agree, neither agree nor disagree, disagree, strongly disagree). All questions showed significant agreement using the Kappa statistic with Q1 and Q12 rated ‘very good’ (0.88-0.89), Q2, Q6, Q9 and Q11 rated ‘good’ (0.62-0.75), Q3, Q4, Q5, Q7, Q8, Q13 and Q14 rated ‘moderate’ (0.42-0.59), and Q10 rated ‘fair’ (0.29). There were no significant differences between pre- and post- pilot test scores for any questionnaire items using Wilcoxon rank sum tests.

	Agreement	Cohen kappa			Wilcoxon Rank Sum	
Variable	Number (%); n=17	Value	95% CI	Significance	Z-score	Significance
Question 1: I ask them about the nature of their work?	16 (94)	0.89	0.70 to 1.08	p<0.001	-1	p=0.32
Question 2: I ask them about the effects of work on their asthma symptoms?	13 (77)	0.62	0.29 to 0.95	p<0.001	-1.2	p=0.26
Question 3: I think it is important to recognize when work is the cause of asthma symptoms?	14 (82)	0.56	0.16 to 0.97	p=0.01	-1.7	p=0.08
Question 4: I feel confident to screen patients for occupational asthma based on their clinical history?	10 (59)	0.42	0.12 to 0.72	p=0.002	-0.4	p=0.7
Question 5: I have sufficient knowledge of patients' work practices to screen for occupational asthma?	11 (65)	0.52	0.20 to 0.84	p<0.001	-1.6	p=0.1
Question 6: I feel confident to make a diagnosis of occupational asthma?	13 (77)	0.69	0.43 to 0.95	p<0.001	-2	p=0.05
Question 7: I have access to specialist input for occupational asthma when I feel I need it?	12 (71)	0.46	0.09 to 0.83	p=0.02	-1.3	p=0.18
Question 8: I am aware of clinical guidelines for assessing patients with suspected occupational asthma?	11 (65)	0.49	0.21 to 0.77	p<0.001	-0.3	p=0.74
Question 9: I follow occupational asthma guidelines when assessing a working-age asthmatic?	14 (88) <sup>1</sup>	0.75	0.43 to 1.07	p=0.003	-0.6	p=0.56
Question 10: I lack the time to screen asthmatic patients for occupational asthma?	8 (47)	0.29	-0.03 to 0.61	p=0.04	-1.1	p=0.25
Question 11: I have no incentive to screen asthmatic patients for occupational asthma?	13 (77)	0.62	0.32 to 0.91	p<0.001	-0.38	p=0.71
Question 12: I am not motivated to screen patients for occupational asthma?	16 (94)	0.88	0.64 to 1.11	p<0.001	-1	p=0.32
Question 13: I have experience of managing cases of suspected occupational asthma in my clinical practice?	11 (65)	0.59	0.28 to 0.90	p<0.001	-0.7	p=0.48
Question 14: A diagnosis of occupational asthma can improve patient outcomes?	13 (77)	0.54	0.19 to 0.89	p=0.01	-2	p=0.05

**Table 5.2. Test-retest reliability of 14 questionnaire items based on percentage agreements, Cohen kappa statistics and Wilcoxon rank-sum tests.**

<sup>1</sup>n=16 due to one non-response. All analyses were considered significant at the 5% level.

#### 5.4.2 Demographics

12,958 healthcare professionals were sent a hyperlink to the questionnaire by e-mail. A breakdown of the total by role and specialty is shown in Table 5.1, along with response rates. Response rates were 3-89% amongst doctors and 9% for nurses. 603 participants completed the questionnaire (a further 142 participants started but did not complete enough demographic information or questions for analysis), of whom 394 (65%) were doctors and 209 (35%) were nurses. 234 responders were male (39%), and the mean number of years' experience was 19.8 years (SD=12). Of the doctors, 222 (56%) were consultants or GPs, and 172 (44%) were trainees. Demographics of responders, also grouped by specialty, are shown in Table 5.3.

			All participants; n=603	General Practice; <i>n=189 (31%)</i>	General Practice undertaking OH sessions; <i>n=16 (3%)</i>	Occupational Medicine; <i>n=134 (22%)</i>	General Hospital Medicine; <i>n=97 (16%)</i>	Respiratory Medicine; <i>n=150 (25%)</i>	Occupational Lung Disease specialists; <i>n=17 (3%)</i>
<u>Gender</u> ; <i>Number of males (%)</i>			234 (39)	39 (21)	11 (69)	88 (66)	37 (38)	47 (31)	12 (71)
<u>Role</u>	<u>Doctors</u> ;	Total; <i>Number (%)</i>	394 (65)	62 (33)	14 (87)	123 (92)	96 (99)	82 (55)	17 (100)
		Consultants; <i>Number (% of total doctors)</i>	222 (56)	54 (87)	13 (93)	112 (91)	6 (6)	20 (24)	17 (100)
		Trainees; <i>Number (% of total doctors)</i>	172 (44)	8 (13)	1 (7)	11 (9)	90 (94)	62 (76)	0 (0)
	<u>Nurses</u> ; <i>Number (%)</i>		209 (35)	127 (67)	2 (13)	11 (8)	1 (1)	68 (45)	0 (0)
<u>Experience</u> ; <i>Mean years (SD)</i>			19.8 (11.5); range 1-51	23.2 (10.1)	23.0 (7.9)	26.8 (8.3)	6.4 (5.8)	16.8 (10.4)	27.4 (11.4)

**Table 5.3. Demographics of participants completing the questionnaire, including data grouped by role and specialty. OH=occupational health.**

#### 5.4.3 Questionnaire responses by role

A summary of all Likert responses to questions Q1-14 is shown in Table 5.4.

573/603 (95%) of respondents strongly agreed (62%) or agreed (33%) with the question “*I ask them [patients] about the nature of their work?*” The mean statistic was 1.49 (SD=0.69) where 1=minimum (strongly agree) and 5=maximum (strongly disagree). Nurses were significantly more inclined to agree with the question (mean statistic=1.33; SD=0.54) than doctors (mean statistic=1.52; SD=0.75; Mann-Whitney U-test  $p=0.004$ ).

530/603 (88%) of respondents strongly agreed (51%) or agreed (37%) with the question “*I ask them about the effects of work on their asthma symptoms?*” and the mean statistic was 1.66 (SD=0.82). Again nurses were significantly more inclined to agree with the question (mean statistic=1.46; SD=0.6) than doctors (mean statistic=1.77; SD=0.89; Mann-Whitney U-test  $p<0.001$ ).

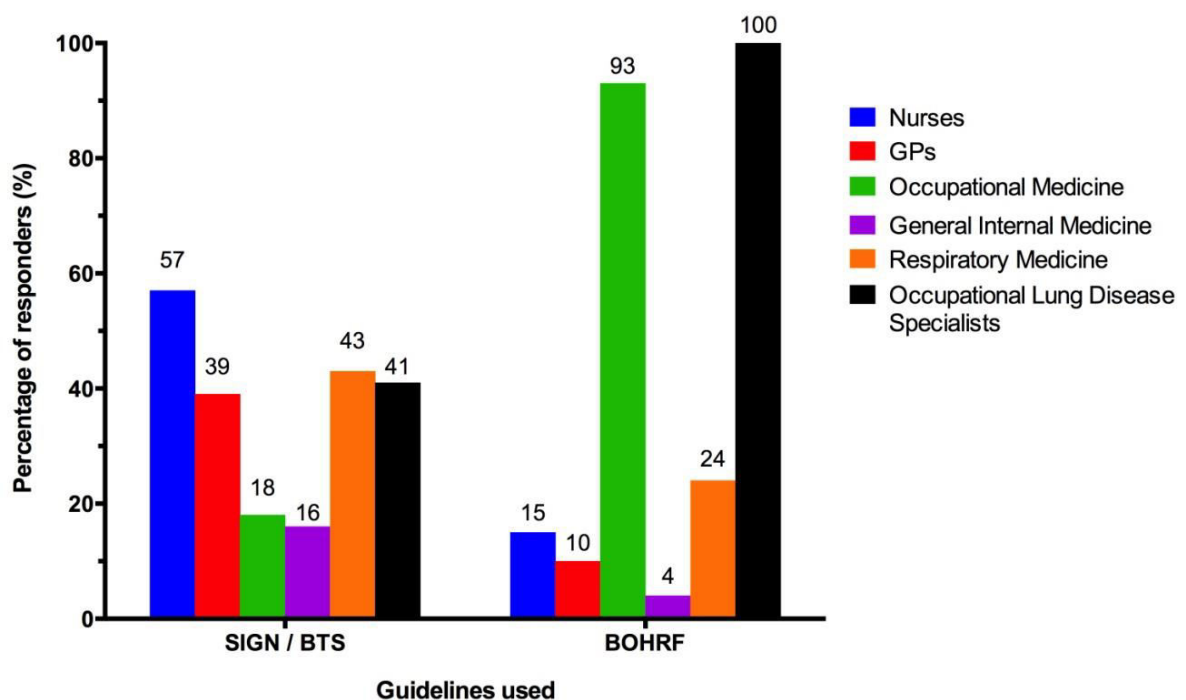
Nurses were less confident to screen for occupational asthma than doctors (mean statistic=2.33; SD=0.98 vs. mean statistic=2.0; SD=0.95; Mann-Whitney U-test  $p<0.001$ ), felt that they had less knowledge (mean statistic=2.62; SD=1.03 vs. mean statistic=2.28; SD=1.09;  $p<0.001$ ) and less time to screen (mean statistic=3.13; SD=1.03 vs. mean statistic=3.47; SD=1.06;  $p<0.001$ ) than doctors.

374/603 (62%) participants followed occupational asthma guidelines, and there was no significant difference between doctors (239/394; 61%) and nurses (135/209; 65%) when tested ( $X^2=0.99$ ;  $p=0.34$ ). 223/374 (60%) used the SIGN/BTS guidelines, 198/374 (53%) used the BOHRF guidelines, and only 47/374 (13%) used the ERS guidelines. Guideline use varied between medical specialty and this is illustrated in Figure 5.1.



	Question Likert responses					Mean statistic <sup>1</sup> (SD)
	<u>Strongly agree;</u> number of responses (%)	<u>Agree;</u> number of responses (%)	<u>Neither agree nor disagree;</u> number of responses (%)	<u>Disagree;</u> number of responses (%)	<u>Strongly disagree;</u> number of responses (%)	
Question 1: I ask them about the nature of their work?	374 (62)	199 (33)	16 (3)	11 (2)	3 (1)	<b>1.46 (0.69)</b>
Question 2: I ask them about the effects of work on their asthma symptoms?	307 (51)	223 (37)	46 (8)	25 (4)	2 (0)	<b>1.66 (0.82)</b>
Question 3: I think it is important to recognize when work is the cause of asthma symptoms?	456 (76)	145 (24)	2 (0)	0 (0)	0 (0)	<b>1.25 (0.44)</b>
Question 4: I feel confident to screen patients for occupational asthma based on their clinical history?	174 (29)	260 (43)	100 (17)	64 (11)	5 (1)	<b>2.11 (0.97)</b>
Question 5: I have sufficient knowledge of patients' work practices to screen for occupational asthma?	142 (23)	203 (34)	144 (24)	103 (17)	11 (2)	<b>2.4 (1.08)</b>
Question 6: I feel confident to make a diagnosis of occupational asthma?	105 (17)	208 (35)	139 (23)	131 (22)	20 (3)	<b>2.6 (1.11)</b>
Question 7: I have access to specialist input for occupational asthma when I feel I need it?	232 (29)	201 (33)	79 (13)	80 (13)	11 (2)	<b>2.07 (1.1)</b>
Question 8: I am aware of clinical guidelines for assessing patients with suspected occupational asthma?	192 (32)	227 (38)	65 (11)	106 (17)	13 (2)	<b>2.21 (1.13)</b>
<sup>2</sup> Question 10: I lack the time to screen asthmatic patients for occupational asthma?	20 (3)	138 (23)	127 (21)	244 (41)	74 (12)	<b>3.36 (1.06)</b>
Question 11: I have no incentive to screen asthmatic patients for occupational asthma?	11 (2)	46 (8)	94 (16)	256 (42)	195 (32)	<b>3.96 (0.97)</b>
Question 12: I am not motivated to screen patients for occupational asthma?	6 (1)	28 (4)	66 (11)	270 (45)	233 (39)	<b>4.15 (0.87)</b>
Question 13: I have experience of managing cases of suspected occupational asthma in my clinical practice?	112 (19)	220 (36)	94 (16)	138 (23)	37 (6)	<b>2.61 (1.2)</b>
Question 14: A diagnosis of occupational asthma can improve patient outcomes?	298 (49)	247 (41)	34 (6)	3 (1)	14 (2)	<b>1.64 (0.81)</b>

Table 5.4. Summary of Likert responses by all participants completing the questionnaire. <sup>1</sup>Mean statistic based on 1=minimum (strongly agree) and 5=maximum (strongly disagree). <sup>2</sup>Question 9 (“I follow occupational asthma guidelines when assessing a working-age asthmatic”) has a binary outcome (Yes/No).



**Figure 5.1. Occupational asthma guideline use by nurses and doctors completing the questionnaire. Responders are grouped by medical specialty and results are displayed as a percentage of each specialty group as a whole. GP=general practitioner; SIGN=Scottish Intercollegiate Guidelines network; BTS=British Thoracic Society (BTS/SIGN, 2012); BOHRF=British Occupational Health Research Foundation (Nicholson et al., 2010).**

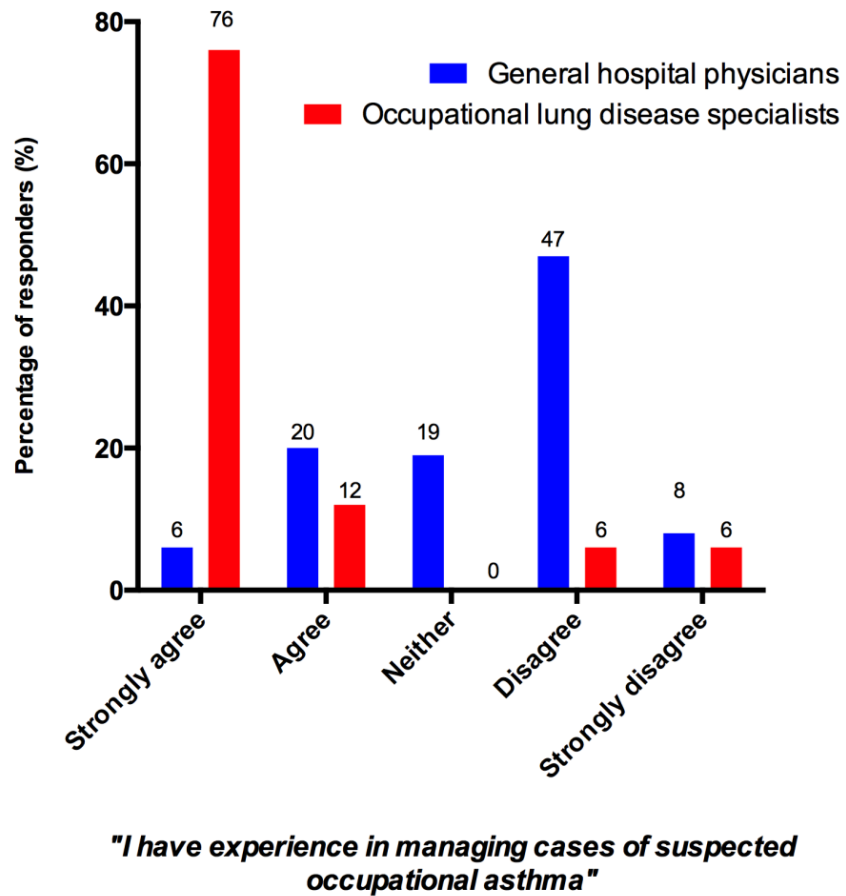
#### 5.4.4 Questionnaire responses by specialty group

Grouping both doctors and nurses by medical specialty highlighted differences in responses to the questions about the nature of work (Q1) and work effect on asthma symptoms (Q2; see Table 5.5). Using a comparison with occupational lung disease specialists as a reference group, only general hospital physicians enquired significantly less about the nature of work ( $p=0.03$ ) or work effect on asthma symptoms ( $p=0.03$ ). There was a significant variation in response across all nursing groups to enquiry about nature of work ( $p=0.03$ ) and work effect ( $p=0.02$ ), though no single group was different to another on hypothesis testing.

Appendix 8 shows responses to Q1-14 grouped by medical specialty, where there was a variation in the degree to which professionals felt confident to diagnose or screen occupational asthma, and in their self-reported knowledge, awareness of guidelines, access to specialist input, and clinical experience. Figure 5.2 illustrates the difference in response between general hospital physicians and occupational lung disease specialists to Q13 regarding clinical experience: general hospital physicians reported significantly less experience in managing cases of suspected occupational asthma than occupational lung disease specialists (Mann-Whitney U-test;  $p < 0.001$ ).

		Doctors							Nurses			
		General Practice; n=62	General Practice undertaking OH sessions; n=14	Occupational Medicine; n=123	General Hospital Medicine; n=96	Respiratory Medicine; n=82	Occupational Lung Disease specialists; n=17	Analysis of variance	Practice nurses; n=127	Occupational health nurses; n=11	Hospital respiratory nurse specialists; n=68	Analysis of variance
Asks about the nature of a patient's work	SA	21 (34)	8 (57)	109 (89)	24 (25)	50 (61)	17 (100)	p<0.001	81 (64)	9 (82)	55 (81)	p=0.03
	A	35 (57)	6 (43)	14 (11)	55 (57)	30 (37)	0 (0)		41 (32)	2 (18)	13 (19)	
	N	2 (3)	0 (0)	0 (0)	9 (9)	1 (1)	0 (0)		4 (3)	0 (0)	0 (0)	
	D	4 (6)	0 (0)	0 (0)	5 (5)	1 (1)	0 (0)		1 (1)	0 (0)	0 (0)	
	SD	0 (0)	0 (0)	0 (0)	3 (3)	0 (0)	0 (0)		0 (0)	0 (0)	0 (0)	
Asks about the effects of work a patient's asthma symptoms	SA	13 (21)	5 (36)	103 (84)	9 (9)	37 (45)	17 (100)	p<0.001	68 (54)	9 (82)	46 (68)	p=0.02
	A	35 (56)	8 (57)	19 (15)	44 (46)	40 (49)	0 (0)		50 (39)	2 (18)	22 (32)	
	N	8 (13)	1 (7)	1 (1)	24 (25)	4 (5)	0 (0)		8 (6)	0 (0)	0 (0)	
	D	6 (10)	0 (0)	0 (0)	17 (18)	1 (1)	0 (0)		1 (1)	0 (0)	0 (0)	
	SD	0 (0)	0 (0)	0 (0)	2 (2)	0 (0)	0 (0)		0 (0)	0 (0)	0 (0)	

**Table 5.5. Responses to questions regarding healthcare professionals' enquiry about (1) the nature of patients' work and (2) work-effect on their asthma symptoms. SA=strongly agree; A=agree; N=neither agree nor disagree; D=disagree; SD=strongly disagree. Analyses of variance measured using non-parametric Kruskal-Wallis tests, significance measured at the 95% confidence level.**



**Figure 5.2. Responses of general hospital physicians and occupational lung disease specialists to the question regarding clinical experience (Q13). General hospital physicians reported significantly less experience in managing cases of suspected occupational asthma (Mann-Whitney U-test;  $p < 0.001$ ).**

#### 5.4.5 Ordinal regression analysis

The ordinal regression analysis of doctors' Likert responses to the question "*Do you ask about the effect of work on asthma symptoms?*" is shown in Table 5.6. The model was a good fit ( $-2\log L$  final-model=539.6) with a significant improvement over the intercept-only model ( $-2\log L$ =873.8;  $p<0.001$ ) and Nagelkerke  $r^2=0.69$ . Being a physician in any specialty other than general hospital medicine increased the likelihood of asking about work effect on symptoms (unadjusted OR=3.03; 95% CI=1.36 to 6.77) but no other factors had a significant effect. Confidence in screening for occupational asthma was associated with greater odds of asking about work effect (unadjusted ORs=49.35-96.64) when compared with the baseline answer of "strongly disagree" though this did not reach statistical significance.

Explanatory variable		Odds Ratio	Lower 95% CL	Upper 95% CL	p-value
Specialty	<i>Non-GHM</i> <i>GHM</i>	3.03 1	1.36	6.77	0.007
Level of training	<i>Foundation year/SHO</i> <i>Registrar level</i> <i>Consultant/GP level</i>	0.66 0.89 1	0.26 0.48	1.63 1.62	0.36 0.68
Self-reported experience in managing cases of occupational asthma	<i>SA</i> <i>A</i> <i>N</i> <i>D</i> <i>SD</i>	1.11 0.95 1.07 0.91 1	0.26 0.29 0.31 0.28	4.70 3.07 3.74 2.91	0.88 0.93 0.91 0.87
Belief that a diagnosis of occupational asthma improves outcomes	<i>SA</i> <i>A</i> <i>N</i> <i>SD</i>	0.57 0.67 0.25 1	0.05 0.06 0.02	6.47 7.68 3.48	0.65 0.75 0.30
Perceived occupational asthma as being important	<i>SA</i> <i>A</i> <i>N</i>	7.58 1.95 1	0.39 0.10	146.22 37.22	0.18 0.66
Confident to screen for occupational asthma	<i>SA</i> <i>A</i> <i>N</i> <i>D</i> <i>SD</i>	96.64 56.26 95.58 49.35 1	0.91 0.58 1.03 0.54	10259.92 5458.89 8883.94 4500.75	0.06 0.08 0.05 0.09
Sufficient knowledge to screen for occupational asthma	<i>SA</i> <i>A</i> <i>N</i> <i>D</i> <i>SD</i>	4.43 1.22 0.57 0.55 1	0.12 0.04 0.02 0.02	158.54 36.63 16.46 15.23	0.41 0.91 0.74 0.73
Confident to diagnose occupational asthma	<i>SA</i> <i>A</i> <i>N</i> <i>D</i> <i>SD</i>	1.08 1.72 1.05 1.67 1	0.03 0.05 0.05 0.05	43.68 60.52 58.56 54.54	0.97 0.77 0.78 0.77
Aware of occupational asthma guidelines	<i>SA</i> <i>A</i> <i>N</i> <i>D</i> <i>SD</i>	6.27 2.99 1.54 1.07 1	0.97 0.53 0.26 0.20	40.45 16.95 9.16 5.81	0.05 0.21 0.63 0.93
No incentive to screen for occupational asthma guidelines	<i>SA</i> <i>A</i> <i>N</i> <i>D</i> <i>SD</i>	1.11 0.92 0.50 0.40 1	0.16 0.14 0.07 0.05	7.83 6.11 3.55 2.91	0.91 0.93 0.49 0.36
Lacks time to screen for occupational asthma	<i>SA</i> <i>A</i> <i>N</i> <i>D</i> <i>SD</i>	1.76 2.34 1.34 1.35 1	0.35 0.64 0.36 0.37	8.78 8.62 5.01 4.98	0.49 0.20 0.66 0.65

**Table 5.6. Ordinal regression analysis of factors associated with likelihood of agreement (by Likert response) with the question “Do you ask [them] about the effect of work on [their] asthma symptoms?” with unadjusted odds ratios and 95% confidence limits. CL=confidence limit; GHM=general hospital medicine; SHO=senior house officer; GP=general practitioner; SA=strongly agree; A=agree; N=neither agree nor disagree; D=disagree; SD=strongly disagree.**

#### 5.4.6 Questionnaire open comment

Several themes were identified from analysis of open comments at the end of the questionnaire:

(1) Lack of knowledge and skill. Some participants highlighted their own lack of skill and knowledge in assessing asthmatics for occupational asthma: a GP: *“I always consider this though feel lacking in skills to accurately diagnose and am unaware of specific guidelines to know whether I follow these”*; a respiratory physician: *“I am unaware that there are specific guidelines related to occupational asthma and hence do not apply them in clinical practice”*; an occupational medicine physician: *“in reality, I struggle to see enough cases of constitutional asthma to keep my clinical confidence and knowledge up to date, let alone in relation to the rarer cases with a work related or occupational link”*.

(2) Retained basic skills only and would refer on when necessary. Some healthcare professionals felt that their practice was appropriate to their level of expertise: a GP: *“I might not read guidelines in detail but follow the appropriate approach. There are levels of assessment and I do a level appropriate to my generalist background”*; some felt that screening for occupational asthma was not in their domain of expertise: a general hospital physician: *“I work on acute admissions, I would request a respiratory review if I felt occupation was leading to problems”*; some felt that they were limited by a lack of resources required to do a detailed investigation: a GP: *“making a diagnosis is difficult as I do not have resources in my clinics to offer peak flow diaries and analysis of results - these presentations are quite rare and so I would simply refer to a specialist based on the clinical history of work exacerbated symptoms”*.

(3) Clinical experience. General hospital physicians lacked clinical experience in managing suspected occupational asthma, and described (i) engaging asthmatics only during acute admissions where emergency treatments were prioritized over further investigation for work-



causes: “patients mainly seen as acute exacerbation of asthma as medical registrar on call - therefore [I’m] more focused on acute management of known asthmatics rather than new diagnosis”, (ii) having never seen a case of occupational asthma: “I have received teaching on occupational asthma so I am aware of it but I have not diagnosed it before”, and (iii) lacking time for assessment while on-call.

(4) Clinical experience amongst GPs with extended roles. Comments concerning examples of good practice were mainly by individuals with an interest in asthma, for example a local community asthma clinical lead, a GP undertaking occupational medicine sessions, and a GP with a PhD in occupational asthma in the 1990s: “I completed my MD thesis as a GP on a new cause of occupational asthma... since then I have diagnosed several other people with that knowledge... My GP partners and nurse are very aware of lung irritants and sensitizers following my work”.

#### 5.4.7 Responses to case study

298 doctors completed the case study of whom 126/298 (42%) were trainees and 172/298 (58%) were consultant or GP level doctors. The numbers of scenarios allocated were as follows: SW=67, SS=73, WW=80 and WS=78. 52% (156/298) of all responders thought that their case represented occupational asthma, and there were no significant differences when overall response was grouped by medical specialty (Kruskal-Wallis  $p=0.55$ ). Table 5.7 shows the responses for each scenario type (SW, SS, WW, WS) grouped by medical specialty. Decreasing proportions of responders believed their case to represent occupational asthma moving through the scenarios, though 82% of occupational physicians designated their WS scenario as occupational asthma.

Scenario	<i>All doctors; n (% of all allocated to scenario)</i>	GPs; n=45	GPs undertaking OH sessions; n=11	Occupational Medicine; n=101	General hospital medicine; n=68	Respiratory medicine; n=17	Occupational lung disease specialists; n=17
<b>SW</b>	59 (88)	6 (100)	3 (75)	23 (92)	13 (93)	10 (77)	4 (80)
<b>SS</b>	66 (90)	14 (93)	2 (100)	19 (100)	18 (95)	11 (73)	2 (67)
<b>WW</b>	18 (23)	5 (42)	2 (50)	5 (18)	4 (20)	2 (14)	0 (0)
<b>WS</b>	13 (17)	3 (25)	0 (0)	23 (82)	2 (13)	4 (29)	1 (14)

**Table 5.7. Number of responders reporting that each scenario was occupational asthma, grouped by medical specialty. Where percentages are given the denominator is the number of doctors from a specialist group who have been allocated to that particular scenario. GP=general practitioners; OH=occupational health; SW=Strong work/weak non-work factors; SS=strong work/strong non-work; WW=weak work/weak non-work; WS=weak work/strong non-work.**

146/298 responders (49%) would refer the case to an occupational lung disease specialist, and no differences were observed between medical specialty groups (Kruskal-Wallis  $p=0.36$ ) or level of training (Kruskal-Wallis  $p=0.79$ ). The majority of responders were confident in their diagnosis (very confident 31/298 (10%), confident 145/298 (49%), somewhat confident 98/298 (33%), unsure 22/298 (7%), very unsure 2/298 (1%), and no differences between medical specialty groups (Kruskal-Wallis  $p=0.44$ ) or level of training (Kruskal Wallis  $p=0.44$ ) were observed.

When deciding whether a case was occupational asthma or not, the timing of onset of symptoms (mean statistic=4.7; SD=0.57) and understanding the nature of the patient's work (mean statistic=4.6; SD=0.58) [where 1=minimum (not at all important) and 5=maximum (extremely important)] were more important than the patient's belief about work-relatedness,

presence/absence of non-work related risk factors, and whether other workers were affected. Factors contributing to decision-making are shown in Appendix 9, and grouped by medical specialty.

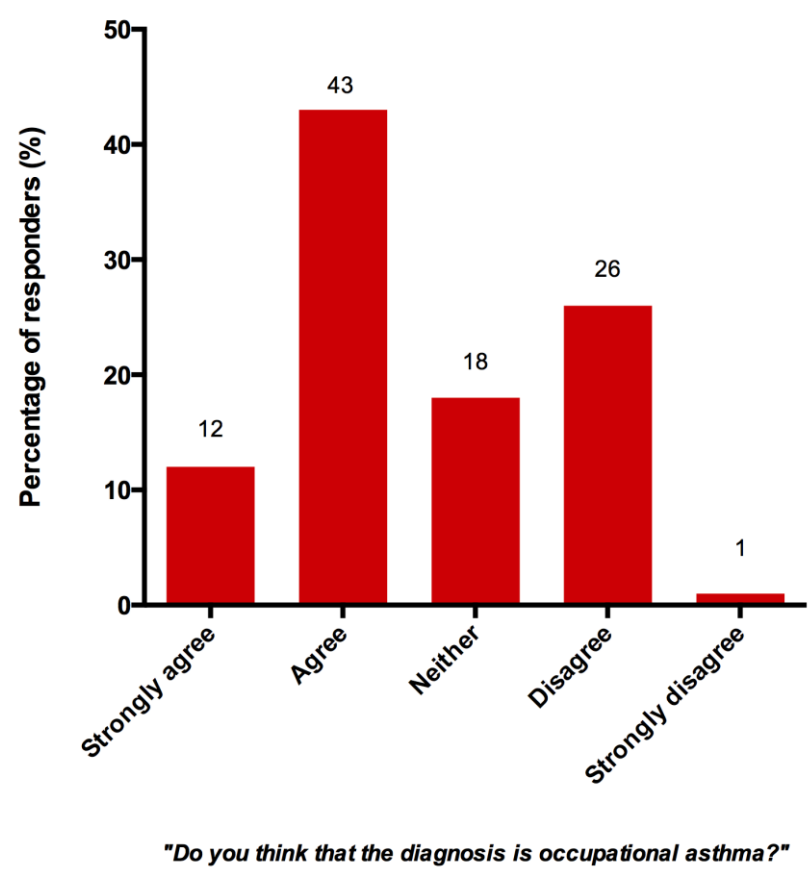
#### 5.4.8 Case study open comments

An open-ended comments section, intended to ascertain whether any further information would be desirable for decision-making, revealed the following: 9 responders (8 occupational medicine physicians, 1 occupational lung disease specialist) wanted to obtain a history relating to cleaning agent exposure and/or safety-data sheets from cleaning agents in use; 19 responders (5 GPs, 10 occupational physicians, 4 respiratory physicians) requested serial peak flow measurements; 3 responders (2 occupational physicians, 1 GP) requested more information about the smoking history; 5 responders (2 respiratory physician, 3 occupational physicians) requested serum-specific immunology or skin prick testing to flour/enzymes, and 2 occupational physicians wanted to undertake workplace visits.

#### 5.4.9 Grey case scenarios (SS, WW)

The 153 responses to grey case scenarios (either SS or WW) were analyzed separately, in order to determine the main factors that determine how doctors allocate causation when faced with diagnostic uncertainty. Likert responses to the question “*Do you think that the diagnosis is occupational asthma?*” were transformed into the binary outcomes ‘yes’ (strongly agree/agree) and ‘no’ (neither agree nor disagree/disagree/strongly disagree) as a bimodal frequency distribution was seen in response to this question (shown in Figure 5.3). The binary logistic regression analysis is shown in Table 5.8. The model was a good fit (-2logL=113.5 intercept-only model;  $\chi^2=97.13$ ;  $p<0.001$ ) and Nagelkerke  $r^2=0.63$ . Allocation of a diagnosis of occupational asthma to a grey case was influenced by being a GP (unadjusted OR=47.2; 95% CI=2.3 to 967.18) or a GP with session in occupational health (unadjusted OR=84.18; 95% CI=2.7 to 2627.53), and also by strong work factors in the

medical history (unadjusted OR=66.49; 95% CI=18.98 to 232.94), but not by strong non-work factors.



**Figure 5.3.** Bimodal distribution of responses to the question “*Do you think that the diagnosis is occupational asthma?*” for grey case scenarios SS and WW. 153 participants were allocated grey-case scenarios. SS=strong work/strong non-work; WW=weak work/weak non-work.

Explanatory variable		Odds Ratio	Lower 95% CL	Upper 95% CL	p-value
Specialty	GP	47.20	2.30	967.18	0.01
	GP-OH	84.18	2.70	2627.53	0.01
	Occupational medicine	12.97	0.88	190.46	0.06
	General hospital medicine	33.64	0.97	1169.56	0.05
	Respiratory medicine	8.55	0.35	207.10	0.19
	Occupational Lung Disease	1			
Level of training	Foundation year/SHO	0.34	0.02	4.84	0.43
	Registrar	0.64	0.09	4.44	0.65
	Consultant/GP	1			
Work factors	Strong work	66.49	18.98	232.94	<0.001
	Weak work	1			
Non-work factors	Strong non-work	1			
	Weak non-work	1			
Self-reported confidence in diagnosing occupational asthma	SA	0.42	0.04	4.79	0.48
	A	0.62	0.09	4.17	0.63
	N	0.97	0.17	5.60	0.98
	D	1			
Experience in managing cases of occupational asthma	SA	0.31	0.03	3.78	0.36
	A	0.18	0.02	1.67	0.13
	N	0.38	0.04	3.82	0.41
	D	0.42	0.05	3.70	0.43
	SD	1			
Sufficient knowledge to screen for occupational asthma	SA <sup>1</sup>	1			
	A	0.45	0.08	2.57	0.37
	N	0.14	0.02	1.27	0.08
	D	0.20	0.02	2.37	0.20

**Table 5.8. Binary logistic regression analysis of yes/no responses to the question “Do you think that the diagnosis is occupational asthma?” with unadjusted odds ratios and 95% confidence limits. CL=confidence limit; GP=general practitioner; GP-OH=general practitioner with session in occupational health; SHO=senior house officer; SA=strongly agree; A=agree; N=neither agree nor disagree; D=disagree; SD=strongly disagree.**

<sup>1</sup>“SA” value used as baseline variable due to small number of responses (n=1) to “SD”.

## 5.5 Discussion

### 5.5.1 Summary

This study aimed to define the important barriers to identifying occupational asthma from the point of view of healthcare professionals (nurses and doctors) who attend working-age asthmatic patients in their clinical practice. Response rates from most professional groups were disappointingly low, which introduces a significant non-response bias into the gathered data, and limits the strength of their interpretation.

Most questionnaire responders asked asthmatic patients about the nature of their work (95%) and the effect of work on their asthma symptoms (88%). Nurses were more inclined to do so than doctors, but were less confident, less knowledgeable, with less time to screen for occupational asthma. The only medical specialty who enquired less about work and work-effect than occupational lung disease specialists, were general hospital physicians, who were 3 times less likely to enquire than other groups, and also reported a lack of experience managing cases of occupational asthma, less subject knowledge and awareness of guidelines, and lower perceived importance of the subject matter. 62% of all responders followed occupational asthma guidelines, mainly SIGN/BTS (60%) and BOHRF (53%) guidance, though use varied between medical specialties (4-100%).

In this sample of survey responders there was no difference in the allocation of causation to the different scenarios, or confidence in that allocation, by medical specialty. Timing of the onset of symptoms and the nature of patients' work were the most important factors in deciding whether a case was occupational or not. In cases of diagnostic uncertainty (grey cases) GPs and GPs with sessions in occupational health were more likely than other groups to describe a case as occupational asthma, and strong work-factors were more important in that decision than the absence or presence of non-work factors in the history.

### 5.5.2 Limitations

This is the first study that has aimed to understand barriers to diagnosis of occupational asthma on the part of a variety of UK healthcare professionals in detail. However it has a number of limitations that require further discussion. It is necessary initially to consider whether all of the important barriers to diagnosis were included in the questionnaire: barriers were identified from two Canadian cross-sectional studies – a telephone interview study of 42 workers with asthma symptoms (Poonai et al., 2005) and a postal questionnaire of 201 Canadian pulmonologists (Parhar et al., 2011). In order to increase the sensitivity of the questions included, ideas generated from the Canadian studies were augmented by discussion with 2 occupational lung disease clinical experts, and by review of a cross-sectional survey of Dutch GPs with regard to guideline adherence (Lugtenburg et al., 2011). Furthermore, an evaluation of an educational module for GPs on occupational asthma, from Sheffield, which is to date the only UK-based study to have addressed barriers to diagnosis, highlighted deficiencies in background knowledge and clinical experience in primary care (Barber et al., 2010); both of these aspects were addressed in the questionnaire.

Guidance was followed in order to construct and sequence questionnaire items (Williams, 2003) and avoid the cognitive biases associated with surveys. The questionnaire was then piloted to establish construct validity and test-re-test reliability, which showed all items to be at least “fair” or greater using the Cohen kappa statistic (Field, 2004; Landis and Koch, 1977). A number of strategies adapted from the Dillman method (Dillman, 2007) were employed to increase response rate, including (1) using Likert responses (Bowling, 1997), which are quick to complete, easy to code and avoid non-committal (“don’t know”) answers (Williams, 2003), (2) using an Internet based survey tool, which has fewer steps to completion, gathers and codes data automatically, and is more cost-effective than a postal questionnaire (Rosenbaum and Lidz, 2007), and (3) using e-mail reminders 4-weeks after the initial contact. However despite these strategies response rates varied and were very low for most groups: GPs (3%),

respiratory-oriented nurses (9%), occupational physicians (11%) and hospital physicians (16%).

One might expect a low response to Internet-based questionnaires; certainly postal questionnaires are associated with lower response rates than interview-led ones (Cartwright 1986). In general practice a number of reasons for increasing lack of engagement with surveys have been cited, including increasing workload and stress, hostility from service providers towards academic general practice, disinterest in research, and perceived threats to professional activity (McDonald, 1993). Furthermore GPs will not complete studies if they disagree with, or are not interested in, the research topic (Cartwright, 1978; Herbelein and Baumgartner, 1978; Sibbald et al., 1994). However, low response rates do not necessarily affect the validity of questionnaire data, particularly when the target population is a group with homogenous opinions, attitudes or demographics (Leslie, 1972). Indeed in studies that have evaluated research questionnaires distributed to GPs, the demographics and disease-related beliefs of non- or late responder GPs did not differ significantly from early responders (Cockburn et al., 1988; Bostick et al., 1992). Nonetheless the response rates in the current study are disappointing and much lower than a pooled estimate for published questionnaire studies (mean response rate=57.5%, 95%CI: 55.2% to 59.8%; Cook et al., 2009): therefore there is likely to be significant non-response bias. This probably manifests as a bias towards interested or motivated individuals with experience of managing cases of work-related respiratory diseases, as reflected in both the open comments section and the fact that GPs were more inclined to call a grey case occupational asthma than other professionals. With the exception of general hospital physicians and notwithstanding the fact that trainees were well represented in the sample (44%), responders tended to be experienced nurses and doctors (mean number of years' clinical experience across specialties=16.8-27.4).



### 5.5.3 Interpretation of findings

Results would indicate that most healthcare professionals ask about the nature of work and work effect regardless of role or specialty, and to the same extent as a group of occupational lung disease specialists. Equally with the case study, physicians across all specialties allocate work causation to the same extent and with the same degree of confidence as occupational lung disease specialists (with the exception of GPs with grey cases). Despite this, non-occupational lung disease specialists appeared to display lower confidence in diagnosing and screening for occupational asthma, and specific guideline use was low amongst GPs (10-39%), general hospital physicians (4-16%) and respiratory physicians (24-43%). Therefore it is perhaps surprising that non-specialists report high levels of screening activity, and there was such homogeneity of response. This certainly does not reflect established data from a range of other sources: (1) inactivity and low prevalence of recorded occupational asthma in the primary care audit (Walters et al., 2012; chapter 3), (2) the inadequate healthcare consultations experienced by the majority of interviewed workers with occupational asthma symptoms (chapter 4), (3) previous reports of low identification rates (de Bono et al., 1999) and low- or delayed referral activity in UK primary care (Hoyle et al., 2010; Fishwick et al., 2007), and (4) poor enquiry by attending doctors seen in two Canadian studies (Poonai et al., 2005; Parhar et al., 2011). Therefore a significant proportion of the responses to questions about screening should be explained by non-responder bias, and by cognitive biases (response bias) associated with answering “suggestive” questionnaire items. Indeed, as previously stated, there is further evidence of this in the enthusiastic allocation of work causation to grey cases by GPs and GPs with an interest in occupational health.

It is not surprising that general hospital physicians asked less about work and work effect, since the group was made up of mostly trainees (94%) with less average clinical experience (6.4 years) than other specialties. Respiratory medicine doctors comprised a high percentage of trainees (76%) but one would expect them to have greater clinical experience and subject knowledge, since occupational lung diseases form a significant part of their training

curriculum in the UK (Joint Royal Colleges of Physicians' Training Boards, 2010). Lack of background knowledge and skill amongst general medicine trainees is reflected in open comments that suggest they retain very little subject knowledge and would expect to seek specialist help for screening. In the case of nurses, all those sampled would be expected to have an interest and clinical experience in respiratory disease, due to the nature of the organizations recruited. Practice nurses who attend adult asthmatics were a difficult group to target and reach in their entirety, and this necessitated contacting specialist respiratory groups, which meant sampling individuals motivated enough to join a professional society. Therefore one might anticipate high levels of enquiry about work and work effect, which probably overestimates the performance of practice nurses generally. This is notwithstanding the relatively lower self-reported confidence in screening, background knowledge and time available to screen, when compared to doctors.

## 5.6 Conclusion

In the sample of responders included in this study, GPs, non-specialist physicians and nurses report high levels of patient enquiry about the nature of work and work effect on their asthma symptoms; however this is likely to overestimate practice generally due to an over-representation of enthusiasts in the sample. However, low awareness and adherence to guidelines is reported amongst non-specialists, which better reflects other data suggesting that there is poor enquiry about occupational asthma, particularly in primary care. A group represented predominantly by trainee general physicians report less screening activity, confidence, awareness of guidelines and background knowledge than other specialties. Therefore there is some evidence of low knowledge, guideline use and clinical experience of screening for occupational asthma amongst non-specialists.

## CHAPTER 6: FEASIBILITY STUDY OF A PRIMARY CARE SCREENING TOOL FOR OCCUPATIONAL ASTHMA

### 6.1 Introduction

#### 6.1.1 Background

Occupational asthma is associated with a societal cost to the UK of £100 million per annum for each year of incident cases, and most of the financial burden falls on the taxpayer and the employee rather than the employer (Ayres et al., 2011). This cost is somewhat avoidable since those individuals with a short latency between symptom onset and diagnosis or removal from exposure to a sensitizing agent have a better prognosis in terms of lung function and quality of life (Nicholson et al., 2010). However, in cohort studies from the UK and from Canada, mean delays of 3-4 years between symptom onset and diagnosis or referral to a specialist have been identified (Fishwick et al., 2007; Santos et al., 2007; Poonai et al., 2005). There is evidence that primary healthcare professionals (GPs and practice nurses) do not ask asthmatic patients about the effect of work on their symptoms (Poonai et al., 2005; Fishwick et al., 2007; Walters et al., 2012) or take action when there is a high probability of work causation (Hoyle et al., 2010; de Bono and Hudsmith, 1999). This is notwithstanding the publication of guidance for physicians, GPs and primary care nurses on assessment of patients with suspected occupational asthma (BTS, 2012; BOHRF, 2010): these suggest that all working-age patients with new onset or re-activated asthma symptoms should be asked their occupation (to highlight high-risk work) and whether the symptoms are related to their work (Fishwick et al., 2008; Nicholson et al., 2010). Those patients whose symptoms are work-related or concur with a high-risk occupation should be referred for specialist assessment (Fishwick et al., 2012).

#### 6.1.2 Efficacy of screening questions for occupational asthma

Two simple questions (“Are your symptoms better on days away from work?” and “Are your symptoms better on holiday?”) have a sensitivity of between 58-100% in identifying

occupational asthma, sensitivity being below 90% in only two studies (Malo et al., 1991; Vandenplas et al., 2005). Specificity can be lower however (45-100%), being over 70% in one small study only (Nicholson et al., 2010). UK guidelines recommend that these two questions be used for screening in primary care and elsewhere (SIGN/BTS, 2012; Fishwick et al, 2012) with subsequent referral to a specialist if either question is answered in the affirmative.

The positive predictive value of a screening or diagnostic test is the probability that an individual with a positive test result actually has the disease (Altman and Bland, 1994). Positive predictive value is affected by the baseline prevalence of the disease in a population of interest; thus, the same diagnostic test will have a different predictive accuracy according to the population in which it is tested. Screening questions for occupational asthma have been validated in tertiary clinic populations with high disease prevalence, and have been demonstrated to have relatively low specificity (45-70% in all but one study). Therefore when applied to a population of working-age asthmatics in primary care with low baseline prevalence (0.3% using data from chapter 3) they may lead to a large number of false positive tests (Altman and Bland, 1994). The efficacy of these screening questions in a primary care population has not been demonstrated, nor is it known whether they are clinically effective in terms of case identification, reducing diagnostic latency and improving health outcomes.

### 6.1.3 Aim

The aims of this study were to evaluate the feasibility of initiating an electronic screening tool for primary healthcare professionals, its utility in collecting data on work-effect and the willingness of individual healthcare workers to use it.

## 6.2 Methods

A prospective feasibility study was undertaken over a 3-month period in 4 primary care practices in Birmingham, UK.

### 6.2.1 Practice recruitment

Four practices were recruited purposively by the primary author by direct approach to a group of Birmingham GPs who had recently collaborated with Heart of England NHS Foundation Trust respiratory physicians on commissioning respiratory services in the region, or worked in secondary care respiratory medicine. There were no specific inclusion criteria for individual patients since recruitment was at the cluster (practice) level. All 4 practices were undertaking asthma reviews with working-age patients, both formally as part of the Quality Outcomes Framework (NHS Information Centre, 2011) and opportunistically, for patients with urgent appointments due to symptomatic asthma, medication reviews, or at consultations for other medical problems.

### 6.2.2 Asthma template modification

Recruited practices used different electronic health records [Vision (INPS, Battersea, London, UK): practice A and B, SystmOne [sic] (TPP, Horsforth, Leeds, UK): practice C, EMIS (Egton Medical Information Systems, Yeadon, Leeds, UK): practice D]: each practice running a customized electronic template for asthma reviews, where data were captured using searchable Read codes. Practices were required to modify their existing templates to embed a screening tool consisting of the following questions:

1. What is your occupation?
2. Are your asthma symptoms better on days away from work?
3. Are your asthma symptoms better away from work on holiday?

A statement about the efficacy of the questions was included in the template with the questions (see Figure 6.1) but no further training or incentives were provided for staff. The lead GPs from each practice were instructed to alert all practice staff to the template changes, though only 2 did so (Practices A and D). Each practice was given the telephone and email contact details of the primary author to enable clinical advice from the Heart of England NHS

Foundation Trust occupational lung disease unit if required during use of the tool. As this was a feasibility study, no clinical outcomes for individual patients were measured. The study ran for 3 months from the start date and data were collected at the end of that period.

### 6.2.3 Data gathering

Baseline practice-level data were gathered including: (i) list-size, (ii) number of Read-coded asthmatics, (iii) number of asthmatics of working-age (16-64 year old), and (iv) current prevalence of Read-coded occupational asthma. At the end of the study period all exposed healthcare professionals (GPs, practice nurses) were invited to complete an online questionnaire intended to evaluate utility and willingness to use the tool. The online questionnaire was administered through the cloud-based forms creator [www.surveymonkey.com](http://www.surveymonkey.com) (Palo Alto, California, USA). Questionnaire items were developed through discussion with occupational lung disease specialists, 5-point Likert ordinal scales (strongly agree, agree, neither agree nor disagree, disagree, strongly disagree) were used for closed responses (Bowling, 1997) and opportunities for open comments were also included. The questionnaire is shown in Appendix 10.

### 6.2.4 Data analysis

Baseline practice-level data were tabulated for comparison between practices. Descriptive analyses were undertaken using questionnaire data. Data were categorized by role (doctor, nurse) and cross-tabulated with response variables to identify any associations. Analysis of variance was undertaken using Kruskal-Wallis tests for ordinal data. All analyses were considered significant at the 5% level.

### 6.2.5 Ethical approval

Ethical approval for the study was granted by the Yorkshire and The Humber - South Yorkshire National Research Ethics Service (NRES) committee (REC reference number: 13/YH/0291) and NHS Assurance for Research in Primary Care was granted by Birmingham

and the Black Country Comprehensive Local Research Network (Consortium Reference: consortium 372.129572).

## 6.3 Results

### 6.3.1 Practice level data

Practice-level data from each of the 4 primary care practices are shown in Table 6.1. List sizes varied between 3,475 (small practice with 4 GPs) and 24,660 (large practice with 3 separate clinical sites) and asthma prevalence was 5.6-8.2% at all practices. Across all 4 practices 56.3-65.2% of asthmatics were of working age, amongst whom the prevalence of Read-coded occupational asthma was 0-0.7%.

	Practice A	Practice B	Practice C	Practice D
Location	Small Heath	Small Heath	Harborne	Moseley
List size	16,019	11,193	24,660 <sup>1</sup>	3,475
Number (and prevalence %) of Read-coded asthmatics	966 (6)	919 (8)	1383 (6)	248 (7)
Number (and % of all asthmatics) of Read-coded asthmatics of working-age	544 (56)	599 (65)	879 (64)	152 (61)
Number (and prevalence % amongst working-age asthmatics) of Read-coded occupational asthma	4 (0.7)	3 (0.5)	3 (0.3)	0 (0)

**Table 6.1. Practice-level data gathered from each of 4 participating primary care practices. <sup>1</sup>Across 3 clinical sites.**

### 6.3.2 Feasibility of initiating screening tool

A GP from Practice A with background clinical experience in occupational lung disease created two buttons within the practice's existing asthma review template for acknowledging where an occupational enquiry had been undertaken (current employment) and questions regarding work effect on symptoms had been asked, together with opportunities for recording

responses in free text entry (shown in Figure 6.1). Any recorded data then appeared on a patient's electronic health record and was automatically Read coded making it auditable via a specific database search. Twelve GPs and 7 practice nurses at Practice A undertook adult asthma reviews and were informed of the template modification by the lead GP through opportunistic face to face discussion and by email circulation.

Consultation Summary Guidelines Add List Tasks Vision+ View Window Help

Initial Filter

- 17 Problems
- 314 Consultation
- 2 Drug Allergies & Adverse Re
- 4 Recalls and Reviews
- 2 Patient Preference
- 117 Medical History
- 44 Therapy
- 12 Lifestyle
- 13 Examination Findings
- 4 Immunisations
- 39 Miscellaneous
- 19 All Test Results
- New Registration Exam
- 20 Maternity
- Well Person Clinic
- 5 HP Interventions
- 2 Elderly
- 2 Disease Registers

Health promotion

- Clinical information missing
- Interventions not recorded

Current Recalls

Immunisations Due in Ne...

- Influenza 30/09/2012 o/d
- Polio/yellitis 1st 01/03/1998 o/d
- Rubella 1st 01/01/1999 o/d
- Tetanus ++ 01/01/2002 o/d

Current Actions

- Notes Summarised
- OMNIA COPD MONITORING
- At Risk of Osteoporosis
- Diabetes Mellitus
- Lower Limb Fractures
- Upper Limb Fractures

OMNIA ASTHMA MONITORING

Asthma Disturbs SLEEP Asthma NOT disturbing SLEEP

2- Have you had your usual symptoms during the day ( Cough, wheeze, chest tightness or breathlessness)?

Asthma daytime SYMPTOMS Asthma NEVER causes daytime SYMPTOMS

3- Has your asthma interfered with your activities ( housework, work, school, sports social etc)?

Asthma limiting ACTIVITIES Asthma NOT limiting ACTIVITIES

**Asthma and Work**

Some patients asthma is made worse by their jobs . Ask the patient about their occupation and specifically the effect of

You can do this by asking the following 2 work questions

" Are your asthma symptoms better AWAY from work , on days off ?"

" Are your asthma symptoms better Away from work, on holiday ? "

( enter response in free text using buttons below )

Patients Occupation Effect of work on symptoms School

11/05/2006 Occupations GU Nurse

**Written Asthma Management Plan**

Under Development

Asthma Management Plan

No data recorded.

**QOF Marker of Asthma Review - Confirms and Summarises Asthma Revi**

**Figure 6.1. Screenshot of occupational asthma screening tool incorporated into the existing asthma review template on a Vision dummy patient healthcare record at Practice A. This is the screen image seen by a practice healthcare professional when undertaking an asthma review. When a grey button marked “Patients [sic] Occupation” or “Effect of work on symptoms” is pressed each patient is labelled with an auditable Read code. Free text can then be entered underneath to clarify the responses - the example occupation shown here is a “GU nurse”. Once entered this information appears on a patient’s health record.**



A GP with no direct clinical experience in occupational lung disease at Practice B made the same modifications as Practice A to the existing practice asthma review template on their Vision electronic health database. Only 1 GP and 2 practice nurses were exposed to the changes and the nurses were not informed that the changes to the template had been made.

A GP at Practice C with lead responsibility at the practice for chronic respiratory diseases management and commissioning local respiratory services modified the existing asthma review template on the SystmOne electronic health record at Practice C. Read-coded buttons for occupational enquiry and 2 questions regarding work effect were created in a similar manner to Practice A and B (Figure 6.2). 19 doctors and 9 nurses undertaking asthma reviews were exposed to the template modifications though were not informed of the changes made.

A GP at Practice D with responsibility within the practice for chronic respiratory diseases management, and with experience working as a clinical assistant in a secondary care asthma clinic modified an existing asthma review template on the EMIS Web electronic health record at the practice to include (i) a tick box for occupational history enquiry and (ii) closed (yes/no) responses for questions about work relatedness of symptoms, all with opportunities for free text entry (Figure 6.3). Auditable Read codes were assigned to each closed response, and free text comments also appeared in an individual patient's electronic health record (Figure 6.4). Only the aforementioned GP and one practice nurse were exposed to the screening tool.

<b>Patient</b> Name: _____ NHS Number: _____	
<b>Asthma Management</b> <b>Asthma Management &amp; Plan :</b> <div style="display: flex; justify-content: space-between;"> <div style="width: 40%;"> <p>Asthma clinical management plan <span style="border: 1px solid black; display: inline-block; width: 150px; height: 20px; vertical-align: middle;"></span></p> <p>Management Plan <small>Tick one</small></p> <p>Asthma Control Steps <small>Tick one</small></p> <p>Treatment Compliance <small>Tick one</small></p> <p>Asthma medication review <span style="float: right;"><input type="checkbox"/></span></p> </div> <div style="width: 55%;"> <div style="border: 1px solid black; padding: 2px; margin-bottom: 2px;"><input type="checkbox"/> Asthma management plan given</div> <div style="border: 1px solid black; padding: 2px; margin-bottom: 2px;"><input type="checkbox"/> Change in asthma management plan</div> <div style="border: 1px solid black; padding: 2px; margin-bottom: 2px;"><input type="checkbox"/> Health education - asthma</div> <div style="border: 1px solid black; padding: 2px; margin-bottom: 2px;"><input type="checkbox"/> Step down change in asthma management plan</div> <div style="border: 1px solid black; padding: 2px; margin-bottom: 2px;"><input type="checkbox"/> Step up change in asthma management plan</div> <div style="border: 1px solid black; padding: 2px; margin-bottom: 2px;"><input type="checkbox"/> Asthma control step 1</div> <div style="border: 1px solid black; padding: 2px; margin-bottom: 2px;"><input type="checkbox"/> Asthma control step 2</div> <div style="border: 1px solid black; padding: 2px; margin-bottom: 2px;"><input type="checkbox"/> Asthma control step 3</div> <div style="border: 1px solid black; padding: 2px; margin-bottom: 2px;"><input type="checkbox"/> Asthma control step 4</div> <div style="border: 1px solid black; padding: 2px; margin-bottom: 2px;"><input type="checkbox"/> Asthma control step 5</div> <div style="border: 1px solid black; padding: 2px; margin-bottom: 2px;"><input type="checkbox"/> Asthma treatment compliance satisfactory</div> <div style="border: 1px solid black; padding: 2px; margin-bottom: 2px;"><input type="checkbox"/> Asthma treatment compliance unsatisfactory</div> </div> </div>	
<b>Exacerbation :</b> <div style="display: flex; justify-content: space-between;"> <div style="width: 40%;"> <p>Number of asthma exacerbations in past year <span style="border: 1px solid black; display: inline-block; width: 50px; height: 20px; vertical-align: middle;"></span></p> <p>Emergency admission, asthma <span style="float: right;"><input type="checkbox"/></span></p> <p>Recurrent chest infection <span style="float: right;"><input type="checkbox"/></span></p> <p>Productive cough <span style="float: right;"><input type="checkbox"/></span></p> <p>Sputum Type <small>Tick one</small></p> </div> <div style="width: 55%;"> <div style="border: 1px solid black; padding: 2px; margin-bottom: 2px;"><input type="checkbox"/> Productive cough -clear sputum</div> <div style="border: 1px solid black; padding: 2px; margin-bottom: 2px;"><input type="checkbox"/> Productive cough-yellow sputum</div> <div style="border: 1px solid black; padding: 2px; margin-bottom: 2px;"><input type="checkbox"/> Productive cough -green sputum</div> </div> </div>	
<b>Occupational Asthma :</b> <p>Some patients where Asthma is made worse by their occupation. Ask the patient about their occupation and specifically about the effect of work on their symptoms.</p> <p><b>Ask the following Questions:</b></p> <ol style="list-style-type: none"> <li>1. Are your Asthma symptoms better away from work, on days off.</li> <li>2. Are your Asthma symptoms better away from work on holiday.</li> </ol> <p><b>A YES answer to these questions make work related Asthma more likely.</b></p> <p>Occupational asthma <span style="float: right;"><input type="checkbox"/></span></p> <p>Patient Occupation <span style="border: 1px solid black; display: inline-block; width: 100px; height: 20px; vertical-align: middle;"></span></p>	

**Figure 6.2. Printout of occupational asthma tool screenshot on SystmOne electronic record at Practice C as seen by healthcare professionals during an asthma review. The documentation of the presence of work-related symptoms in the text boxes is Read-coded and auditable, and the data appear in an individual's health record.**

Summary Consultations Medication Problems Investigations Care History Diary Documents Referrals Gareth Walters Gareth Walters New Consultation

Save Template Cancel Template Spell check Search

Template Known

Report Management - 1 SCR - 7 GDSGP - 1 (1) Registration - 322 (4) Lab Reports - 3 (3) Tasks - 359 (2) Mail Inbox - 22 (22)

Inactive TRANSMISSION, Test (Mr) Born: 01-Jan-1960 (54y) Gender: Male NHS No.: 999 000 5249 Usual GP: (Dr)

**Asthma**

Pages

- Asthma screening
- GP Contract - Asthma**
- Associated asthma details
- Smoking status

View -> My Record (No shared data.)

**Examinations/Investigations**

Height  cm 06-Dec-2013 165 cm

Weight  kg 06-Dec-2013 80 kg

Body mass index  Calculate 06-Dec-2013 29.4 kg/m2

Rhinitis  No previous entry

☐ No nasal symptoms  No previous entry

Asthma control test  /25 01-Jul-2013

☒ Occupation history  No previous entry

Is your asthma better away from work, on days off  No previous entry

Are your asthma symptoms better away from work, on holiday  No previous entry

**Other details**

Predicted peak expiratory flow rate  Calculate 05-Aug-2011 627 L/min

Peak expiratory flow rate measured using EN 13826 device  L/min 05-Aug-2011 160 L/min

personal best  L/min No previous entry

Asthma step  28-Sep-2006 Asthma cont...

TRANSMISSION, Test (Mr)

**Figure 6.3.** Screenshot of the occupational asthma screening tool incorporated into existing asthma review template on EMIS Web database at Practice D, as seen by practice healthcare professionals. Ticking the “occupational history” box or responding (yes/no) to either or both of the 2 questions about work relatedness of asthma symptoms automatically creates auditable Read codes. Free text entry boxes are also seen.

Date	Consultation Text
	<p>still sob/despise finishing os</p> <p>rpt abx give doxycycline/sputum not back/ may need cxr/bloods etc if no better</p> <p>sputum results just in</p> <p>moraxella C sensitive to tetracycline</p> <p>patient advice the same</p>
09-Apr-2014	
History	<p>Diagnosis by specialist nurse, with COPD last year. Would like information on diagnosis.</p> <p>Would like information about best way to manage cough</p> <p>Chest infection, treated successfully.</p> <p>Now, only has coughing fits, set of by triggers, dust, smoke, exercise, hot, with green and white sputum. No chest tightness, no respiratory distress, no wheeze.</p> <p>Managed on 2 puffs symbicort daily and montelukast at night.</p> <p>Has reduced hours at work, to four days a week, feeling very tired.</p>
Examination	<p>Examn. of respiratory system • O/E - chest expansion normal • O/E - breath sounds normal • Blood oxygen saturation 99 % • Peak exp. flow rate: PEFR/PFR 280 L/min • Peak expiratory flow rate measured using EN 13826 device 280 L/min • Oral steroids last used</p>
Social	<p>Works as community occupational therapist, coughing fits induced by allergens in work environment, eg dust, smoke.</p> <p>Feels embarrassed at work now, when has coughing fits, as colleagues comment on it, and always suggest seeing her GP.</p> <p>No pets.</p> <p>Non-smoker</p> <p>Non drinker alcohol (social only).</p>
Assessment	<p>No nasal symptoms • Occupation history • Work aggravated asthma • Condition made worse by work • Asthma causes daytime symptoms most days • Asthma limits activities most days • Asthma disturbs sleep frequently • Increases inhaled steroids appropriately</p>
Problem	Acute exacerbation of asthma (First)
Comment	<p>send sputum away for C&amp;S</p> <p>OS start treatment</p>

**Figure 6.4. Screenshot of an electronic health record for an individual adult patient with asthma at Practice D. An opportunistic asthma review has been undertaken on the 9<sup>th</sup> April 2014 using the modified asthma template after the patient has attended Practice D with a cough. Occupational history and presence of a work effect on symptoms have been coded and free text entered regarding occupation (community occupational therapist) entered.**

### 6.3.3 Exposure to screening tool

Practices ran the feasibility study for 3-4 months and there was a variation in the number of asthma reviews undertaken during the study periods (71-425). Practices varied in the number of healthcare professionals engaged in undertaking asthma reviews (2-28 professionals per practice), and therefore in the number exposed to the screening tool (shown in Table 6.2).

	<b>Practice A</b>	<b>Practice B</b>	<b>Practice C</b>	<b>Practice D</b>
Location	Small Heath	Small Heath	Harborne	Moseley
Database used	Vision	Vision	SystmOne	EMIS Web
Number of staff exposed to template				
Total	19	3	28	2
Nurses	7	2	9	1
Doctors	12	1	19	1
Number returning study questionnaires (%)				
Total	16 (84)	1 (33)	6 (21)	1 (50)
Nurses	7	0	2	0
Doctors	9	1	4	1
Length of exposure to template (months)	3	3	4	3
Number of asthma reviews undertaken during study period	71	271	425	114
Number (%) of times occupation/work effect questions used	60 (85)	0 (0)	4 (1)	64 (56)

**Table 6.2. Number of asthma reviews undertaken and use of the screening tool at each recruited practice during the feasibility study period.**

#### 6.3.4 Questionnaire responses

Questionnaires were returned by 24 healthcare professionals exposed to the screening tool (15 GPs, 9 practice nurses) and response rate was 46% (21-84% across all 4 practices; see Table 6.2). One practice nurse registered a response but failed to answer any questions. Of the remaining 23 professionals 13 did not use the screening tool (10 GPs, 3 practice nurses) and reasons given for non-use are shown in Table 6.3.

Reasons given for not using occupational asthma screening tool
<ul style="list-style-type: none"><li>• “Not involved in asthma management”</li><li>• “Didn't notice it/ was not informed about it”</li><li>• “Not seen relevant patients and did not know it was there”</li><li>• “New to practice [and not aware]”</li><li>• “Great idea - probably not use because of the skew distribution of patients I see, lack of awareness and the pressure of QOF [Quality Outcomes Framework] at the end of March 2014”</li><li>• “Not required”</li><li>• “Don't see pt [patients] with regard to asthma, book into respiratory clinic”</li><li>• “Seen only young children recently!!”</li><li>• “The few I did ask were not work related and mostly used alternative asthma template so questions missed”</li><li>• “Wasn't aware of it!”</li><li>• “Unaware”</li><li>• “Not seen it yet - think not had many asthma reviews lately”</li><li>• “I did not see it in the template- although have now had a look and it is there”</li></ul>

**Table 6.3. Responders' reasons for not using occupational asthma screening tool, taken from questionnaire open comments.**

Nine professionals who did use the screening tool (n=10 screening tool “users”, but 1 nurse did not enter any further responses) responded to 10 Likert statements about the screening tool (5 GPs, 4 nurses). These responses are summarized in Table 6.4. There were no statistically significant differences between responses when grouped by job role (GP, practice nurse). Responders did not experience technical or procedural difficulties with the screening tool and its use did not add significantly to their workload during an asthma review (Figure 6.5). Responders were less confident (44% agreed or strongly agreed) about how to act when patients had work-related asthma symptoms (Figure 6.6) and 78% agreed/strongly agreed that further training in managing health aspects of suspected occupational asthma would improve the screening tool. All responders agreed that if the screening tool was rolled-out they would continue to use it (Figure 6.7) with 78% identifying no further potential problems with its use. 67% of responders felt that the screening tool had added something beneficial to the asthma review though no further open comments were offered.

		Practice nurses; n=4	General practitioners; n=5	TOTAL; n=9	Analyses of variance
Q1. I experienced difficulties asking patients questions about their occupation and whether their symptoms were better away from work	SA A N D SD	0 (0) 0 (0) 0 (0) 3 (75) 1 (25)	0 (0) 0 (0) 0 (0) 3 (60) 2 (40)	0 (0) 0 (0) 0 (0) 6 (67) 3 (33)	P=0.66
Q2. The screening tool was user-friendly (clear, concise, logical)	SA A N D SD	1 (25) 3 (75) 0 (0) 0 (0) 0 (0)	1 (20) 4 (80) 0 (0) 0 (0) 0 (0)	2 (22) 7 (78) 0 (0) 0 (0) 0 (0)	P=0.87
Q3. I experienced (technical/IT) difficulties using the screening tool in the asthma review template	SA A N D SD	0 (0) 0 (0) 0 (0) 3 (75) 1 (25)	0 (0) 0 (0) 0 (0) 3 (60) 2 (40)	0 (0) 0 (0) 0 (0) 6 (67) 3 (33)	P=0.66
Q4. Using the template added significantly to my workload during the consultation	SA A N D SD	0 (0) 0 (0) 0 (0) 3 (75) 1 (25)	0 (0) 0 (0) 2 (40) 1 (20) 2 (40)	0 (0) 0 (0) 2 (22) 4 (44) 3 (33)	P=0.69
Q5. I am confident in how to act on the results of these screening questions	SA A N D SD	0 (0) 2 (50) 1 (25) 1 (25) 0 (0)	1 (20) 1 (20) 3 (60) 0 (0) 0 (0)	1 (11) 3 (33) 4 (44) 1 (11) 0 (0)	p=0.70

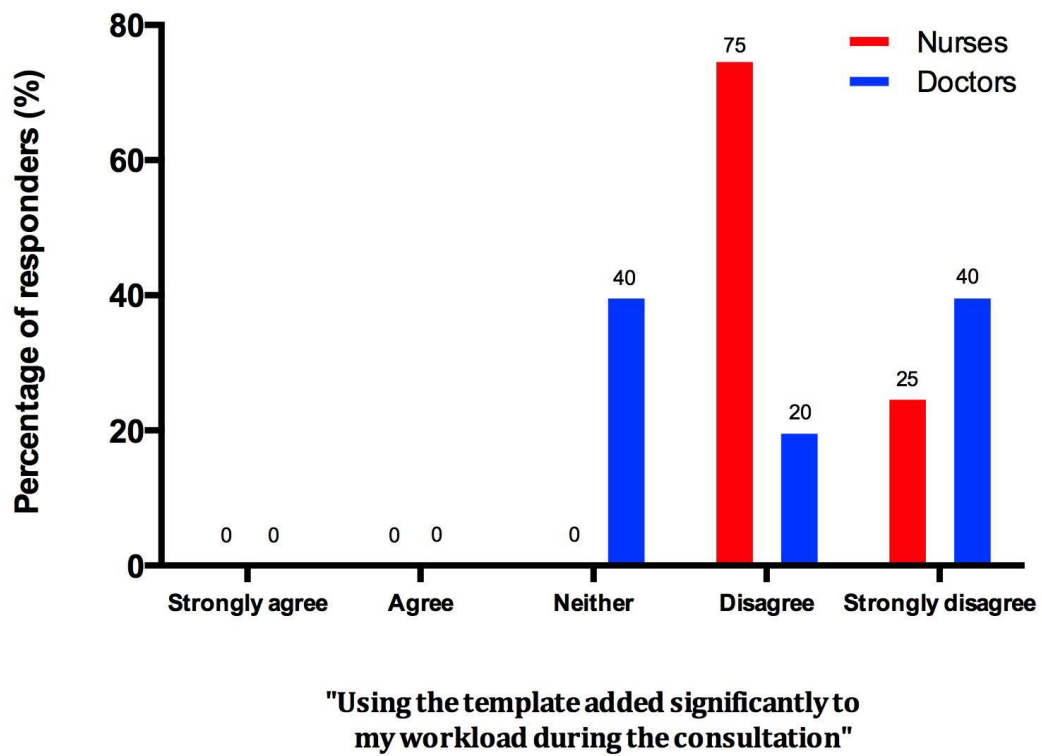
**Table 6.4. Healthcare professionals' responses to questions regarding the use of an occupational asthma screening tool categorized by job role (GP, nurse).**

SA=strongly agree; A=agree; N=neither agree nor disagree; D=disagree; SD=strongly disagree; IT=information technology. Analyses of variance were measured using non-parametric Kruskal-Wallis tests, significance measured at the 95% confidence level.

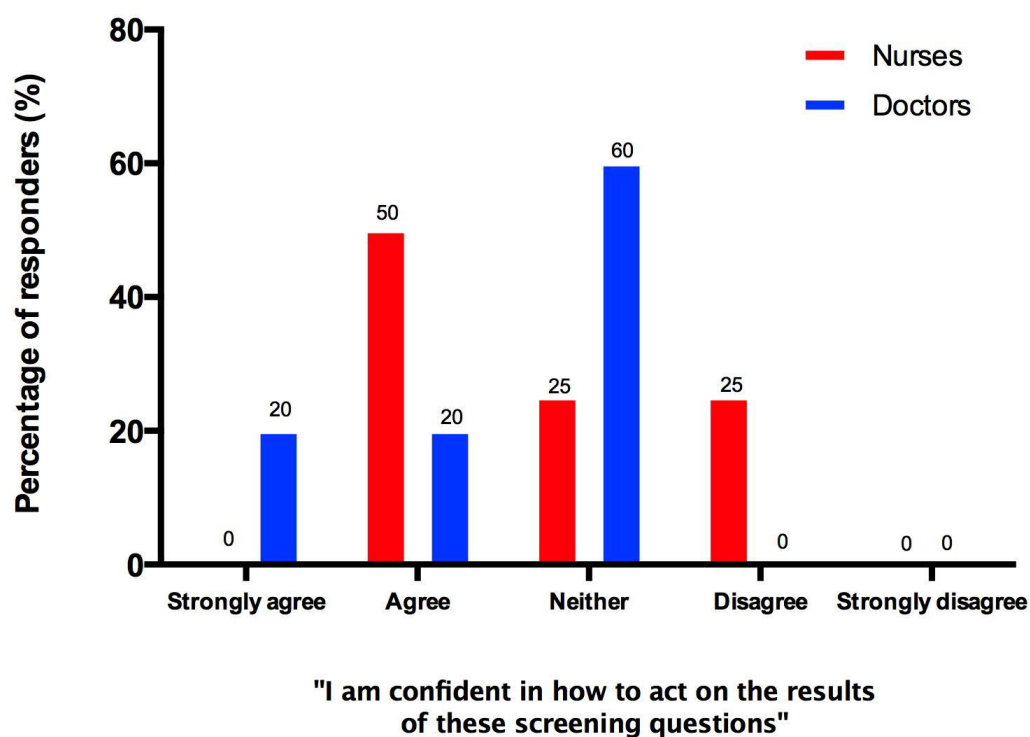


		Practice nurses; n=4	General practitioners; n=5	TOTAL; n=9	<i>Analyses of variance</i>
Q6. Would training for you in how to manage the health aspects of cases of suspected occupational asthma improve the use of the screening tool?	SA A N D SD	1 (25) 3 (75) 0 (0) 0 (0) 0 (0)	1 (20) 2 (40) 0 (0) 2 (40) 0 (0)	2 (22) 5 (56) 0 (0) 2 (22) 0 (0)	P=0.34
Q7. Would further training for you in how to advise the employer / manage the work situation in cases of suspected occupational asthma improve the use of the screening tool?	SA A N D SD	1 (25) 0 (0) 1 (25) 2 (50) 0 (0)	1 (20) 1 (20) 1 (20) 2 (40) 0 (0)	2 (22) 1 (11) 2 (22) 4 (44) 0 (0)	P=0.80
Q8. If this tool was kept in your asthma template would you keep using it?	SA A N D SD	1 (25) 3 (75) 0 (0) 0 (0) 0 (0)	1 (20) 4 (80) 0 (0) 0 (0) 0 (0)	2 (22) 7 (78) 0 (0) 0 (0) 0 (0)	P=0.87
Q9. If this tool was kept do you envisage any on-going or emergent problems with its use?	SA A N D SD	0 (0) 0 (0) 1 (25) 3 (75) 0 (0)	0 (0) 0 (0) 1 (20) 3 (60) 1 (20)	0 (0) 0 (0) 2 (22) 6 (67) 1 (11)	P=0.56
Q10. Do you think this tool has added anything beneficial to your asthma reviews?	SA A N D SD	0 (0) 3 (75) 1 (25) 0 (0) 0 (0)	1 (20) 2 (40) 2 (40) 0 (0) 0 (0)	1 (11) 5 (56) 3 (33) 0 (0) 0 (0)	p=1.00

Table 6.4 continued..



**Figure 6.5. Responses to Q4 regarding the burden of additional questions regarding occupation and work relatedness of asthma symptoms by job role.**



**Figure 6.6. Responses to Q5 regarding healthcare professionals' confidence in acting on patients' replies to occupational asthma screening questions, categorized by job role.**

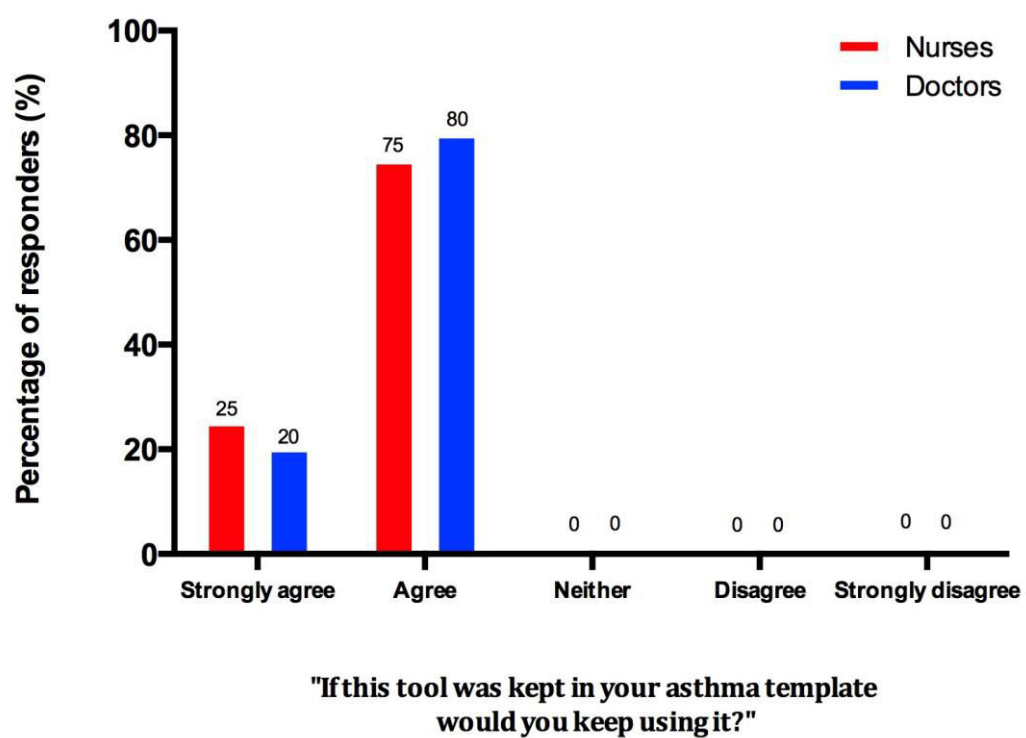


Figure 6.7. Responses to Q8 regarding healthcare professionals' opinions on ongoing use of a screening tool, categorized by job role.

## 6.4 Discussion

### 6.4.1 Summary

A screening tool for occupational asthma was successfully incorporated into existing electronic templates for asthma disease monitoring using 3 different patient management software systems (Vision, SystmOne, EMIS Web). The template modification at each practice was made rapidly (less than 30 minutes) and in each case by a GP with no new resources for the task or incentive to do so. When healthcare professionals were given information about the addition of a screening tool for a 3-4 month feasibility study, its uptake during asthma reviews was high (85% of reviews), but where no further information was offered uptake was lower (0-56% of reviews). Questionnaire responses suggested that the principal barrier to use of the tool was lack of awareness of its existence. Healthcare professionals who did use the screening tool found it to be user-friendly (clear, concise, logical) with no perceived procedural or IT difficulties. However they did identify a need for concurrent training in managing the health aspects of occupational asthma.

### 6.4.2 Limitations

It is necessary to consider the limitations of this feasibility study, particularly when considering the design of a future cluster trial of the clinical effectiveness of a screening tool. The study was undertaken in 4 practices in Birmingham, UK (2 city centre, 2 suburban) and therefore is not representative of the whole of the West Midlands or indeed the UK. The GPs contacted from 2 of the recruited practices had worked as clinical assistants in respiratory medicine in secondary care and 1 contact GP was the nominal lead clinician for respiratory diseases in his practice, so were therefore more experienced with asthma than many GPs. The practices were recruited directly by the primary author and contact GPs gave their time and resources through goodwill alone, which may have introduced an “enthusiasm bias” where any difficulties encountered recruiting healthcare professionals and practices less eager to help, are not appreciated. Questionnaire response rates were variable across the 4 practices (although similar for nurses and GPs) – low at practice C (21%) where no information about

the screening tool was given at the outset, and high at Practice A (84%) where all professionals were informed about the template change at the outset by an enthusiastic GP. This may have produced a non-response bias, and given that half of those who did complete the questionnaire did not use the tool at all, most lack of response is likely to be prompted by unawareness of its existence or being dismissed as unimportant.

#### 6.4.3 Interpretation

Baseline prevalences of diagnosed occupational asthma at recruited practices (0-0.7%) were low and consistent with prevalence estimates in Birmingham, UK from earlier work (Walters et al., 2012), suggesting that none of the recruited practices in this study were outliers (ie. high performers in identifying occupational asthma cases). List sizes, the number of registered asthmatics (6-8% of practice patient populations) and proportion of asthmatics of working age (56-65%) at recruited practices were consistent with previous estimates (Walters et al., 2012; National Health Service Information Centre, 2011; Lung and Asthma Information Agency, 2011; Asthma UK, 2004).

Modifying the asthma review template with additional questions using 3 software programmes in common usage in NHS primary care proved to be technically straightforward and was undertaken by GPs with little difficulty. Therefore it is reasonable to predict that this could be achieved in most NHS primary care practices in the UK by clinical or non-clinical staff with IT administration rights. The application of Read codes to data entered into the individual patient record when using the screening tool would make searching for, and gathering trial data, simple and rapid, as well as providing a straightforward tool for clinical audits.

The main barrier to the use of an occupational asthma screening tool was a lack of awareness of it as a feature in the asthma template, and so changing the format of a template, where visible statements on the screen merely act as prompts for the professional, and

documentation is not a mandatory requirement, does not predict its use. The utility of the screening tool increased when healthcare professionals were given brief instruction on its use (at Practice A). However no occupational asthma disease-specific education was given to any staff member and screening tool use was high despite this. The questionnaire responses did suggest that further education on managing the health aspects of occupational asthma would be welcomed at all practices and improve the usefulness of the tool, and this finding is consistent with the educational barriers identified in the previous studies in chapters 4 and 5.

Two other reasons for non-use of the tool were identified: (i) some healthcare professionals were not involved in adult asthma management and had not undertaken asthma reviews (this would not preclude GPs from seeing asthmatic patients with urgent problems and completing an annual review opportunistically however), and (ii) one professional had by chance not seen an asthmatic patient of late and therefore not been exposed to the tool. Two reasons for the observed low use of the screening tool in Practices B and C (identified by the contact GPs in both practices) were (i) including annual asthma medication reviews in the coding of asthma consultations, and (ii) the prioritization of documenting key disease indicators in patient records (eg. peak flow measurements, asthma control questions) at the expense of questions about work and occupation, as a requirement for the Quality Outcomes Framework (QOF; National Health Service Information Centre, 2011) in the run up to the end of the 2014 tax year (the study ran between January and April).

## 6.5 Conclusion

In conclusion the electronic screening tool for occupational asthma can be easily and quickly incorporated into existing asthma disease management systems in a variety of software programmes. It can be used by the healthcare professional without technical difficulty and is acceptable without adding any great extra burden to the current asthma review. Its utility could be greatly improved by user instruction and training in further clinical management of the patient with work related asthma symptoms.

## CHAPTER 7: GENERAL DISCUSSION

### 7.1 Summary

The overall aim of this work was to identify the main barriers to the diagnosis of occupational asthma for affected workers and attending healthcare professionals. Many workers with occupational asthma symptoms exhibited health beliefs that predisposed to delayed disclosure of symptoms to health professionals and employers, such as poor insight into the onset of symptoms, misattribution of symptoms to other illnesses, fear of financial loss due to illness, or low confidence in their GP to make a diagnosis. Some workers were unable to negotiate inadequate healthcare consultations to get to a diagnosis or find a workplace solution.

Occupational asthma was not well recognized in primary care, despite the publication of national guidance to aid assessment of adults with new-onset or recurrent asthma in terms of the risk of respiratory sensitization attached to their work, and the effect of work on their asthma symptoms. The rate of documented occupational asthma in primary care was very low when compared to UK estimates for prevalence of the disease, and a retrospective analysis of electronic patient records revealed that enquiry about occupational risk and the effect of work on asthma symptoms by GPs and practice nurses was very poor. In a cross-sectional questionnaire study of GPs, respiratory nurses, and occupational, general and respiratory physicians, only general medicine physicians reported lower rates of enquiry into work and the effects of work on asthma than occupational lung disease experts. However a poor questionnaire response rate led to an over-representation of occupational asthma enthusiasts amongst the sample of physicians, severely limiting generalizability of the results. Despite this, general medicine physicians and respiratory nurses were deficient in subject knowledge, awareness of guidelines and experience in managing cases of suspected occupational asthma, when compared with experts.



An electronic tool for GPs and practice nurses to screen working-age asthmatics for occupational asthma was developed, and incorporated into a variety of software patient management systems in common usage, for use at asthma review consultations. The uptake of the tool by GPs and nurses was higher when instruction for its use was given by the lead GP, and those that did use it found it to be user-friendly with no perceived procedural or technical difficulties. A need for further training in managing the health aspects of occupational asthma was also identified in those using the screening tool.

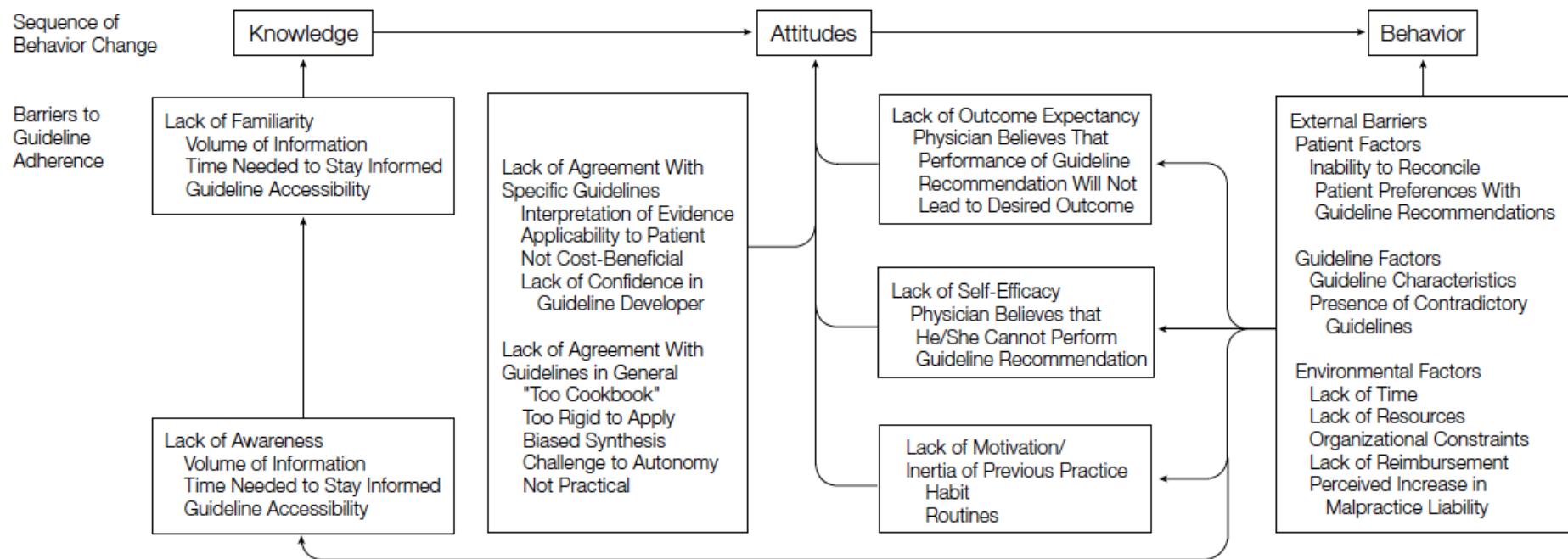
*7.2 Hypothesis 1: That poor recognition of occupational asthma in primary care is because primary healthcare professionals do not follow UK guidance for occupational asthma by asking working-age asthmatics about their work or the effect of work on their symptoms.*

The first point of contact with a healthcare professional for the majority of workers with work-related asthma symptoms is the GP, and 50% of patients referred for suspected occupational asthma have seen their GP >5 times before referral occurs (Fishwick et al., 2007). Therefore the burden of identifying potential cases of occupational asthma, in individuals with new adult-onset asthma symptoms, falls heavily on primary care professionals.

The data from primary care in this thesis, which showed a 5-fold underestimation of the prevalence of occupational asthma, and a clear lack of enquiry about occupational risk and work-relatedness of symptoms, shows that there has been no improvement in case identification 15 years after a study which reported under-recognition of occupational asthma in primary care to be approximately 50% (de Bono and Hudsmith, 1999). The implication is that the publication of concise guidance for healthcare professionals in assessing working-age asthmatics and screening for occupational asthma, either as part of general asthma guidelines (BTS/SIGN, 2012), or specific occupational asthma guidelines for GPs and practice nurses (BOHRF, 2010a) and any physician (Fishwick et al., 2012), have had no impact whatsoever

on improving case recognition in primary care. Indeed from the questionnaire study in this thesis, self-reported BOHRF guideline use amongst general and respiratory physicians and GPs was very low (4-24%), and use of the section on occupational asthma in the general BTS/SIGN asthma guideline was less than expected for all specialty groups studied (16-57%). This supports questionnaire data from the study by Barber et al. (2010), which demonstrated very limited awareness of the BOHRF guidelines, with 72% of respondents stating either that they had never heard of them, or had heard of them but had never seen them.

There is a perception that clinical guidelines are not popular with GPs and physicians, and although some authors have described them as anti-intellectual, as averaging practice, discouraging person-centred medicine, cost-saving, and encouraging litigation (Deutsch et al., 1998; Delmothe, 1998), surveys of healthcare professionals including GPs have consistently reported high levels of satisfaction and few barriers to their use (Farquhar et al., 2002; Lugtenberg et al., 2011). GPs in the UK have found clinical guidelines to be useful means of accessing subject-specific information, where key factors in their uptake are brevity, perceived authority and a resonance with daily practice (Watkins et al., 1999). Cabana et al. (1999) systematically reviewed published studies and texts of barriers to physician adherence to clinical guidelines, and after concluding that these were setting-specific and not generalizable, produced a framework of guideline-related knowledge, attitudes and behaviour (Figure 7.1) that could be used to identify barriers and improve guideline adherence.



**Figure 7.1. Barriers to physician adherence to practice guidelines in relation to behaviour change (taken from Cabana et al., 1999).**

7.3 Hypothesis 2: *That delay in diagnosis of occupational asthma is partly due to multiple societal factors affecting whether an individual worker will disclose his or her occupational asthma symptoms to a healthcare professional or employer.*

Workers with occupational asthma symptoms revealed a broad range of important beliefs relating to how they perceived their occupational asthma symptoms, how they sought medical help, and how they handled employment issues such as sickness absence or presenteeism.

Varied and poor understanding of the nature of symptoms, fear of job loss on disclosing symptoms, lack of confidence in a solution, as well as external motivating factors such as lack of supportive family, colleagues or employer, and poor occupational health provision may all contribute towards delayed diagnosis. These findings are not unexpected, indeed previous studies have cited a variety of barriers to engagement with healthcare services and employers, such as fear of financial loss through unemployment or absenteeism (Bradshaw et al. 2007), loss of status and working relationships (Gordon et al., 1997) and a lack of awareness of workplace hazards (Poonai et al., 2005; Santos et al., 2007). These are basic insights and fundamental beliefs for many workers that should be accounted for by all clinicians engaged with workers with occupational asthma symptoms, and by specialists and policy makers planning educational interventions in the workplace to increase identification of occupational asthma.

HSE-initiated workplace interventions have been undertaken in the motor-vehicle repair industry (HSE, 2007a), baking and food processing industry (HSE, 2008) and manufacturing industry (HSE, 2007b), and there is evidence that these have been successful in reducing the incidence of occupational asthma due to certain agents (for example, glutaraldehyde and latex in the NHS; Stocks et al., 2013b; Walters et al. 2013). However these interventions have been multi-modal in nature, for example those aimed at flour and baking enzymes in craft

bakeries (employer training, inspection, introduction of workplace exposure limits for flour and baking enzymes), isocyanates in spray painting at motor vehicle repairers (awareness days, inspection and enforcement of process improvements) and rosin-based solder flux fume (colophony) in manufacturing (inspection, guidance pack for employers, introduction of work exposure limits for colophony), with little emphasis on worker education, whose impact has not been studied in any detail.

7.4 Hypothesis 3: *That healthcare professionals lack knowledge and experience in managing cases of occupational asthma, which prevents them from screening and diagnosing occupational asthma in working-age adults.*

There is evidence from this work that healthcare professionals lack knowledge and experience in dealing with work-aspects of asthma. Educational deficits were observed in the questionnaire study where respiratory nurses (in primary and secondary care) were less knowledgeable and confident than doctors at screening and managing occupational asthma; general hospital physicians (mainly trainees) reported a lack of subject knowledge and little clinical experience in screening and managing occupational asthma. One might hypothesize that a lack of background knowledge and awareness, due to a deficit in formal undergraduate and/or postgraduate occupational lung disease education, alongside poor clinical involvement with workers with occupational asthma during postgraduate training are major barriers to diagnosis in subsequent clinical practice. Indeed in the feasibility study the majority of healthcare professionals using the screening tool identified a need for further education in managing health aspects of occupational asthma.

Barber et al. (2010) evaluated a new e-learning tool based on key statements from BOHRF (2010a) and BTS guidelines (Fishwick et al., 2012) for occupational asthma by administering a post-module questionnaire, which was returned by 32% of 1041 total users. This is a low response rate, but typical for evaluations for e-learning modules in the opinion of the authors

(Barber et al., 2010). The majority of responders stated that the module had significantly improved their subject knowledge, and although pre-module awareness and use was low, they reported that they were likely to use the evidenced-based guidelines in the future. The authors cited a paucity of undergraduate and postgraduate occupational lung disease training for current GPs as potential reasons for underlying knowledge deficiencies. Similar experiences have been reported in the USA (Glazer et al., 2008) and Canada (Tarlo et al., 2009) where case-based learning has been used to improve evidence-based practice.

*7.5 Hypothesis 4: That there are a number of motivating factors (for example: perceived importance, resource constraints) which predispose to lack of consideration of the diagnosis of occupational asthma in adults.*

There was no evidence from the questionnaire study that healthcare professionals' motivations (internal motivations: perceived benefit to patient, practice or society of making a diagnosis of occupational asthma, current routine, perceived self-motivation; external motivations: perceived background prevalence of the disease, time pressure, access to specialist services, reimbursement) impacted on work-related enquiry and guideline use. It is worth restating that the study was biased towards occupational asthma enthusiasts, and in addition closed questions predispose to certain cognitive biases that may have led to underestimation of these factors as barriers in screening and diagnosis. However, the feasibility study showed that screening guidance, which was reproduced from UK national guidelines (Fishwick et al., 2012), was clear and concise, with both nurses and GPs using it with little observed difficulty.

It appears that GPs do not act on patients' concerns about work-related symptomatology or high-risk work processes when information is offered, either by further enquiry or referring for specialist advice. Interviews with patients with occupational asthma symptoms revealed

inadequacies in primary care consultations, where in addition to complete lack of enquiry, GPs were perceived as dismissive of crucial work-related observations about symptoms, with a tendency to oversimplify or trivialize symptoms, or mis-diagnose patients particularly with repeated benign infections. Interviewed workers often thought that this was due to the GP being unfamiliar with work processes, but occasionally they believed they were being seen as malingerers, out for financial gain, and therefore the GP was reluctant to investigate further. It should be stated that this was not always the case, and a few workers felt that their GP took a systematic approach and carefully explored a number of diagnoses leading to delayed diagnosis. However, there is evidence elsewhere that crucial information is missed or ignored in primary care. In the primary care audit, where patients with new adult-onset asthma had an occupation documented, a large minority of those (32%) was employed in a high-risk occupation with no demonstration of any further enquiry or action taken. Indeed in the study by Hoyle et al. (2010) only 26% of patients with respiratory illnesses and exposed to asthmagens were referred for specialist opinion. Given that healthcare professionals in primary care do not appear to lack motivations to identify occupational asthma, this suggests a deficit in occupational asthma subject knowledge, ignorance of occupational risk, and a lack of experience in assessing and managing patients with work-related asthma symptoms.

## 7.6 Future direction

Two simple questions (“Are your symptoms better on days away from work?” and “Are your symptoms better on holiday?”) have been validated for identifying cases of occupational asthma (Malo et al., 1991; Vandenplas et al., 2005), and they are recommended for use as screening questions for general physicians and in primary care (Fishwick et al., 2012; BOHRF, 2010a). It is clear from this work that although they are not currently well used in primary care, these simple questions are easy to use and incorporate into existing patient management systems. It is not yet known whether such a screening tool is clinically effective in primary care populations in terms of increasing case identification, reducing diagnostic

latency and improving health outcomes such as serial FEV<sub>1</sub> decline. Therefore in order to test such hypotheses, a randomized controlled clinical effectiveness trial in a primary care population is warranted.

The poor response rate to the Internet based questionnaire limited the conclusions that could be drawn about healthcare workers' barriers to diagnosing occupational asthma, as described above. A number of strategies were employed to maximize response rate, and apart from occupational lung disease specialists who had a high rate of response (89%), this resulted in a disappointing response from other specialty groups of interest (3-16%). Therefore rather than repeating the same experiment and expecting a better response rate, a different study design would be recommended to test the hypothesis that educational deficits amongst healthcare professionals are their most significant barriers to diagnosis. Despite the purported evidence that both subject knowledge and clinical experience are low amongst generalist physicians, particularly GPs, it is not known whether the use of educational programmes to remedy deficiencies can lead to better guideline adherence in practice, increased case recognition, reduced latency to diagnosis and better health-related outcomes for workers.

Workers' beliefs about occupational asthma vary substantially: specifically how their symptoms are perceived, how they react to their presence, and subsequently how they behave and negotiate with healthcare professionals and employers. It has been seen that lack of accurate understanding about asthma symptoms, lack of confidence or knowledge that symptom disclosure will effect a solution, and fear of financial loss from disclosure are all major barriers to diagnosis, and are likely to contribute to delayed diagnosis. It is not known whether workplace interventions that focus on addressing workers' variation in understanding of symptomatology, and their barriers to symptoms disclosure, can equip them with the knowledge and skills to reach a diagnosis with good health and employment outcomes. This specifically needs urgent investigation.



### 7.7 Conclusion

The burden from occupational asthma is a suitable and urgent target for intervention. There are clearly significant financial and health costs for workers, and also great costs to the rest of society in the UK. There is also a clear case for improving diagnosis, as there are significant delays that have been shown to lead to poor health and financial outcomes for the worker. Despite the availability of guidelines for healthcare professionals, valid tools for screening, and clear pathways to diagnosis, little progress has been made with respect to case recognition in working age populations, and a better understanding of the barriers to diagnosis will now enable design of targeted interventions to increase diagnosis. Future interventions for healthcare professionals should focus on subject knowledge and management of the affected worker with occupational asthma symptoms, and this should start at undergraduate level for healthcare professionals with a clear theme through ongoing professional development. For workers, any workplace intervention strategy should take into account the variation in their basic insights about asthma and asthma symptoms.

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## ABSTRACTS, PRESENTATIONS AND PAPERS ARISING FROM THIS THESIS

### Chapter 3: Assessment for occupational asthma in primary care

1. Presented as a poster at the Midland Thoracic Society Spring Meeting, April 2012 in Solihull, West Midlands, UK;
2. Published as a paper in the journal *Occupational Medicine* in July 2012:  
Walters G. I., McGrath E. E., Ayres J. G. (2012). Audit of the recording of occupational asthma in primary care. *Occupational Medicine* **62**:570-3.

### Chapter 4: Understanding health beliefs and behaviour in workers with occupational asthma

1. Presented as a poster at the Midland Thoracic Society Spring Meeting, April 2014 in Solihull, West Midlands, UK and winner of the Harold Thomas Travelling Fellowship prize.
2. Abstract accepted by the European Respiratory Society for presentation at the Annual Congress, Munich September 2014.

### Chapter 5: Barriers to identifying occupational asthma for healthcare professionals

1. Awarded the Sandy Elder Prize from the Scottish group of the Society of Occupational Medicine, UK, 2013.

### Chapter 6: Feasibility study of a primary care screening tool for occupational asthma

1. Abstract accepted by the British Thoracic Society for presentation at the Winter Meeting, London, December 2014.

## APPENDIX 1

### PATIENT INFORMATION SHEET VERSION 1.0; 16<sup>TH</sup> DECEMBER 2012

**Study Title:** Health-seeking behaviour of workers with occupational asthma symptoms

**Principal Investigator:** Dr. Gareth Walters, Research Associate and Specialist Registrar, Occupational Lung Disease Unit, Heart of England NHS Foundation Trust

**Patient Information Sheet:** Version 1.0. Date: 16<sup>th</sup> December 2012

We would like to invite you to take part in our research study. Before you decide we would like you to understand why the research is being done and what it might involve for you. This information sheet explains the purpose and the conduct of this study and what will happen to you if you take part. One of our research team will go through the information sheet with you and answer any questions you might have. They can also provide you with any additional information you might require. This should take about 15 minutes in total.

#### **Why are we doing the study?**

When asthma is caused by work we call that occupational asthma (OA). There are many materials in the workplace that can cause asthma, and work is a common cause of new asthma symptoms (breathlessness, wheeze, cough and chest tightness) in adults in the UK. However, there is often a long delay in the diagnosis of OA after symptoms start, and up to half of cases go undiagnosed. This is partly because the sufferer is reluctant to seek help from a healthcare professional. It is not known why workers with asthma symptoms delay seeing a doctor or nurse, and so the aim of this study is to explore the reasons why that might be and find out which factors are more important.

#### **What sort of study are we going to undertake?**

We are planning to use a 'qualitative method' for the study. This means that we will ask you questions about your asthma symptoms, your workplace and your GP and you can give any answer you wish. When we look at the answers you give we will compare your answers with those of other participants and look for patterns (themes). We hope that when we put these together, they will tell us how workers think and behave when they develop asthma symptoms at work.

#### **What will happen to me if I take part?**

You would be invited to a 10- to 30-minute interview at the Occupational Lung Disease Unit, Heart of England NHS Trust with Dr. Gareth Walters (principal researcher). You will be asked about your asthma symptoms, the effect on your work and the care you have received from your employer and from your GP. There are no correct or incorrect answers and so we would value any responses that you give to questions. You do not have to answer every question if you don't wish to. We will

record the interview with a Dictaphone (handheld digital audio recording device). You will then be free to leave and we will analyze a transcript of the answers that you give, in order to look for themes.

### **Why have I been invited?**

We have invited you because you are a worker with asthma symptoms and there is a possibility you might have OA. Therefore we are interested in how you came to seek help for your condition, and the relationship you had with your employer and your doctor when the symptoms started.

### **Do I have to take part?**

No, you can decide not to take part if you don't want to. This won't affect your routine care in any way.

### **What are the possible disadvantages and risks of taking part?**

Some patients feel anxious when talking about their own health problems or how their asthma symptoms affect their work. Some patients feel uncomfortable when talking about relationships with their employer or GP, for fear of their opinions being reported back to either party. However we would not tell your GP or employer about your participation in the research. There are no monetary payments for you for taking part in the study.

### **What are the possible benefits of taking part?**

This study is looking at the process of diagnosis and so there is no intended benefit to your health from taking part. Taking part in this research would not change your routine NHS care in any way at all. Some participants may feel they have a better understanding of OA, merely by taking part in the research and thinking about their own health.

### **Will my taking part in the study be kept confidential?**

Yes, Dr. Gareth Walters will code your data so that your personal details are removed and you can't be identified. If you make reference to a particular workplace or GP practice in your responses to questions, these will also be removed when the recording is transcribed. The data will be stored securely at the Heart of England NHS Trust, and then archived following Trust guidelines once the study is completed. The only people who will have access to your data are members of the research team.

### **Will you tell my GP about the answers I gave to study questions?**

No, we would not tell your GP either that you are participating in the study, or about the responses you give to questions. If you particularly wanted us to inform your GP about your participation then we would do so in writing. The specialist doctor responsible for your care at the Occupational Lung Disease Unit would know about your participation in the study, but only the principal researcher (Dr. Gareth Walters) would be able to identify you, as your details are anonymized.

**Will you tell my employer about the study?**

No, we would not communicate with your employer, either about your participation, or about the responses you give to questions

**What will happen to the results of the study?**

We will send you a summary of the results if you wish. This will be a detailed account of the anonymized responses from all the participants. We also intend to publish the results of the research in a peer-reviewed medical journal. Your personal details would not be identified in any publication or communication.

**Who is organizing the research?**

The research is being organized through the Heart of England NHS Foundation Trust, of which the Birmingham Chest Clinic and Occupational Lung Disease clinic is a part.

**What will happen if I wish to take part?**

If you agree to take part, Dr. Gareth Walters will check your understanding and ask you to sign a consent form. You will also be given a copy of this to keep for your own records.

**What will happen if I don't want to carry on with the study?**

You may decide you do not wish to participate in the study at all. Also, if you do decide to take part, you are then free to change your mind at any point during the study and withdraw your consent. No further information for the study would be sought after this point. Be assured that this will not affect your routine NHS care in any way.

**Further information and contact details**

Further information about the study can be found by contacting the principal investigator:

**Principal Investigator:** Dr. Gareth Walters MB ChB MRCP (UK), Specialist Registrar in Respiratory Medicine, at the Occupational Lung Disease clinic, Birmingham Chest Clinic, 151 Great Charles Street, Birmingham, B3 3HX

More information for patients about clinical research can be found on the National Institute for Health Research website at <http://www.crncc.nihr.ac.uk/>.

APPENDIX 2

CONSENT FORM VERSION 1.0; 16<sup>TH</sup> DECEMBER 2012

**Study Title:** Health-seeking behaviour of workers with occupational asthma symptoms

**Principal Investigator:** Dr. Gareth Walters MRCP BSc (Hons.)  
Specialist Registrar in Respiratory Medicine  
Heart of England NHS Foundation Trust

**Participant Identification Number for this study:**

Please initial  
box

I have had time to read the Patient Information Sheet and I understand it.

The study has been fully explained to me and my questions about it have been answered.

I understand what the study involves and what I am expected to do.

I am willing to allow access to my medical records for the purposes of the study. However, I understand that personal details, such as my name or address will not be published.

I am free to stop being in the study at any time. If I wish to stop, this will not affect the quality of care that I receive in the future.

I understand that the Heart of England NHS Trust will keep my data in accordance with principles of confidentiality and ethical research set out for the NHS.

I agree to take part in the above named study.

\_\_\_\_\_  
Name of Patient

\_\_\_\_\_  
Signature

\_\_\_\_\_  
Date

\_\_\_\_\_  
Researcher

\_\_\_\_\_  
Signature

\_\_\_\_\_  
Date

1 copy for medical notes; 1 copy for site file; 1 copy for patient.



### APPENDIX 3

#### SEMI-STRUCTURED INTERVIEW SCHEDULE VERSION 2.0; 18/07/2013

##### Demographics

How old are you please?

How long have you been working for your current employer?

When were you diagnosed with occupational asthma?

##### Patient's understanding of their symptoms

When did you first realize you had problems with your breathing?

What symptoms or problems did you notice that caused you concern?

Did you understand that the symptoms might be asthma?

*(If not) Prompt: What did you think the symptoms might represent? Did you think that the symptoms might represent a disease?*

Did you see a pattern between the symptoms and your work?

Did you think that work might be causing your symptoms?

##### Patient's initial response to symptoms

What course of action did you take when you first noticed your symptoms?

Did you take time off work or did you continue at work whilst having symptoms?

*Prompt: Could you justify why you decided this? Why did you continue at work?*

Did you seek any help either at work, from health and safety or from your General Practitioner or other health care professional with your symptoms initially?

When did you first seek medical assistance?

Was there a distinct event (*illness event*) that caused you to seek help?

##### The General Practitioner and occupational asthma

How long have you known your General Practitioner / Practice Nurse?

Can you describe your relationship with them?

*Prompt: Do you trust them?*

Prompt: *Do you feel able to share information with them?*

Did they ask you about your job and the effect of work on your symptoms?

Did they make the link between work and your symptoms?

Did they make a diagnosis from your symptoms?

What actions resulted from consulting your General Practitioner?

Prompt: *Did your General Practitioner communicate with the specialist?*

### Social structure and occupational asthma

Were there people or factors that helped you seek a diagnosis?

Prompt: *Do you have family, relatives or close friend that you spoke with about your symptoms?*

Is there any one in particular that you have shared your diagnosis or problems with? If so, why?

Are there any colleagues at work who you could share it with and why?

Are there any colleagues at work you wouldn't share it with and why?

### The employer and occupational asthma

Do your employers know about your occupational asthma?

Did they have any input into diagnosing your symptoms?

Did you communicate with them about the symptoms before your diagnosis of occupational asthma?

(If not) Prompt: *If not, why not?*

(If so) Prompt: *If so, how did you find their reaction, and how did that make you feel about your symptoms?*

Prompt: *Did you feel that your doctor (Occupational Health Professional, General Practitioner, medical specialists) communicated well with you?*

Do you feel that there was good communication between all parties involved in your care?

Did you think that there was delay in the process of diagnosis?

Prompt: *If so, where do you think that was?*

## APPENDIX 4

### QUESTIONNAIRE DOMAINS: BELIEFS AND ATTITUDES HYPOTHESIZED TO BE BARRIERS TO IDENTIFYING OCCUPATIONAL ASTHMA ON THE PART OF THE HEALTHCARE WORKER

#### Knowledge and experience

- Awareness of occupational asthma
- Disease-specific declarative knowledge for occupational asthma
- Knowledge of work processes and occupations
- Procedural knowledge on screening and diagnosing occupational asthma
- Knowledge and experience in managing cases of occupational asthma
- Awareness of guidelines for occupational asthma

#### Attitudes

- Perceived confidence in clinical diagnosis of occupational asthma
- Perceived confidence in managing cases of occupational asthma
- Perceived importance of a diagnosis of occupational asthma
- Perceived prevalence of occupational asthma
- Outcome expectancy (benefit to patients)
- Inertia (internal motivation, changing habits)

#### External barriers

- Guideline-related (clarity, complexity, access)
- Time pressure with existing routines (complexity, occupational history)
- Access to specialist services
- Reimbursement

## APPENDIX 5

FINAL QUESTIONNAIRE VERSION 1.0; 5<sup>TH</sup> FEBRUARY 2013

### **Questionnaire title: Wheezy while you work**

“This is a short anonymous questionnaire for healthcare professionals who attend to adult asthmatics of working-age in their clinical practice. It will take you 3- 5 minutes to complete. All questions are multiple choice in nature, and there is an opportunity for open comment at the end. You may also choose to go on and complete an optional case study of a worker with asthma after the questionnaire”

**I see asthmatic patients of working age (16-64 years old) in my clinical practice?**

Yes / No

*Participants who answer ‘No’ do not need to continue with the questionnaire, and are forwarded to a page that thanks them for their participation, and says ‘As you do not see working-age asthmatic patients in your clinical practice, you do not need to continue with the questionnaire’. Those who answer yes continue to the rest of the questionnaire below:*

*Please respond to the following statements, by circling the response that best reflects how you feel:*

**I ask them about the nature of their work? [Likert response]**

Strongly Agree      Agree      Neither agree nor disagree      Disagree      Strongly disagree

**I ask them about the effects of work on their asthma symptoms? [Likert]**

**I think it is important to recognize when work is the cause of asthma symptoms [Likert]**

**I feel confident to screen patients for occupational asthma based on their clinical history [Likert]**

**I have sufficient knowledge of patients’ work practices to screen for occupational asthma [Likert]**

**I feel confident to make a diagnosis of occupational asthma [Likert]**

**I have access to specialist input for occupational asthma when I feel I need it [Likert]**

**I am aware of clinical guidelines for assessing patients with suspected occupational asthma [Likert]**

**I follow occupational asthma guidelines when assessing a working-age asthmatic**  
*[Yes/No]*

*[If answer Yes only: Which guidelines do you use? SIGN-BTS; BOHRF-BTS; ERS]*

**I lack the time to screen asthmatic patients for occupational asthma** *[Likert]*

**I have no incentive to screen asthmatic patients for occupational asthma** *[Likert]*

**I am not motivated to screen patients for occupational asthma** *[Likert]*

**I have experience of managing cases of suspected occupational asthma in my clinical practice** *[Likert]*

**A diagnosis of occupational asthma can improve patient outcomes** *[Likert]*

**Do you wish to comment on anything here? (Leave blank if not)** *[Blank box for comments]*

***A little bit about you, please respond to the following statements***

**You are a...**      Nurse   /   Doctor

*[...If answer 'doctor' What grade of doctor are you? (pick the answer that best fits)*

Foundation / SHO level;      Registrar / ST3+ / Staff grade;      GP / Consultant / Associate specialist]

**You are...**      Male   /   Female

**How many years clinical experience do you have?** *[open question]*

**Which area do you work in mainly? (Pick the answer that best fits)**

General Practice;    General Practice + Occupational Medicine sessions;    Occupational Medicine

General Hospital Medicine;    Respiratory Medicine (ie. Chest physician or Respiratory Specialist Nurse)

“Thank you for your responses, we are very grateful for your participation. You can finish here, or you can go on and complete an optional case study about a 42-year old bakery worker with asthma symptoms. Obviously we would like you to complete the case study if you have the time. Once you have read the case, there are some multiple choice questions to answer. It will take another 3-5 minutes of your time”

**I wish to complete the case study**      Yes   /   No

*Those answering 'no' finish the questionnaire at this point.*

*Those answering 'yes' are taken to the case study and greeted with the phrase: 'Please read the following short case study and answer the questions'.*

*The participant is randomised to one of the four case studies, but all participants receive the same multiple-choice questions that follow.*

CASE STUDY: SCENARIO 1 = SW; strong work, weak non-work (definite occupational asthma)

A 42-year old man comes to see you in your clinic complaining of intermittent wheeze and shortness of breath. This has come on gradually over the last 12 weeks. He has noticed that on some nights he will wake up and feels short of breath, and he has a discomfort in his chest when he plays football on a cold morning. He has noticed that his symptoms tend to improve at weekends. He has also noticed that he tends to get a runny nose while he is at work. At first he thought this was due to a cold but he has not really had any other symptoms to suggest this. Examination, including chest examination, is essentially normal.

You send him for pulmonary function testing which shows an FEV<sub>1</sub> 3.14L (75% predicted) and FVC 5.09L (98% predicted), FEV<sub>1</sub>/FVC ratio=62%. Post-bronchodilator his FEV<sub>1</sub> is 4.11L (an increase of 31%). His Peak expiratory flow pre-bronchodilator is 8.34L/sec (90% predicted) and post-bronchodilator is 10.33L/sec (an increase of 24%). He also has a positive methacholine test (non-specific bronchial reactivity) with a PC<sub>20</sub> of 0.88 mg/ml.

He currently works as a baker in the bakery department of a local superstore. This involves him in mixing and preparing raw ingredients for the products to be baked, then putting the mixed ingredients into baking tin or on a baking tray. The job is quite dusty at times. Another person will then usually do the baking of the products and take them out to be put on display. He also helps with cleaning. He has done this job for about 6 months. Prior to this he has had a number of other jobs in the catering and food preparation industry but this is his first time working as a baker.

He has no previous history of chest problems or other illnesses in the past. Nobody in the family has any problems with previous chest problems or allergies that he is aware of. He has never smoked regularly. When talking with the other people at work he has been told that the person who previously did his job had to leave because of similar problems. The patient feels his current problems are caused by work and wants your advice.

CASE STUDY: SCENARIO 2 = SS; strong work, strong non-work (intermediate probability occupational asthma)

A 42-year old man comes to see you in your clinic complaining of intermittent wheeze and shortness of breath. This has come on gradually over the last 12 weeks. He has noticed that on some nights he will wake up and feels short of breath, and he has a discomfort in his chest when he plays football on a cold morning. He has noticed that his symptoms tend to improve at weekends. He has also noticed that he tends to get a runny nose while he is at work. At first he thought this was due to a cold but he has not really had any other symptoms to suggest this. Examination, including chest examination, is essentially normal.

You send him for pulmonary function testing which shows an FEV<sub>1</sub> 3.14L (75% predicted) and FVC 5.09L (98% predicted), FEV<sub>1</sub>/FVC ratio=62%. Post-bronchodilator his FEV<sub>1</sub> is 4.11L (an increase of 31%). His Peak expiratory flow pre-bronchodilator is 8.34L/sec (90% predicted) and post-bronchodilator is 10.33L/sec (an increase of 24%). He also has a positive methacholine test (non-specific bronchial reactivity) with a PC<sub>20</sub> of 0.88 mg/ml.

He currently works as a baker in the bakery department of a local superstore. This involves him in mixing and preparing raw ingredients for the products to be baked, then putting the mixed ingredients into a baking tin or on a baking tray. The job is quite dusty at times. Another person will then usually do the baking of the products and take them out to be put on display. He also helps with cleaning. He has done this job for about 6 months. Prior to this he has had a number of other jobs in the catering and food preparation industry but this is his first time working as a baker.

He did have asthma as a child and he thinks his current symptoms feel similar to that, although he has not been troubled with chest symptoms since he was 10 or 11. He has also had hay fever intermittently since he was a child and this has been a little more noticeable in the last 3 or 4 years. He has two brothers and two sisters, and one of his brothers has asthma, one sister has hay fever. He has two children, one of whom has asthma. He smoked between the ages of 18 and 35 approximately one pack of cigarettes a day. When talking with the other people at work he has been told that the person who previously did his job had to leave because of similar problems. The patient feels his current problems are caused by work and wants your advice.



CASE STUDY: SCENARIO 3 = WW; weak work, weak non-work (intermediate probability occupational asthma)

A 42-year old man comes to see you in your clinic complaining of intermittent wheeze and shortness of breath. This has been worse over the last 12 weeks, but has probably been present for about 18 months. He has noticed that on some nights he will wake up and feels short of breath, and he has a discomfort in his chest when he plays football on a cold morning. He has not noticed any real improvement at weekends or while on holiday. Examination, including chest examination, is essentially normal.

You send him for pulmonary function testing which shows an FEV<sub>1</sub> 3.14L (75% predicted) and FVC 5.09L (98% predicted), FEV<sub>1</sub>/FVC ratio=62%. Post-bronchodilator his FEV<sub>1</sub> is 4.11L (an increase of 31%). His Peak expiratory flow pre-bronchodilator is 8.34L/sec (90% predicted) and post-bronchodilator is 10.33L/sec (an increase of 24%). He also has a positive methacholine test (non-specific bronchial reactivity) with a PC<sub>20</sub> of 0.88 mg/ml.

He currently works behind the bakery counter of a local superstore dealing with customers. He is not involved in baking bread or other bakery products as these arrive at the store pre-prepared and cooked. He also helps with cleaning. He has done this job for about 6 months. Prior to this he has had a number of other jobs in the catering and food preparation industry but this is his first time working in a bakery.

He has no previous history of chest problems or other illnesses in the past. Nobody in the family has any problems with previous chest problems or allergies that he is aware of. He has never smoked regularly. None of his colleagues at work have noticed any problems with dust or had similar symptoms. The patient feels his current problems are caused by work and wants your advice.

CASE STUDY: SCENARIO 4 = WS; weak work, strong non-work (definitely not occupational asthma)

A 42-year old man comes to see you in your clinic complaining of intermittent wheeze and shortness of breath. This has been worse over the last 12 weeks, but has probably been present for about 18 months. He has noticed that on some nights he will wake up and feels short of breath, and he has a discomfort in his chest when he plays football on a cold morning. He has not noticed any real improvement at weekends or while on holiday. Examination, including chest examination, is essentially normal.

You send him for pulmonary function testing which shows an FEV<sub>1</sub> 3.14L (75% predicted) and FVC 5.09L (98% predicted), FEV<sub>1</sub>/FVC ratio=62%. Post-bronchodilator his FEV<sub>1</sub> is 4.11L (an increase of 31%). His Peak expiratory flow pre-bronchodilator is 8.34L/sec (90% predicted) and post-bronchodilator is 10.33L/sec (an increase of 24%). He also has a positive methacholine test (non-specific bronchial reactivity) with a PC<sub>20</sub> of 0.88 mg/ml.

He currently works behind the bakery counter of a local superstore dealing with customers. He is not involved in baking bread or other bakery products as these arrive at the store pre-prepared and cooked. He also helps with cleaning. He has done this job for about 6 months. Prior to this he has had a number of other jobs in the catering and food preparation industry but this is his first time working in a bakery.

He did have asthma as a child and he thinks his current symptoms feel similar to that, although he has not been troubled with chest symptoms since he was 10 or 11. He has also had hay fever intermittently since he was a child and this has been a little more noticeable in the last 3 or 4 years. He has two brothers and two sisters, and one of his brothers has asthma, one sister has hay fever. He has two children, one of whom has asthma. He smoked between the ages of 18 and 35 approximately one pack of cigarettes a day. None of his colleagues at work have noticed any problems with dust or had similar symptoms. The patient feels his current problems are caused by work and wants your advice.

Questions for all case studies

**Do you think that the diagnosis is occupational asthma?** *[Likert]*

Strongly Agree      Agree      Neither agree nor disagree      Disagree      Strongly disagree

**How confident are you?** *[Likert]*

Very confident      Confident      Somewhat confident      Unsure      Very unsure

**Would you refer this case to a specialist in occupational lung diseases?**

Yes    /    No    /    I am a specialist in OLD

Which of the following factors was important in contributing to your decision that this was or was not a work-related illness?

**Your understanding of the nature of his work?** *[Likert]*

Not at all important      Low importance      Neutral      Somewhat important      Extremely important

**The timing of onset of symptoms in relation to work** *[Likert]*

**His report that other workers were affected or not** *[Likert]*

**His belief about whether the illness was work-related or not** *[Likert]*

**The presence or absence of other potential causes and risk factors outside work** *[Likert]*

**Do you wish to comment on anything here? (Leave blank if not)** *[open question]*

## APPENDIX 6

INVITATION LETTER VERSION 2.0; 13<sup>TH</sup> MAY 2013



### **Invitation letter to participants for the study:**

#### **“What are the barriers to the identification of occupational asthma by the healthcare professional?”**

Dear Colleague,

I am a Respiratory Medicine Specialist Registrar in the West Midlands and a Research Associate at the Institute of Occupational and Environmental Medicine, University of Birmingham. I am undertaking this study as part of my doctoral research at the Institute, and would be very grateful if you could take 5 minutes to complete an online questionnaire concerning your clinical practice with **working-age asthmatic patients**. Your responses do not require you to enter personal details, beyond your age and current professional role, and results will be linked anonymously and automatically to a database for analysis. In addition to appearing in my thesis, results will be disseminated by publication in a peer-reviewed journal.

The study can be accessed online via the following link:

<https://www.surveymonkey.com/s/Wheezy>

The questionnaire contains a number of statements, each of which is followed by a list of potential responses that reflect how you might feel about the statement. You will generally be required to pick one from the list. There are opportunities for more open-ended answers as well. There is no obligation for you to complete all of the questions, give open-ended answers, or indeed undertake the questionnaire at all if you do not wish to. There is also an optional case scenario with associated questions that you will be invited to complete at the end of the study.

Local R&D approval for the study has been granted by Heart of England NHS Foundation Trust Research and Development Directorate (**R&D Code: 2013013RM**). If you have any questions I can be contacted by email at [gareth.walters@heartofengland.nhs.uk](mailto:gareth.walters@heartofengland.nhs.uk). The British Occupational Health Research Foundation produces guidance for health professionals on work-related asthma, and this guidance can be found online at: <http://www.bohrf.org.uk/projects/asthma.html>. More general information about the effect of work on asthma symptoms can be found at [www.occupationalasthma.com](http://www.occupationalasthma.com).

Thank you in anticipation,

Dr. Gareth Walters MRCP(UK); Specialist Registrar and Research Associate, University of Birmingham

## APPENDIX 7

TEXT INSERT VERSION 1.0; 16<sup>TH</sup> MAY 2013

**Calling all West Midlands physicians who see working-age patients with asthma in their clinical practice!**

I would like you to take 3-5 minutes to complete an anonymous online survey about your practice with **working-age asthmatic** patients please. There is also an optional case study after the survey. It is part of my research into occupational asthma at the Institute of Occupational and Environmental Medicine, **University of Birmingham**, and the survey will also be going to other physicians, nurses and GPs as well as their trainees elsewhere in the UK. The study is sponsored by the Heart of England NHS Foundation Trust, and the study can be accessed online via the following link:

<https://www.surveymonkey.com/s/Wheezy>

Thank you in great anticipation

Gareth Walters

SpR and Research Associate Respiratory Medicine,

Heart of England NHS Foundation Trust

# APPENDIX 8: Q1-Q14 RESPONSES BY SPECIALTY

		Doctors							Nurses			
		General Practice; <i>n</i> =62	General Practice undertaking OH sessions; <i>n</i> =14	Occupational Medicine; <i>n</i> =123	General Hospital Medicine; <i>n</i> =96	Respiratory Medicine; <i>n</i> =82	Occupational Lung Disease specialists; <i>n</i> =17	<i>Analysis of variance</i>	Practice nurses; <i>n</i> =127	Occupational health nurses; <i>n</i> =11	Hospital respiratory nurse specialists; <i>n</i> =68	<i>Analysis of variance</i>
Q1. Asks about the nature of a patient's work	<i>SA</i>	21 (34)	8 (57)	109 (89)	24 (25)	50 (61)	17 (100)	p<0.001	81 (64)	9 (82)	55 (81)	p=0.03
	<i>A</i>	35 (57)	6 (43)	14 (11)	55 (57)	30 (37)	0 (0)		41 (32)	2 (18)	13 (19)	
	<i>N</i>	2 (3)	0 (0)	0 (0)	9 (9)	1 (1)	0 (0)		4 (3)	0 (0)	0 (0)	
	<i>D</i>	4 (6)	0 (0)	0 (0)	5 (5)	1 (1)	0 (0)		1 (1)	0 (0)	0 (0)	
	<i>SD</i>	0 (0)	0 (0)	0 (0)	3 (3)	0 (0)	0 (0)		0 (0)	0 (0)	0 (0)	
Q2. Asks about the effects of work a patient's asthma symptoms	<i>SA</i>	13 (21)	5 (36)	103 (84)	9 (9)	37 (45)	17 (100)	p<0.001	68 (54)	9 (82)	46 (68)	p=0.02
	<i>A</i>	35 (56)	8 (57)	19 (15)	44 (46)	40 (49)	0 (0)		50 (39)	2 (18)	22 (32)	
	<i>N</i>	8 (13)	1 (7)	1 (1)	24 (25)	4 (5)	0 (0)		8 (6)	0 (0)	0 (0)	
	<i>D</i>	6 (10)	0 (0)	0 (0)	17 (18)	1 (1)	0 (0)		1 (1)	0 (0)	0 (0)	
	<i>SD</i>	0 (0)	0 (0)	0 (0)	2 (2)	0 (0)	0 (0)		0 (0)	0 (0)	0 (0)	
Q3. Thinks it is important to recognize when work is the cause of asthma	<i>SA</i>	38 (61)	10 (71)	115 (94)	48 (50)	65 (79)	17 (100)	p<0.001	95 (75)	10 (91)	58 (85)	p=0.006
	<i>A</i>	23 (37)	4 (29)	9 (6)	48 (50)	16 (20)	0 (0)		32 (25)	1 (9)	10 (15)	
	<i>N</i>	1 (2)	0 (0)	0 (0)	0 (0)	1 (1)	0 (0)		0 (0)	0 (0)	0 (0)	
	<i>D</i>	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)		0 (0)	0 (0)	0 (0)	
	<i>SD</i>	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)		0 (0)	0 (0)	0 (0)	
Q4. Feels confident to screen for occupational asthma based on history	<i>SA</i>	9 (15)	4 (29)	73 (59)	7 (7)	26 (32)	16 (94)	p<0.001	14 (11)	8 (73)	17 (25)	p=0.001
	<i>A</i>	30 (48)	7 (50)	40 (33)	47 (49)	39 (48)	1 (6)		59 (47)	2 (18)	32 (47)	
	<i>N</i>	13 (21)	1 (7)	8 (6)	24 (25)	11 (13)	0 (0)		31 (24)	1 (9)	11 (16)	
	<i>D</i>	9 (14)	2 (14)	2 (2)	17 (18)	6 (7)	0 (0)		21 (16)	0 (0)	7 (10)	
	<i>SD</i>	1 (2)	0 (0)	0 (0)	1 (1)	0 (0)	0 (0)		2 (2)	0 (0)	1 (2)	

**Table A1. Responses to questions regarding healthcare professionals' practice with working-age asthmatics, grouped by medical specialty. SA=strongly agree; A=agree; N=neither agree nor disagree; D=disagree; SD=strongly disagree. Analyses of variance measured using non-parametric Kruskal-Wallis tests, significance measured at the 95% confidence level.**

Q5. Has sufficient knowledge to screen for occupational asthma	SA A N D SD	4 (7) 15 (24) 25 (40) 15 (24) 3 (5)	4 (29) 6 (43) 3 (21) 0 (0) 1 (7)	79 (64) 33 (27) 10 (8) 1 (1) 0 (0)	5 (5) 27 (28) 30 (31) 33 (34) 1 (1)	13 (16) 32 (39) 27 (33) 10 (12) 0 (0)	13 (76) 3 (18) 1 (6) 0 (0) 0 (0)	p<0.001	5 (4) 53 (42) 31 (24) 33 (26) 5 (4)	8 (73) 3 (27) 0 (0) 0 (0) 0 (0)	11 (16) 30 (44) 16 (24) 10 (15) 1 (1)	p<0.001
Q6. Feels confident to make a diagnosis of occupational asthma	SA A N D SD	6 (10) 18 (29) 20 (32) 15 (24) 3 (5)	3 (21) 8 (57) 1 (7) 1 (7) 1 (7)	50 (41) 52 (42) 16 (13) 4 (3) 1 (1)	5 (5) 25 (26) 30 (31) 35 (37) 1 (1)	12 (15) 37 (45) 25 (30) 8 (10) 0 (0)	14 (82) 3 (18) 0 (0) 0 (0) 0 (0)	p<0.001	6 (5) 35 (27) 28 (22) 48 (38) 10 (8)	0 (0) 4 (36) 1 (9) 5 (46) 1 (9)	9 (13) 25 (37) 17 (25) 14 (21) 3 (4)	p=0.023
Q7. Has access to specialist input for occupational asthma	SA A N D SD	9 (15) 30 (48) 8 (13) 11 (18) 4 (6)	3 (21) 7 (50) 1 (7) 2 (14) 1 (7)	79 (64) 34 (28) 5 (4) 5 (4) 0 (0)	17 (18) 38 (40) 27 (28) 13 (13) 1 (1)	39 (48) 26 (32) 8 (10) 9 (11) 0 (0)	15 (88) 1 (6) 0 (0) 1 (6) 0 (0)	p<0.001	34 (27) 43 (34) 21 (16) 25 (20) 4 (3)	6 (55) 4 (36) 0 (0) 1 (9) 0 (0)	30 (44) 16 (24) 8 (12) 13 (19) 1 (1)	p=0.13
Q8. Is aware of clinical guidelines for occupational asthma	SA A N D SD	9 (14) 28 (45) 7 (11) 14 (23) 4 (7)	5 (36) 6 (43) 1 (7) 0 (0) 2 (14)	81 (66) 36 (29) 4 (3) 2 (2) 0 (0)	4 (4) 14 (15) 17 (18) 57 (59) 4 (4)	23 (28) 36 (44) 12 (15) 11 (13) 0 (0)	17 (100) 0 (0) 0 (0) 0 (0) 0 (0)	p<0.001	25 (20) 65 (51) 19 (15) 15 (12) 3 (2)	5 (45) 6 (55) 0 (0) 0 (0) 0 (0)	23 (34) 34 (50) 5 (7) 6 (9) 0 (0)	p=0.009
Q10. Lacks time to screen for occupational asthma	SA A N D SD	6 (10) 20 (32) 12 (19) 22 (36) 2 (3)	0 (0) 5 (36) 3 (21) 5 (36) 1 (7)	1 (1) 10 (8) 17 (14) 59 (48) 36 (29)	4 (4) 23 (24) 25 (26) 42 (44) 2 (2)	2 (2) 15 (18) 21 (26) 36 (44) 8 (10)	0 (0) 1 (6) 1 (6) 5 (29) 10 (59)	p<0.001	4 (3) 48 (38) 30 (24) 39 (31) 6 (5)	0 (0) 1 (9) 1 (9) 6 (55) 3 (27)	3 (4) 15 (22) 14 (21) 30 (44) 6 (9)	p=0.009

**Table A1 continued. Q5 to Q10.**

Q11. Has no incentive to screen for occupational asthma	SA A N D SD	2 (3) 11 (18) 12 (19) 27 (44) 10 (16)	0 (0) 5 (36) 2 (14) 4 (29) 3 (21)	1 (1) 0 (0) 7 (6) 37 (30) 77 (63)	2 (2) 15 (16) 23 (24) 51 (53) 5 (5)	1 (1) 3 (4) 12 (15) 42 (51) 24 (29)	1 (6) 0 (0) 0 (0) 3 (18) 13 (76)	p<0.001	2 (2) 10 (8) 23 (18) 58 (45) 34 (27)	0 (0) 0 (0) 2 (18) 4 (36) 5 (46)	2 (3) 2 (3) 11 (16) 29 (43) 24 (35)	p=0.26
Q12. Is not motivated to screen for occupational asthma	SA A N D SD	1 (2) 4 (6) 11 (18) 30 (48) 16 (26)	0 (0) 2 (14) 2 (14) 4 (29) 6 (43)	3 (2) 3 (2) 4 (3) 35 (29) 78 (63)	1 (1) 9 (9) 22 (23) 53 (55) 11 (12)	0 (0) 1 (1) 8 (10) 43 (52) 30 (37)	0 (0) 0 (0) 0 (0) 1 (6) 16 (94)	p<0.001	1 (1) 7 (5) 10 (8) 65 (51) 44 (35)	0 (0) 0 (0) 2 (18) 4 (36) 5 (46)	0 (0) 2 (3) 5 (7) 34 (50) 27 (40)	p=0.26
Q13. Has experience of managing cases of suspected occupational asthma	SA A N D SD	6 (10) 17 (27) 9 (14) 24 (39) 6 (10)	3 (23) 6 (46) 1 (8) 2 (15) 1 (8)	54 (44) 51 (41) 7 (6) 6 (5) 5 (4)	6 (6) 19 (20) 18 (19) 45 (47) 8 (8)	10 (12) 35 (43) 19 (23) 15 (18) 3 (4)	13 (76) 2 (12) 0 (0) 1 (6) 1 (6)	p<0.001	11 (9) 51 (40) 21 (17) 32 (25) 11 (9)	3 (27) 8 (73) 0 (0) 0 (0) 0 (0)	6 (9) 31 (46) 18 (26) 11 (16) 2 (3)	p=0.005
Q14. A diagnosis of occupational asthma can improve patient outcomes	SA A N D SD	31 (50) 25 (40) 5 (8) 0 (0) 1 (2)	9 (64) 5 (36) 0 (0) 0 (0) 0 (0)	72 (59) 35 (29) 6 (5) 1 (1) 7 (6)	34 (35) 58 (60) 4 (4) 0 (0) 0 (0)	45 (56) 31 (38) 3 (4) 1 (1) 1 (1)	15 (88) 1 (6) 1 (6) 0 (0) 0 (0)	p=0.004	55 (44) 55 (44) 12 (10) 0 (0) 2 (2)	3 (27) 7 (64) 0 (0) 1 (9) 0 (0)	34 (51) 27 (40) 3 (4) 0 (0) 3 (5)	p=0.54

**Table A1 continued. Q11 to Q14.**



## APPENDIX 9: FACTORS CONTRIBUTING TO ATTRIBUTION OF OCCUPATIONAL CAUSATION

		Overall; n=298	GPs	GP with sessions in OH	Occupational medicine	General hospital medicine	Respiratory medicine	OLD specialist	Analysis of variance
Your understanding of the nature of his work	NAAI	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	<b>p&lt;0.001</b>
	LI	1 (0)	1 (2)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	
	N	13 (4)	1 (2)	0 (0)	2 (2)	6 (9)	4 (7)	0 (0)	
	SI	76 (26)	11 (24)	2 (18)	12 (12)	30 (44)	18 (32)	3 (18)	
	EI	208 (70)	32 (71)	9 (82)	87 (86)	32 (47)	34 (61)	14 (82)	
	<sup>1</sup> Mean statistic (min=1; max=5; SD)	4.6 (0.58)	<b>4.6 (0.65)</b>	<b>4.8 (0.40)</b>	<b>4.8 (0.42)</b>	<b>4.4 (0.65)</b>	<b>4.5 (0.63)</b>	<b>4.8 (0.63)</b>	
The timing of onset of symptoms in relation to work	NAAI	1 (0)	0 (0)	0 (0)	1 (1)	0 (0)	0 (0)	0 (0)	p=0.613
	LI	1 (0)	0 (0)	0 (0)	1 (1)	0 (0)	0 (0)	0 (0)	
	N	8 (3)	2 (4)	1 (9)	1 (1)	2 (3)	1 (2)	1 (6)	
	SI	68 (23)	12 (27)	0 (0)	26 (26)	17 (25)	10 (18)	3 (18)	
	EI	220 (74)	31 (69)	10 (91)	72 (71)	49 (72)	45 (80)	13 (76)	
	Mean statistic (min=1; max=5; SD)	4.7 (0.57)	4.6 (0.57)	4.8 (0.6)	4.6 (0.65)	4.7 (0.53)	4.8 (0.46)	4.7 (0.59)	
His report that other workers were affected or not	NAAI	31 (10)	5 (11)	2 (18)	5 (5)	9 (13)	9 (16)	1 (6)	<b>p=0.03</b>
	LI	58 (20)	12 (27)	1 (9)	17 (17)	15 (22)	12 (21)	1 (6)	
	N	72 (24)	11 (24)	2 (18)	22 (22)	21 (31)	11 (20)	5 (29)	
	SI	103 (35)	13 (29)	4 (36)	42 (42)	18 (27)	18 (32)	8 (47)	
	EI	34 (11)	4 (9)	2 (18)	15 (15)	5 (7)	6 (11)	2 (12)	
	Mean statistic (min=1; max=5; SD)	3.2 (1.18)	<b>3.0 (1.2)</b>	<b>3.3 (1.4)</b>	<b>3.4 (1.1)</b>	<b>2.9 (1.2)</b>	<b>3.0 (1.3)</b>	<b>3.5 (1.0)</b>	
His belief about whether the illness was work-related or not	NAAI	11 (4)	1 (2)	2 (18)	5 (5)	1 (1)	2 (4)	0 (0)	p=0.28
	LI	42 (14)	4 (9)	1 (9)	17 (17)	12 (18)	6 (11)	2 (12)	
	N	111 (37)	14 (31)	4 (36)	43 (43)	23 (34)	21 (37)	6 (53)	
	SI	119 (40)	26 (58)	2 (18)	31 (31)	27 (40)	25 (45)	8 (47)	
	EI	15 (5)	0 (0)	2 (18)	5 (5)	5 (7)	2 (4)	1 (6)	
	Mean statistic (min=1; max=5; SD)	3.29 (0.90)	3.4 (0.76)	3.1 (1.4)	3.1 (0.9)	3.3 (0.9)	3.3 (0.9)	3.5 (0.9)	
The presence or absence of other potential causes and risk factors outside work	NAAI	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	p=0.18
	LI	12 (4)	2 (4)	0 (0)	2 (2)	2 (3)	3 (5)	3 (18)	
	N	26 (9)	4 (9)	1 (9)	9 (9)	3 (4)	6 (11)	3 (18)	
	SI	163 (55)	24 (53)	4 (36)	52 (51)	47 (69)	29 (52)	7 (41)	
	EI	97 (33)	15 (33)	6 (55)	38 (38)	16 (24)	18 (32)	4 (23)	
	Mean statistic (min=1; max=5; SD)	4.16 (0.74)	4.2 (0.8)	4.5 (0.7)	4.2 (0.7)	4.1 (0.6)	4.1 (0.8)	3.7 (1.0)	

**Table A2.** Responses to the question “Which of the following factors was important in contributing to your decision that this was or was not a work-related illness?” grouped by medical specialty. <sup>1</sup>Mean statistic based on 1=minimum (NAAI) and 5=maximum (EI). GP=general practitioner; OH=occupational health; OLD=occupational lung disease; NAAI=not at all important; LI=low importance; N=neutral; SI=somewhat important; EI=extremely important. Analyses of variance measured using non-parametric Kruskal-Wallis tests, significance measured at the 95% confidence level.

## APPENDIX 10. QUESTIONNAIRE FOR HEALTHCARE PROFESSIONALS

### **Occupational asthma pilot study**

You probably know this already, but your practice has been taking part in a feasibility (pilot) study of a **screening tool** for identifying occupational asthma in working-age asthmatics that was developed by me (Dr Gareth Walters, respiratory registrar) at the Institute of Occupational and Environmental Medicine, University of Birmingham and Heartlands Hospital and by Professor Jon Ayres. You may have used the occupational asthma screening-template when seeing a patient with asthma over the last few months. We now need to get a feel for how the tool has worked and so we would be grateful if you could answer a few questions about the pilot which will take you less than 3 minutes to complete. We would be very grateful for your feedback. Thank you, GW

<b>Practice</b>	<input type="text"/>		
<b>Role</b> (please tick)	Nurse <input type="checkbox"/>	GP <input type="checkbox"/>	Other <input type="text"/>

*Please answer the following question/statements by circling one of the responses:*

1. Did you use the occupational asthma template?

**Yes**

**No**

If not, why not?

2. When using the template, I experienced difficulties asking patients questions about their occupation and whether their symptoms were better away from work.

**Strongly agree**

**Agree**

**Neither Agree nor disagree**

**Disagree**

**Strongly disagree**

If you did experience difficulties, what were they?

3. The screening tool was user friendly (clear, concise, logical).

Strongly agree      Agree      Neither Agree nor disagree      Disagree      Strongly disagree

4. I experienced (technical/IT) difficulties using the screening tool in the asthma review template.

Strongly agree      Agree      Neither Agree nor disagree      Disagree      Strongly disagree

If so, what were they?

5. Using the template added significantly to my workload during the consultation.

Strongly agree      Agree      Neither Agree nor disagree      Disagree      Strongly disagree

If so, in what way?

6. I am confident in how to act on the results of these screening questions.

Strongly agree      Agree      Neither Agree nor disagree      Disagree      Strongly disagree

7. Would training for you in how to advise patients / manage the health aspects of cases of suspected occupational asthma improve the use of the screening tool?

Strongly agree      Agree      Neither Agree nor disagree      Disagree      Strongly disagree

8. Would further training for you in how to advise the employer / manage the work situation in cases of suspected occupational asthma improve the use of the screening tool?

Strongly agree      Agree      Neither Agree nor disagree      Disagree      Strongly disagree

9. If this tool was kept in your asthma template would you keep using it?

Strongly agree      Agree      Neither Agree nor disagree      Disagree      Strongly disagree

10. If this tool was kept do you envisage any ongoing or emergent problems with its use?

**Strongly agree      Agree      Neither Agree nor disagree      Disagree      Strongly disagree**

If so, what are they?

11. Do you think this tool has added anything beneficial to your asthma reviews?

**Strongly agree      Agree      Neither Agree nor disagree      Disagree      Strongly disagree**

Please tell me more:

Many Thanks

Gareth Walters, SpR and research associate