

Occupational asthma in cleaners: a challenging black box

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An abiding feature of current occupational epidemiology has been the observation—in Europe and North America—of high rates of asthma-like symptoms in professional cleaners.¹ In comparison with ‘referent’ occupational groups such as office workers, risk estimates of around 1.5 have repeatedly been reported.² Since there are very large numbers of people who work as cleaners one might expect these to be reflected in the experience of clinicians who see patients with asthma and in particular those with a special interest in work-related disease. This does not, however, appear to be the case. In the past decade, for example, fewer than 200 cases of occupational asthma (OA) in cleaners, most of them female, were reported to the UK national surveillance scheme for occupational respiratory diseases (SWORD).

In this context ‘OA’ refers to disease that is believed to have been induced *de novo* by an exposure encountered at work; it is distinguished from ‘work-exacerbated’ asthma, a term used to describe pre-existing or coincident disease that is provoked by one or more exposures in the workplace. OA usually reflects an immune response to an airborne sensitising agent and a small number of cleaning materials—notably so-called quaternary ammonium compounds—are recognised to be respiratory sensitisers. However, the clinical pattern of asthma among cleaners appears to deviate from the traditional picture of immune-mediated OA: there is a low prevalence of atopy, scarce bronchial reversibility and no evidence of eosinophilic airway inflammation and oxidative stress biomarkers.³ Beyond rare cases induced by presumed immunological responses to recognised sensitising agents, no definitive causal agents have been established, but chlorine bleach and ammonia have been frequently reported as potential irritant causal agents.^{1,4} Indeed, a large number of cleaning chemicals products would more usually be classified as

‘irritant’, readily capable of provoking short-lived symptoms that might be consistent with those of asthma but improbable causes of specific sensitisation. In SWORD reports, the agents suspected to be responsible for OA in cleaners have included generic terms such as ‘cleaning materials’, ‘sterilising’ or ‘disinfecting agents’, ‘biocides’, ‘fumes’ and ‘gases’ but also more specific agents such as bleach or chlorine, enzymes, diisocyanates and flour. OA may also develop as the result of an accidental high exposure to a non-sensitising respiratory irritant at work⁵ when it is generally described as ‘irritant-induced’ asthma. Although such exposures probably occur quite frequently in the context of cleaning—both domestic and industrial cleaning—no cases have been reported to SWORD. Whether repeated irritant exposures at lower levels can give rise to new asthma remains controversial, but at least in this context is clearly an important issue.

In a linked study, Vizcaya *et al*⁶ have tried further to elucidate some of the underlying mechanisms of asthma among cleaners using a panel study nested in the EPIASLI-2 cohort which was set up principally to study asthma in professional cleaners in Spain.⁷ Briefly, 21 female cleaners with asthma, selected from a previous case-control study nested in the same cohort, recorded in daily diaries over a 15-day period their exposures to a list of 14 irritant cleaning products and their respiratory symptoms; in addition they made thrice-daily (morning, afternoon and evening) measurements of forced expiratory volume in 1 s (FEV₁) and peak expiratory flow. The aim was to clarify the temporal sequence of short-term respiratory effects following occupational cleaning product exposures. The authors report that lung function was reduced soon after the use of irritant products such as hydrochloric acid and solvents, and that the observed decrease persisted or even worsened, through the morning following an exposure. In addition, they observed a positive trend with the number of *spray* products used. Interestingly, this effect was stronger in non-atopic than atopic cleaners with lower respiratory symptoms. Of note, atopic cleaners had fewer working hours with exposure to cleaning products limiting the possibility

of explaining this discrepancy as result of a different underlying pathogenic mechanism.

The main strength of this study is its panel design that allows the authors to draw probable causal conclusions about the short-term effects of cleaning exposures on respiratory symptoms and lung function. Major confounders such as smoking and acute respiratory infection were taken into account, but there is curiously little information on the type, frequency and doses of any asthma medication making it impossible to know whether any of the reported effects were modified by this important variable. Important limitations remain: the small sample size limits the generalisability of the results and it may be that the exposures encountered by some cleaners in Spain are unusual. Self-reported exposure assessments to cleaning products, even if recorded in daily diaries, cannot rule out a residual recall bias particularly for noxious agents with pungent odours such as bleach, solvents and ammonia. Specific inhalation challenge tests to establish a causal relationship between specific cleaning agents and respiratory outcomes were not performed. Finally, because the cleaners in this study already had a diagnosis of asthma the respiratory effects of cleaning products reported here can be interpreted only as disease provocation and tell us little about the mechanisms of any new ‘asthma’ induced by this occupation.

Most of these limitations are shared by previous similar studies and it remains difficult to overcome them because of the several peculiar challenges of studying asthma among professional cleaners. In this workforce recruitment and response rates are often low due to the reluctant collaboration of private employers, the typically rapid turnover of workers and the potential for job insecurity that may cause under-reporting of respiratory symptoms; cleaners are frequently a marginalised occupational group. Personal quantitative exposure assessment (ideally both external and internal) is difficult and limited by often incomplete knowledge of the specific agents, alone and in combination, in cleaning products. Product labels can be misleading and technical safety data sheet may not list all the hazardous agents present. Finally, specific inhalation challenge tests with suspected work triggers, considered the reference standard for the diagnosis of OA, may not be always feasible because of lack of specialised hospital centres with trained staff expert in state-of-the-art diagnostic methods and procedures.⁸ A collaborative

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effort with a multidisciplinary approach involving also molecular experiments could help overcome these limitations.

It is not unusual to study the association between an exposure and a health outcome before knowing the underlying causal agent (the so called 'black box' approach); most of the current known occupational carcinogens were discovered through this approach. Nevertheless if we want to implement efficient preventive interventions among professional cleaners it is requisite that we identify specific causal agents and/or exposures; and understand the underlying pathogenic mechanisms, in particular because irritant agents are likely to have a no adverse health effect threshold that could be used to establish limit values to protect exposed workers. Finally asthma in individual cleaners, if recognised early, could be potentially cured by avoidance of further exposure to the causal agents in the workplace; it is mandatory, for both public health and ethical reasons, that

further research investigations finish the work of unpacking this challenging black box.

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