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Work aggravated asthma: A review of reviews

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Work aggravated asthma: A review of reviews

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Seven percent of the adult population have asthma, a condition commonly made worse by inhaling irritant exposures at work; termed work-aggravated asthma (WAA). A variety of factors that cause WAA have been identified by this review, including inhaled exposures, physical factors and behavioural issues.

WAA is common. A recent comprehensive review identified that more than 1 in 5 workers with asthma have this condition. There are, however, no GB based prevalence estimates.

WAA is an unpleasant condition. Workers complain of cough, wheeze, chest tightness and shortness of breath that is aggravated at work. These symptoms are likely to influence work absence, presenteeism and work efficiency. Their presence is also associated with significant adverse socio-economic impacts for workers and workplaces.

A variety of medical tests may be needed to help make a diagnosis of WAA, and distinguish this from occupational asthma. These tests are normally only available in specialised units with a particular interest in occupational asthma.

Very little GB data exists about interventions to reduce the associated burdens to the individual and the workplace. These include improving asthma treatments, worker education and training and assessing risks posed by various known hazards with particular relevance to asthma.

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KEY MESSAGES

Asthma is common in adults. Seven percent of the adult population have this condition, and most people with asthma have to take medication to control the disease. Asthma is commonly made worse by inhaled exposures at work, these exposures being mostly irritant in nature. This is termed work aggravated asthma (WAA). WAA is defined as pre-existing or concurrent asthma that is worsened by workplace conditions.

A variety of general and specific types of inhaled occupational exposures that cause WAA have been identified by this review. Physical factors such as changes in temperature, physical exercise requirements and behavioural issues, including stress, also contribute to WAA. Material Safety Data Sheets may give important information about irritant properties of inhaled agents.

Other risk factors for WAA are poorly understood, as previous studies have assessed widely varying populations in terms of geography, workplace sector and demography.

WAA is common. Prevalence estimates vary, but the most recent review specifically of WAA (Henneberger, 2011) identified that more than 1 in 5 workers (21.5%) with asthma have this condition. The upper limit of all estimates was 58%, seen in a population of workers with asthma followed up by their general practitioners. There were no GB based prevalence estimates identified.

WAA is an unpleasant condition. Workers complain of episodes of cough, wheeze, chest tightness and shortness of breath that can be aggravated at work in the short term, or consistently over a longer period of time. The mechanisms by which irritants and other exposures cause WAA are poorly understood. Asthma medication may need to be increased during work periods. Symptoms of WAA are likely to influence work absence, presenteeism and work efficiency. Health care use in this condition is similar to those with occupational asthma, and high in comparison to those without asthma.

It is important to consider other medical diagnoses, the most important of which is occupational asthma. The latter is where someone becomes sensitised to a substance in the workplace inducing asthma subsequently in that individual. This distinction is important, as actions required following a diagnosis may differ at the individual and workplace levels. A variety of medical tests may be needed to help make this distinction, and these are available only in GB secondary care centres with a specific interest in occupational asthma.

WAA has a significant associated socio-economic impact as judged by unemployment and ability to work. There are very little data relating to sickness absence and presenteeism in this group of workers. Consequently, there are potential significant gains if interventions are designed to reduce WAA.

Very little GB data exists about interventions to reduce the associated burdens to the individual and the workplace. Those currently described include improving asthma control with medication where needed, worker education and training, assessing risks posed by various known hazards with particular relevance to asthma, and intervening to reduce relevant exposures where necessary using standard hygiene based control approaches. Protective levels of exposure are not identified and will likely vary between individuals with asthma.

How generalisable are these messages? The reviews assessed covered a wide range of studies from differing geographies, worker populations, demographics and industrial sectors. Given this, there is no reason to suspect that these are not generalisable to GB working populations.

EXECUTIVE SUMMARY

Asthma is common in adults. 7% of the adult population have this condition. Most asthma requires medication to control the disease. Asthma is commonly made worse by inhaled exposures at work, and most of the agents responsible are irritant in nature. This is termed work aggravated asthma (WAA), defined as "pre-existing or concurrent asthma that is worsened by workplace conditions".

This review of reviews was undertaken to provide a narrative on the factors that can aggravate pre-existing asthma in the workplace. Whilst this process did not include a formal gap analysis, knowledge gaps are identified where appropriate. Eight *a priori* defined questions were used to focus extraction of information from relevant reviews. These were;

A. Is there a definition of WAA? If yes, what is it?
B. What is the prevalence of WAA?
C. Which causative agents are associated with the onset of WAA?
D. What are the risk factors associated with WAA?
E. What symptoms are associated with WAA?
F. How is a diagnosis of WAA made?
G. Are there any successful interventions for the prevention and treatment of WAA?
H. Is there a socio-economic burden associated with work-aggravated asthma?

Fifty-two abstracts were reviewed, from which 19 papers were obtained and assessed. Sixteen of these were included in the final evidence table (Table 2). Three of the sixteen articles were specifically related only to WAA, rather than more general reviews of work-related asthma.

Question A: is there a definition of WAA? If yes, what is it?
Various definitions were identified. The most recent and most comprehensive review (Henneberger, 2011) defined WAA as pre-existing or concurrent asthma that is worsened by workplace conditions, and four diagnostic criteria that need to be considered. These were **(i)** the presence of pre-existing or concurrent asthma, **(ii)** a temporal relationship between asthma and work, **(iii)** conditions at work that can exacerbate asthma and **(iv)** that occupational asthma is unlikely.

Question B: what is the prevalence of work-aggravated asthma?
WAA was identified to be very common. Many articles were cited within the reviews and prevalence estimates varied. Studies varied in their populations, geography and the definition of WAA used. Henneberger (2011) estimated a median prevalence amongst all those with asthma of 21.5%, with a range of 13% to 58%.

Question C: which causative agents are associated with the onset of WAA?
Many possible occupational exposures that were potentially able to cause WAA were mentioned in the review articles, and summarised in Table 3. The general categories of exposures identified were inhaled agents (allergens and non-allergens), physical exposures (for example extremes of temperature) and behavioural states (for example stress). In terms of inhaled non-allergen exposures, the following were consistently mentioned; (i) general exposures to known irritant chemicals, dusts, vapours, fumes, gases, fibres, aerosols, volatile agents and (ii) specific exposures identified; construction dusts, perfumes, fragrances, ozone, sulphur dioxide, odours, pollens, moulds, ambient air pollution, wood dusts, endotoxins, environmental tobacco smoke, inorganic dust, mineral dusts, cleaning agents; including ammonia, hydrochloric acid, monochloramine, sodium hydroxide, quaternary ammonium compounds and monoethanolamine.

Question D: what are the risk factors associated with WAA?
Factors other than exposure to a known cause of WAA were difficult to identify. Certain were suggested (gender, asthma severity, ethnic origin) but data were partly contradictory, suggesting that more work is needed in a variety of worker populations and geographies.

Question E: what symptoms are associated with WAA?
WAA is associated with typical asthma symptoms (episodes of wheeze, chest tightness, cough and shortness of breath), reported to be worse at work (or on work days) and / or improved on rest days. Work-related nasal symptoms may also be present. Workers with WAA may also increase their use of asthma medication whilst at work. Aggravation of symptoms can be short lived over a number of hours or days, or can occur consistently every day.

Question F: how is a diagnosis of work-aggravated asthma made?
The diagnostic process was felt to be potentially difficult. Access to clinicians with an interest in occupational lung diseases and to standard investigations, only available in these secondary care based centres, would normally be required. Most importantly, occupational asthma due to sensitisation should be excluded. In brief here, the initial step in confirming a diagnosis of WAA is to clarify whether or not an individual worker has asthma (the British Thoracic Society SIGN Asthma Guidelines, for example, can assist here). Work-related asthma is then confirmed by establishing a temporal relationship between asthma and work. That is, workers will complain of worse asthma symptoms at work or on work days in comparison to rest days.

Once work-related asthma is established, the main distinction required is whether a worker has WAA or OA. Simple case information may assist making this distinction, including medical and occupational history and details of likely workplace exposures. Generally, however, it is not reliable to distinguish WAA from OA based solely on the medical and occupational history, and normally more investigations are needed. This normally includes a combination of lung function, allergy, challenge and other tests.

Serial peak expiratory flow (PEF) measures may be particularly useful to help diagnose asthma, and also to whether there are any changes in these measures that relate to workplace exposures. Allergy skin prick tests may also help if a worker is exposed to a high molecular weight allergen; a positive test might support a diagnosis of OA but these tests have to be interpreted carefully in light of all other case information. Challenge tests, either to known irritant exposures or to agents inhaled at work (specific inhalation challenge, SIC) will also assist the diagnostic process further, Again interpretation of these tests must also be in light of all other case information. For example, a negative SIC could be strong support for the presence of WAA, but these tests can have false negative results.

Exhaled nitric oxide and sputum cellular examination may also be useful when making a diagnosis of OA or WAA, but generally have less information and evidence supporting their use.

Question G: Are there any successful interventions for the prevention and treatment of WAA?
Improving asthma control by intervention at the individual level (improving treatment, ensuring treatments are being taken as prescribed, education relating to these issues) was effectively the single identified personal intervention, although it is not known whether standard asthma treatments are as effective for treating work-related symptoms as they are for treating non work-related symptoms. Workplace based interventions were discussed in general terms only. For example, reduction in exposures following workplace assessment was often cited, rather than immediate removal of the worker from the workplace. It was evident that “safe” levels of exposure to irritants may not be easy to derive, and that there are significant individual

differences in susceptibility between workers. Any potential job change should take into account not only the physical but also the financial and psychological benefits. Finally, education about these issues for the employer was also identified as important, given their central role in supporting and paying for any interventions.

Question H: Is there a socio-economic burden associated with WAA?
There are good data to support the fact that WAA is associated with a significant socioeconomic burden. Job change, job loss and reduction in work efficiency were all associated with WAA, and in many cases the sizes of these effects were similar to those seen in occupational asthma. There are significant personal, workplace and societal financial gains to be made by reducing the burden of WAA.

CONTENTS

INTRODUCTION

There is substantial clinical and epidemiological evidence to suggest that inhaled agents at work can cause asthma in workers, or aggravate existing asthma. Estimates suggest that, on average, between 9-15% of all adult asthma is caused by these exposures (Blanc 1999, Balmes, Mannino, Meredith).

The term **work-related asthma** includes (i) **occupational asthma (OA)**, caused by an allergy to an agent encountered at work, (ii) **irritant induced asthma (IIA)** caused by exposure to an irritant in the workplace and (iii) **work aggravated asthma (WAA)**. IIA, not dealt with in any more detail specifically in this report, normally develops after an accidental single high dose exposure at work to an inhaled irritant. An example would be a high dose exposure to chlorine gas in a paper pulp mill. This might cause asthma symptoms to develop within 24 hours and persist for at least 3 months after the single exposure. A proportion of these individuals might consequently develop persisting asthma; termed IIA.

The presence of WAA, the subject of this review, implies that pre-existing asthma is made worse (aggravated or exacerbated) by a particular exposure at work, but that the asthma was not actually caused by that exposure. This would include workers previously diagnosed with occupational asthma who have moved to a new workplace where the current irritant exposures may aggravate their asthma control. WAA is thought to be more common than OA, and can affect a high proportion of working asthmatics (Henneberger 2007). Given that WAA is commonly identified from a series of studies that will be reviewed here, it is perhaps rather unusual that it has not been the subject of investigation or research in as much detail as occupational asthma or irritant induced asthma.

In May 2012, HSE, together with the Asthma Partnership Board (APB), reviewed its current approach to occupational asthma; they agreed that HSE's suite of interventions and focus of activity in relation to asthma caused by work was appropriate. However, it was agreed that there was a potential evidence gap in relation to WAA.

This report consequently reviews all relevant review articles available regarding WAA from the last five years. If deemed to be of exceptional significance, earlier reviews were also included. The aim of this report was to summarise these reviews and provide a narrative on the factors that can aggravate pre-existing asthma in the workplace. Whilst this process did not include a formal gap analysis, knowledge gaps are identified where appropriate.

METHODOLOGY

A literature search was carried out to identify review articles concerning work-aggravated asthma (WAA). The evidence in this report was taken from the output of this literature search, focused in particular on answering specific questions, listed below. Where individual reviews had duplicate or similar summaries in relation to these questions, examples were cited in the text, rather than noting all the evidence from each review, in order to make this process more concise.

A priori questions used;

A. Is there a definition of WAA? If yes, what is it?
B. What is the prevalence of WAA?
C. Which causative agents are associated with the onset of WAA?
D. What are the risk factors associated with WAA?
E. What symptoms are associated with WAA?
F. How is a diagnosis of WAA made?
G. Are there any successful interventions for the prevention and treatment of WAA?
H. Is there a socio-economic burden associated with work-aggravated asthma?

The literature was searched using MEDLINE, Google Scholar and Google. The search terms were agreed by the study team to be as follows;

- Work aggravated asthma
- Work aggravated asthma review
- Asthma made worse at work
- Asthma made worse at work review
- Work exacerbated asthma review

The main conclusions, following review of the literature, were recorded into an evidence table (Table 2) incorporating a summary of each paper.

RESULTS

All titles identified from PubMed were taken into consideration for this review, whilst the first 20 web pages of both Google Scholar and Google articles were assessed for suitable articles. As this process was a review of review articles, no critical appraisal or grading of the strength of the evidence of the data was undertaken. Table 1 illustrates the overall total numbers of identified results from the search strategy.

Fifty-two abstracts were reviewed, from which 19 papers were obtained and assessed, with 16 of these included in the final evidence tables. Those finally included were all review articles that were written either specifically about WAA, or contained a section relating to WAA within the paper. Three of the sixteen articles identified were specifically related only to WAA, rather than more general reviews of work-related asthma (Henneberger, 2007; Vandenplas, 2007; Henneberger, 2011). Table 2 details the evidence taken from each of the 16 relevant reviews. None of the reviews systematically graded evidence relating to the peer reviewed publications each review summarised.

A. Is there a definition of work-aggravated asthma? If yes, what is it?
Various broadly similar definitions were identified in the reviews. The most recent definitive review (Henneberger, 2011) defined WAA as "pre-existing or concurrent asthma that is worsened by workplace conditions". Concurrent asthma in this context was defined as "asthma with onset while employed in the worksite of interest, but not due to exposures in that worksite".

Additionally, Henneberger proposed four diagnostic criteria as follows: **(i)** the presence of pre-existing or concurrent asthma, **(ii)** a temporal relationship between asthma and work, **(iii)** conditions at work that can exacerbate asthma and **(iv)** that occupational asthma is unlikely. All other reviews offered similar definitions, and some restricted themselves only to pre-existing asthma, rather than including concurrent asthma (Lemiere 2007 cited two such examples; the SENSOR reporting scheme definition (Jajosky) and the ACCP definition (Chan-Yeung)).

B. What is the prevalence of work-aggravated asthma?
It was evident that WAA is common, with large numbers of workers with coincident or pre-existing asthma developing significant worsening or aggravation of their symptoms due to workplaces exposures. Prevalence estimates varied according to the populations studied. For example, Vandenplas (2003) summarised the prevalence estimates available as follows; in groups of patients who had been hospitalised with asthma an estimate of WAA prevalence of 20% (Timmer, 1993), in general practice population a prevalence of between 5-17% (De Bono, 1999; Milton, 1998) an estimate of 20-57% in population based surveys (Abramson, 1995; Balder, 1998; Blanc, 1999; Johnson, 2000) and 16-31% in tertiary asthma clinics (Axon, 1995; Tarlo, 2000). Henneberger (2011) reported a median prevalence estimate of 21.5%, and a range represented by the 12 included studies of between 13% to 58%.

The latter, highest, value was derived from a population of working adults with asthma from a primary care based study of persistent asthma cases (Mancuso, 2003). The studies used to derive these data varied in their populations, geography and the definition of WAA used.

Prevalence estimates quoted generally relied on self-reported asthma symptoms, and not on more objective measures of worsening asthma control such as serial peak expiratory flow (serial PEF) measures. However, three studies were reported that included description of more objective diagnostic criteria for WAA, where either workers were interviewed, measured serial PEF or reported work-related symptoms, medication use and the view of an expert panel

regarding exposure to asthma agents at work was sought (Caldeira, 2006; Bolen, 2007; Henneberger, 2006). These studies reported WAA prevalences of 13%, 14% and 22% respectively, with a median of 14%.

C. Which causative agents are associated with the onset of work-aggravated asthma?
Many reviews listed potential causes of WAA, although separate referencing of the evidence for each of these was not usually included or was variable. However, in order to provide a summary, all potential mentioned causes of WAA are included in Table 3. Various specific exposures and comments are highlighted in this section.

Exposure to cold, dry air, dusts, fumes, aerosols, and exertion were felt to be common in the workplace and able to aggravate asthma symptoms, particularly if the worker was not prescribed or taking optimal asthma medication (Mapp, 2005). Henneberger (2011) summarised that a wide variety of conditions at work could aggravate asthma symptoms as well as those mentioned by Mapp (2005) including; irritant chemicals, common aeroallergens, emotional stress, workplace temperature and physical activity. Interestingly, industries associated with WAA, such as wholesale and retail trade, public administration, transport and public utilities, differed from those classically associated with OA and IIA.

D. What are the risk factors associated with work-aggravated asthma?
This section details individual risk factors associated with WAA, other than the exposure related risks dealt with in section C. The review articles in general reported very few specific risk factors.

Asthma severity may be important as a risk. Vandenplas (2003) summarised that WAA symptoms may reflect exposure to higher than permissible levels of irritants at work, more severe underlying asthma or inappropriate asthma treatment (Malo, 1999). However, conflicting studies cited in the Vandenplas (2003) review observed that subjects with milder asthma had symptoms of WAA (Tarlo, 2000; Larbanois, 2002). No more information are given in these reviews relating to severity.

A further US based study comparing WAA with OA suggested that those with WAA were younger, included a higher proportion of females and non-white workers and those who were more exposed to inorganic dusts (Goe, 2002). Interpretation here is difficult, as these may relate to the demographic of those already in certain jobs. This group also reported that the risk of WAA was highest in those working in public administration (Goe, 2004).

The 2007 Henneberger review cited US data from a health maintenance organization (HMO) suggesting that WAA cases were more likely to be male (Henneberger, 2006), although data from Washington compensation cases of WAA noted an increase in females and less specialist input in comparison to OA cases. Canadian data (Lemiere, 2006) identified less atopy in those with WAA in comparison to those with OA, although both groups had high levels of measured atopy.

The American Thoracic Society statement (Henneberger, 2011) summarised that individuals with WAA were similar to those with OA in respect of asthma severity, dose of asthma treatments and emergency health care use.

Overall, these comments suggest that there were few identified differences between WAA and OA, and whilst certain studies did identify differences, with some conflicting evidence, the studies were carried out in differing geographic and industrial sector settings.

E. What symptoms are associated with work-aggravated asthma?

Symptoms of WAA were reported to be identical to those seen in normal, non work-related asthma (episodes of wheeze, chest tightness, cough and shortness of breath) and also resembled OA given that both are normally reported as being work-related. That is, the symptoms are described as worse at work, and / or better during periods away from work. It was also identified that symptoms in WAA may begin several minutes or hours after relevant exposures occur at work, and could range from a single short term worsening up to daily consistent worsening (Tarlo 2009). Workers may also describe reduced levels of medication needed to control asthma when not at work, or improvement in symptoms when workplace exposures are reduced or eliminated. Workers may also complain of nasal and eye symptoms that are worse at work (Friedman-Jimenez, 2000; Tarlo, 2009).

In more detail, Henneberger (2011) described a clinical series in which specific inhalation challenge was used to distinguish WAA from OA. The investigators found similar asthma severity scores and daily dose of inhaled corticosteroids for both conditions suggesting similar severity of disease in both groups. The level of bronchial hyper responsiveness (or lung "twitchiness" often seen in asthma) was either the same or lower in those with WAA. Similarly, US based data identified that levels of attendance to the emergency room, and being admitted to hospital with asthma, were the same in OA and WAA.

Finally, and not strictly related to symptoms themselves, two epidemiological studies conducted with HMO members reported that the proportion of days with reported asthma symptoms was higher in those with WAA than those with asthma that was not made worse at work (Henneberger, 2002; Henneberger, 2006).

F. How is a diagnosis of work-aggravated asthma made?

From the findings and content of the identified reviews, the diagnostic process was thought to be potentially difficult, and particularly so when a clinician did not have access to a full set of appropriate diagnostic tests. Whilst many of the reviews discussed in detail various diagnostic approaches, a single agreed or consensus approach was not identified. This section briefly summarises the salient points identified, as the exact diagnostic testing detail is not within the scope of this review of reviews.

In general terms, the initial step in confirming a diagnosis of WAA is to clarify whether or not an individual worker has asthma. This part of the diagnosis would be no different from confirming a diagnosis of asthma that does not relate to work. A good example of such an approach is found in the British Thoracic Society Asthma Guidelines (BTS SIGN asthma guidance). Once a diagnosis of asthma is confirmed, it is important to date its likely onset in relation to potential exposures in the workplace.

Work-related asthma is then confirmed by establishing a temporal relationship between asthma and work. In practice, this means that workers will complain of worse asthma symptoms at work, or at least on work days, in comparison to rest days, rest day improvement of asthma symptoms, and / or increased use of asthma medication to control symptoms on work days.

Once work-related asthma is established, the main distinction required is whether a worker has WAA or OA. Simple case information may assist making this distinction; this might include (i) the presence of a latent period (time period with no symptoms exposed at work before asthma and work-related asthma symptoms commence, during which allergy is developing to an agent at work) that might support OA and (ii) exposure to a known cause of OA, that might of course additionally support this diagnosis.

Sources of information to help identify relevant exposures, as such material safety data sheets (MSDS), may be useful, in addition to workplace generated hygiene reports and extended health

information (symptoms in colleagues, for example) that may be available. Given this, it was also summarised that the identification of a cause of WAA is not always possible (Henneberger, 2011).

Generally, however, it was not thought reliable to distinguish WAA from OA based solely on the medical and occupational history (Lemiere, 2007). The 2008 expert panel (Tarlo, 2008) consensus document concluded that WAA should be considered in any patient with worsening asthma and / or who has work-related asthma symptoms, and should then be investigated in a similar way to potential OA, using a combination of investigations that would be available to clinicians with an interest in work-related asthma. In addition to medical and occupational history as highlighted above, other potentially useful diagnostic processes include the use of lung function, allergy, challenge and other tests. These will be briefly summarised below;

(i) Lung function tests; simple measures such as FEV_1 and FVC are useful to document, but do not help distinguish between WAA and OA. Serial peak expiratory flow (PEF) measures may be useful to help diagnose asthma if there is an increase in the variation of these (increased diurnal variation, Henneberger (2002)), and will assess whether there are any changes that relate to workplace exposures. Such changes (for example increased variation in PEF) have been seen in those with WAA (Chiry, 2007). Serial PEF measures can also be measured on return to a work environment, following a break from exposure that potentially caused asthma symptoms (Friedman-Jimenez, 2000).

(ii) Tests of allergy such as skin prick testing or specific IgE are potentially useful if a worker is exposed to a high molecular weight allergen (such as flour or work with laboratory animals). A positive test might support a diagnosis of OA, but also could reflect exposure only in an atopic (or "allergic") individual. Negative tests may help exclude allergy to certain agents but can also be difficult to interpret in certain circumstances.

(iii) Non-specific bronchial challenge testing can be carried out either at a single time point, or during periods of work and rest, to assess any possible work influence on this measure (Tarlo, 2009). These tests cannot reliably distinguish between WAA and OA, although one further review noted that this test might assist in making this distinction (Mapp, 2005). Tarlo (2009) also made the point that certain exacerbating factors such as cold air or strenuous physical activity at work were less likely to be associated with a change in non-specific bronchial hyper reactivity than that seen following exposure to irritant inhaled exposures.

(iv) Specific bronchial challenge to particular workplace allergens may be useful diagnostically (for example challenging a baker with inhaled flour dust, and subsequently assessing the response with regular breathing tests). Henneberger (2011) identified that a negative test would be strong support for a diagnosis of WAA in comparison to OA. However, specific challenge tests can have false negative results if, for example, the incorrect exposure is used, or the worker has been removed from a suspected relevant exposure for a long duration prior to the challenge. In addition, specific challenge testing is also not widely available and ultimately this limits its use, given that WAA is so common (Lemiere, 2007).

(v) Other tests including exhaled nitric oxide and sputum cellular examination were described, but their utility is yet to be fully understood, particularly when making a diagnosis of WAA. Significant alternate diagnoses that should be considered in addition to OA include; chronic obstructive pulmonary disease, hyperventilation, cough and rhinitis, bronchiolitis, hypersensitivity pneumonitis, eosinophilic bronchitis, vocal cord dysfunction and multiple chemical sensitivity syndrome (Cartier, 2011).

G. Are there any successful interventions for the prevention and treatment of work-aggravated asthma?

This question highlighted significant gaps in the knowledge both at the individual and workplace levels.

Individual interventions largely appear to have centred on ensuring that pharmacological and non-pharmacological control of asthma is ideal for each worker. Vandenplas (2003) noted that the effects of standard asthma treatments on symptoms aggravated at work was not well understood, and in particular whether asthma medication has the same efficacy when used for work-related asthma symptoms in comparison to non work- related symptoms.

Henneberger (2011) also summarised a study by Lemiere (2007) in which subjects with work-related asthma, both WAA and OA, visited their physician or emergency department more often than those with asthma with no work-related symptoms. However, WAA was not associated with higher levels of exacerbation (or worsening of asthma symptoms) than OA, and both conditions required less medical resource after being removed from the causative exposure in the workplace. Henneberger (2011) concluded that when an individual worker with WAA can no longer tolerate the work environment, the clinician and worker should look carefully at the potential benefit of removal from work in comparison to the benefits of continuing in work; taking into account not only the physical but also the financial and psychological benefits. This balance has to be judged in every case, and the employer also has certain responsibilities to control exposure to hazards if these are thought to have an associated risk to the health of those with asthma.

Ensuring the worker was well educated and compliant with asthma medication was also thought to be important when aiming to keep workers with WAA in their jobs (Mapp, 2005; Tarlo, 2009), as such interventions may prevent the individual from having to leave the workplace.

In terms of the wider issue of workplace interventions, there were little data presented. Vandenplas (2003) advised that although known irritants should be kept to acceptable limits in workplaces, most of these limits have been extrapolated from animal experiments or from healthy populations. As a consequence, exposure levels required to protect workers from asthma developing WAA may be difficult to derive, and will likely differ between workers given differences in individual susceptibility.

Mapp (2005) specifically stressed that one of the important reasons why WAA should be distinguished from OA was because interventions and preventative measures may differ in their nature. Primary prevention of WAA could be achieved by occupational hygiene measures such as substitution of hazardous material, reducing workplace exposure to respiratory irritants by using a combination of exposure control techniques; improved ventilation or appropriate use of protective equipment.

Given that most of the primary workplace based interventions are usually designed and paid for by the employer, it was felt important to educate workplaces about the benefits of these, including improved productivity, reduced sickness absence and reduced presenteeism, given that these may all be motivators to change the practice of employers (Henneberger, 2011). Henneberger described one study that looked at long-term outcomes in both WAA and OA at an interval of one to four years after diagnosis. Asthma symptoms, as well as functional indices and sputum cells, were all reassessed. All of the individuals, except one worker with OA, were removed from the exposure that had caused their symptoms. Both groups of workers showed significant and equivalent improvement in their symptoms scores, although those with WAA showed a trend towards less improvement in airway hyperresponsiveness (a marker of asthma

severity) compared to the OA group, and also had a smaller reduction in their dose of inhaled corticosteroid. Interestingly, those with WAA showed an increase in sputum neutrophil counts compared to those with OA, the latter group displaying an increase in sputum eosinophils (Malo, 2011).

In summary, various interventions were described at a personal and workplace levels with varying methodologies and degrees of reported success. Central to the success of persuading the employer to deliver such workplace interventions appears to be the development of appropriate education and training that makes the business case for such approaches.

H. Is there a socio-economic burden associated with WAA?

Vandenplas (2003) identified that despite the large number of workers with asthma potentially affected, very little attention has been given to the study of the socio-economic impact of WAA. In the few studies that have been carried out, WAA appears to be associated with a considerable socio-economic impact. Unemployment rates have been found to be equal and high in workers with OA, work aggravated asthma and those with asthma not associated with work (31-39%, Cannon, 1995). However, Larbanois (2002) reported a more frequent reduction in income in those with WAA (65%) and occupational asthma (62%) compared to those workers with asthma unrelated to work (38%). Job change or work loss due to asthma was seen in very high levels for both WAA (54%) compared to those workers with occupational asthma (52%) (Larbanois, 2002). Henneberger (2011) stated that several studies have found that WAA is associated with similar rates of unemployment as occupational asthma and this ranges from 30-50% (Larbanois, 2002; Cannon, 1995).

Most previous available studies (Tarlo, 2000; Henneberger, 2006; Breton, 2006), apart from one (Henneberger, 2002), found that absenteeism (days away from work) was similar in asthma unrelated to work and work aggravated asthma. Presenteeism (being at work but unable to work to full capacity) has been assessed in only one study (Balder, 1998), which concluded that those with work-related asthma symptoms had a reduction in ability to work compared with those who did not report worsening asthma at work.

Two published case series described workers with WAA as having persistent work-related symptoms rather than short periods with symptoms that were related to work (Larbanois, 2002; Cannon, 1995). The frequency and magnitude of self reported symptoms and reduction in earnings were reported to be similar in WAA and in occupational asthma (Larbanois, 2002).

The available evidence suggests that WAA is associated with a significant socioeconomic impact.

Table 1; Number of documents identified for each search term.

Search Term	PubMed	Google scholar	Google
Work aggravated asthma	40	19,900	1,740,000
Work aggravated asthma review	13	22,900	704,000
Asthma made worse at work	56	67,200	6,530,000
Asthma made worse at work review	12	48,500	4,390,000
Work exacerbated asthma review	27	34,400	799,000

Table 2; the evidence from each of the 16 reviews included

Author	Journal & Year	Title	Review paper	Authors main conclusions
Vandenplas O, Toren K, Blanc PD.	Eur Respir J. 2003 Oct; 22 (4):689-97.	Health and socioeconomic impact of work-related asthma.	Yes	Prevalence estimates of work-aggravated symptoms vary largely according to the populations studied; 5-17% in general practice, 16-31% in tertiary asthma clinics, 20% in patients hospitalised for asthma and 20-57% in populations based surveys. These findings provide convincing evidence that workplace exposure can cause exacerbation of respiratory symptoms in a substantial proportion of subjects with pre-existing or coincident asthma. However, most of these findings refer to subjective worsening of asthma symptoms at work without objective lung function correlates. Most controlled experiments amongst asthmatic volunteers failed to demonstrate a physiologically relevant effect of exposure to irritant substances at permissible levels on airway calibre, level of non-specific bronchial hyper responsiveness or airway inflammation. Several studies have also identified considerable discordance between perceived symptoms and physiological parameters suggesting that lung function tests alone may not adequately gauge the impact of exposure to irritant substances. The environmental and host factors that determine worsening of asthma symptoms at work should be identified to enable the medical management of work aggravated asthma (WAA) symptoms. It has been proposed that higher than permissible levels of irritants at work can aggravate asthma, and that work aggravated asthma is due to inappropriately treated asthma or more sever underlying asthma. However other studies have observed workers with work aggravated asthma that have milder symptoms than their colleagues without worsening symptoms at work. One US study found that those with work-aggravated asthma are younger and include a higher proportion of females and non-whites and are more often exposed to inorganic dust than those with occupational asthma.
Zock J-P, Vizcaya D, Le Moual N.	Curr Opin Allergy Clin Immunol. 2010 April; 10(2): 114-120.	Update on asthma and cleaners.	Yes	This paper summarises the recent literature on the relationship between cleaning exposures and respiratory health, in particular asthma. It includes reviews, epidemiological studies, surveillance programmes and exposure studies. It concludes that many cleaning agents are respiratory irritants and some have sensitising properties. Relevant exposure levels to volatile compounds released from cleaning products have been reported to occur during common cleaning activities both in the workplace and in the domestic environment. The most important products that have been

				repeatedly reported include products in spray form, chlorine bleach and other disinfectants. These have been associated with both new-onset and work aggravated asthma.
Tarlo SM, Liss GM, Blanc PD.	Pol Arch Med Wewn. 2009; 119(10):660-666.	How to diagnose and treat work-related asthma.	Yes	Work-related asthma is common. It describes the occurrence of work aggravated asthma as affecting up to 25% or working asthmatics, and that this can be caused both by chemical exposures and physical conditions such as change in temperature or exertional demands. Work aggravated asthma can range from a single short term worsening of asthma at work up to daily worsening at work on a consistent basis. It should be investigated in a similar manner as for sensitiser induced occupational asthma. This could include allergy tests when appropriate, peak flow monitoring and or methacholine challenge during work periods and away from work to assess the presence of work relationship. It notes that some exacerbating factors such as cold air or exercise are less likely to be associated with a shift in methacholine responsiveness compared to common aeroallergens. It summarises that management of work aggravated asthma includes optimising asthma control by reducing exposure to relevant asthma triggers both at work and off work as well as pharmacological asthma treatment. Hygiene measures such as improved ventilation and use of appropriate respiratory protective equipment should also be considered.
Henneberger PK, Redlich CA, Callahan DB *et. al.*	Am J Respir Crit Care Med. 2011; 184:368-378.	An official American Thoracic Society Statement: Work Exacerbated Asthma.	Yes ATS statement	Main conclusions of this statement are; that WAA is defined as pre-existing or concurrent asthma that is worsened by workplace conditions. Epidemiology studies conducted in general populations indicate that WAA occurs in a median of 21.5% of adults with asthma. Many conditions at work can aggravate asthma symptoms including; irritants, chemicals, dusts, second hand smoke, common aeroallergens, and other 'exposures' such as stress, temperature and physical exertion. Workers with WAA resemble those workers with occupational asthma in respect to severity of asthma and medication requirements as well as socio-economic factors such as unemployment and loss of income from work. Compared with asthma unrelated to work WAA is associated with more symptomatic days, a greater utilisation of health care resources and a lower quality of life. WAA should be considered in any patient with asthma who works. There is little evidence relating to the natural history of WAA, although avoidance or reduction of exposure can often lead to improvement in asthma symptoms. Final conclusion that WAA is a common and under recognised adverse outcome resulting from conditions at work. Additional research is needed to improve the

				understanding of the risk factors for, and mechanisms and outcomes of, WAA, and to inform and evaluate preventive interventions.
Tarlo SM, Balmes J, Balkissoon R *et al*.	Chest 2008; 134:1S-41S.	Diagnosis and management of work-related asthma.	Yes American College of Chest Physicians consensus statement	Consideration of WAA should be given to all individuals who present with new-onset or worsening asthma, followed by appropriate investigations and intervention including consideration of other exposed workers. In individuals with irritant-induced or WAA treatment should be optimised and exposure to workplace triggers reduced. If this is not successful then a change in workplace is suggested to control asthma.
Malo J-L.	Occup Med 2005;55:606-611.	Future advances in work related asthma and the impact on occupational health.	Yes	Defines WAA as asthma that worsens at work. States there is little known about the condition. Review concentrates on occupational asthma from sensitisation.
Friedman-Jiménez G, Beckett WS, Szeinuk J, Petsonk EL.	Am J Ind Med 2000;37:121-141.	Clinical evaluation, management and prevention of work related asthma.	Yes	WAA is diagnosed in individuals with symptomatic asthma that is significantly worsened by workplace environmental exposures. This includes people with pre-existing asthma, as well as those with new onset asthma, as long as the asthma is clearly aggravated by environmental exposures in the workplace. The symptoms are the same as those for other forms of asthma; recurrent episodes of cough (productive or non productive) wheeze, chest tightness, and shortness of breath. Patients sometimes present with cough variant or nocturnal asthma. WAA appears to be common and may cause a substantial preventable burden of disability but little has been published on pathophysiology, diagnosis, management or prognosis. The important characteristic of WAA is that exposures in the workplace worsen asthma control. WAA may be less obvious with a late or dual asthmatic reaction, which may occur many hours after the exposure. Concludes that distinguishing between WAA and OA important and finding the specific cause of WAA if present can be helpful in guiding both clinical and public health management.
Lemiere C.	Expert Rev Resp Med 2007;1(1):43-	Occupational and work exacerbated asthma: similarities	Yes	Defines WAA in terms of an epidemiological definition and a clinical definition. Epidemiological definition usually relies on the self-reporting or work related symptoms or job changes because of breathing problems. SENSOR criteria are:

	49.	and differences.		▪ Diagnosis consistent with asthma by health care professional ▪ Symptom-work association ▪ Asthma symptoms or treatment with asthma medications for 2 years prior to entering the new occupational setting ▪ Increased asthma symptoms or increased asthma medications since staring new occupation. This definition only includes those with pre-existing asthma. The clinical definition by the ACCP is a diagnosis of asthma and association between asthma symptoms and work exposure as indicated by ▪ Presence of asthma symptoms or related medication before entering a new exposure setting. ▪ Increase in symptoms or the need for more medication after entering a new work setting. Does mention more current guideline from ACCP will not restrict WAA to those with pre-existing asthma. Based on 5 epidemiological studies, the prevalence of WAA based upon self reported worsening of asthma was 18-34%. Concludes; work related asthma is a highly prevalent condition; OA and WAA are difficult to differentiate in clinical practice; individuals with OA seem to have predominantly an eosinophilic type of inflammation when at work, whereas those with WAA tend to have a neutrophilic type of airway inflammation; individuals with OA see a greater improvement of symptoms after removal from exposure than those with WAA; the socioeconomic outcome of those with OA and WAA seems to be similar (both poor) but more data is needed to confirm this.
Smith A.	Immunol Allergy Clin N Am 2011; 31:663-675.	The epidemiology of work related asthma.	Yes	This review defines WAA but then goes on to discuss mostly OA from sensitisation.
Malo JL, Vandenplas O.	Immunol Allergy Clin N Am 2011; 31:645-662.	Definitions and classification of work related asthma.	Yes	This review defines the different conditions that are defined under the term work related asthma including work-aggravated asthma.
Cartier A, Sastre J.	Immunol Allergy Clin N	Clinical assessment of occupational	Yes	It explains how work related asthma can be categorised into OA and WAA. Defines WAA as worsening of pre-existing asthma or coincident (new-onset) asthma by

	Am 2011; 31:717-728.	asthma and its differential diagnosis.		workplace exposures. The review summarises that although it is difficult to distinguish WAA from OA, a combination of tools will help the clinician to make a diagnosis. Specific inhalation challenge performed in the workplace or laboratory is considered the gold standard to confirm diagnosis.
Smith AM, Bernstein DI.	J Allergy Clin Immunology 2009; 123:551-557.	Management of work related asthma.	Yes	Little published evidence regarding the identification and management of WAA. Important to make correct diagnosis so management is appropriate.
Avila PC, Shusterman DJ.	Postgraduate Medicine 1999; 105(7);39-46.	Work related asthma and latex allergy.	Yes	Very small section (one paragraph) defining WAA.
Henneberger PK.	Curr Opin Allergy Clin Immunol 2007; 7:146-151.	Work exacerbated asthma.	Yes	Review to summarise recent finding on WAA during 2005/2006. Seven articles were used in this review, which concluded that there was general agreement regarding the definition of WAA. However, operational definitions and prevalence estimates varied considerably amongst the studies. Despite this they all led to the conclusion that WAA is common. Numerous conditions at work can exacerbate asthma including; behavioural states, odours, second hand smoke, and physical factors such as extreme temperature change and exercise. WAA cases share many demographic and clinical characteristics with other adults with asthma and occupational asthma, although some differences have been reported. The review recommends that further research is required on all aspects of WAA in order to improve the diagnosis, management and prevention of this condition.
Vandenplas O, Henneberger PK.	Curr Opin Allergy Clin Immunol 2007;7:236-241.	Socioeconomic outcomes in work-exacerbated asthma.	Yes	This review focuses on socioeconomic impact of WAA. It summarises the impact on work disability, income, healthcare costs and psychosocial impact. It concludes that the adverse effects of WAA on work productivity and earning of affected workers are similar to those with occupational asthma caused by sensitisation.
de la Hoz RE.	Curr Opin Allergy Clin Immunol 2011;11:97-102.	Occupational lower airway disease in relation to World Trade Centre inhalation exposure.	Yes	Included for completeness, this review summarised medical evidence relating to predominant airways disease seen as a consequence of the collapse of the World Trade Centre in exposed emergency and other workers.

Table 3; causes of WAA identified from the combination of reviews

Agent category	Specific agents
Inhaled; low and high levels of exposure to predominantly irritants	General exposures to known irritant chemicals, dusts, vapours, fumes, gases, fibres, aerosols, volatile agents. Specific exposures identified; constructions dusts, perfumes, fragrances, ozone, sulphur dioxide, odours, pollens, moulds, ambient air pollution, wood dusts, endotoxins, environmental tobacco smoke, inorganic dust, mineral dusts, cleaning agents; including ammonia, hydrochloric acid, monochloramine, sodium hydroxide, quaternary ammonium compounds and monoethanolamine.
Inhaled; exposures to known and likely sensitisers	Common aeroallergens and occupational allergens (e.g. flour dust, wood dusts). Specific exposures identified; inorganic dusts, cleaning agents with particular relevance to amines, aldehydes, quaternary ammonium compounds, scents with terpines, isothiazolinones, formaldehyde, latex, animal danders.
Physical work environment	Temperature extremes, changes in temperature, physical exertion and varying physical demands of work tasks.
Other	Stress, behavioural states.

DISCUSSION

Asthma is a very common adult respiratory problem in GB. Aggravation of unpleasant asthma symptoms at work has also been identified to be common. Such aggravation can occur due to inhaled exposures, changes in the physical environment (for example workplace temperature changes that workers with asthma may tolerate poorly), or certain other factors including behavioural issues. This is termed work aggravated asthma (WAA).

The findings of this review, relating specifically to WAA, have implications for workers with asthma, those with asthma who wish to choose a career or return to the workplace, employers, health care professionals, and policy makers and legislators.

The implications for workers with asthma are self-evident. Unpleasant asthma symptoms made worse at work will adversely influence sickness absence, work efficiency and ultimately job retention. Indeed, this review has highlighted significant personal and socio-economic impacts related to WAA. Consequently, all interventions designed to reduce WAA will potentially improve these varying impacts.

The implications for those with asthma who wish to choose a career, or return to the work environment following absence, are also clear. Ensuring that risks are minimised for those with asthma commencing work is important, although details of interventions to reduce WAA specifically are not easy to identify from the reviews covered in this work. It is important that employers and workers are educated in relation to possible causes of asthma aggravation at work, and that those entering work for the first time with asthma also have some knowledge of the potential risks to their asthma control posed by the workplace.

The implications for employers are at least three fold. First, it is their responsibility to adhere to local and national legislation designed to minimise risks associated with various (inhaled and other) workplace hazards that may influence respiratory health. Risk assessments should, where possible, also consider workers with asthma and in particular if workers with asthma have WAA. Second, it would seem appropriate to educate workers with pre-existing asthma at work about the risks posed by these hazards to their health, and specifically about the risks of WAA, and how to minimise these. Third, employers should be aware that certain industries associated with WAA, such as wholesale and retail trade, public administration, transport and public utilities, differed from those classically associated with occupational asthma.

The implications for health care professionals include the need to optimise asthma control where possible in all patients with asthma using standard guidance. For example, in GB, adherence to the British Thoracic Society SIGN guidance (BTS SIGN asthma guidance) will assist this process. However, in addition, all health care professionals should enquire about work when assessing adults with asthma, and in particular considering whether there is an agent at work that is causing asthma (OA) or aggravating asthma (WAA). If an occupational cause is suspected (by asthma symptoms that are worse at work, for example, referred to as work-related symptoms and seen in both OA and WAA) further specialist assessment should be undertaken (Fishwick, 2012).

The traditional model used to control exposure to inhaled agents at work relating to asthma is based on control of exposures that actually cause occupational asthma or irritant induced asthma. That is, risk assessment will identify exposure to hazardous agents that are capable of causing asthma, and a combination of approaches, including the hierarchy of control and health surveillance, where appropriate, is applied. The situation with WAA is different; whilst the approaches adopted to reduce occupational asthma or irritant induced asthma may reduce

exposures to agents that cause WAA, this is by no means certain. Reasons for this include different agents being responsible for causing WAA in comparison to OA or IIA, and lack of good exposure response information for exposures that specifically cause WAA. Any workplace-based policies designed to reduce the overall burden of asthma at work consequently need to consider not just OA and IIA, but WAA as well.

There are potentially very significant personal, health and socioeconomic gains to be made by developing simple practical interventions to reduce WAA.

CONCLUSIONS

The most recent definitive review defined WAA as pre-existing or concurrent asthma that is worsened by workplace conditions. Concurrent asthma in this context was defined as asthma with onset while employed in the worksite of interest, but not due to exposures in that worksite.

WAA is common. Large numbers of workers with coincident or pre-existing asthma can develop significant worsening or aggravation of their symptoms due to workplaces exposures. Prevalence estimates varied according to the populations studied. Henneberger (2011) reported a median prevalence estimate of 21.5% of all those with asthma, and a range represented by the 12 included studies of between 13% to 58%. Prevalence estimates based on studies with more objective evidence of WAA noted a range of 13-22%.

A wide variety of exposures and other influences can cause WAA, and include inhaled irritants, allergens, emotional stress, workplace temperature and physical activity levels.

Risk factors for WAA were difficult to identify and differentiate from those related to occupational asthma. Whilst certain studies did identify differences, with some conflicting evidence, these studies were carried out in differing geographic and industrial sector settings.

Symptoms of WAA were reported to be identical to those seen in normal, non work-related asthma; namely work related episodes of wheeze, chest tightness, cough and shortness of breath. These are identical to those seen in occupational asthma caused by workplace sensitisation. Consequently, investigations are normally required to either confirm or exclude a diagnosis of asthma, and subsequently a diagnosis of WAA. Making a diagnosis of WAA in a worker who has confirmed asthma largely involves excluding occupational asthma. This diagnostic process may be complex, and relies on access to specialist facilities and a combination of medical and occupational histories, knowledge of workplace exposures and medical investigations, including lung function, allergy tests and challenge tests.

Once a diagnosis of WAA is established, it is important to ensure that the worker is well educated about the need for, and to be compliant with, asthma medication, as this may prevent the individual from having to leave the workplace.

Workplace actions and interventions are also important here to reduce the symptoms associated with WAA, although there is no current evidence based approach to this. In order to achieve the best outcomes at work, it was felt important to educate workplaces about the benefits of interventions to reduce WAA, including improved productivity, reduced sickness absence and reduced presenteeism, given that these may all be motivators to change their practice.

In the few studies that have been carried out, WAA appears to be associated with a considerable socio-economic impact.

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HSE

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Work aggravated asthma: A review of reviews

Seven percent of the adult population have asthma, a condition commonly made worse by inhaling irritant exposures at work; termed work-aggravated asthma (WAA). A variety of factors that cause WAA have been identified by this review, including inhaled exposures, physical factors and behavioural issues.

WAA is common. A recent comprehensive review identified that more than 1 in 5 workers with asthma have this condition. There are, however, no GB based prevalence estimates.

WAA is an unpleasant condition. Workers complain of cough, wheeze, chest tightness and shortness of breath that is aggravated at work. These symptoms are likely to influence work absence, presenteeism and work efficiency. Their presence is also associated with significant adverse socio-economic impacts for workers and workplaces.

A variety of medical tests may be needed to help make a diagnosis of WAA, and distinguish this from occupational asthma. These tests are normally only available in specialised units with a particular interest in occupational asthma.

Very little GB data exists about interventions to reduce the associated burdens to the individual and the workplace. These include improving asthma treatments, worker education and training and assessing risks posed by various known hazards with particular relevance to asthma.

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