

REVIEW

Systematic review of the effects of domestic paints on asthma related symptoms in people with or without asthma

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Introduction: The association of respiratory problems with chemical emissions from common indoor materials has been studied but the specific effect of domestic paint has not been widely investigated. The aim of this review is to provide a comprehensive evaluation of experimental, occupational and epidemiological studies that examine the adverse respiratory effects of exposure to domestic paints. **Methods:** A literature search of PubMed together with consultation with an expert resulted in 997 articles published up to May 2011 of which 3 experimental, 4 occupational and 13 epidemiological studies met selection criteria and were included in the review. **Results:** The occupational literature suggested that work as a (non-spray) painter increases the risk of asthma, but it provides little insight to the risks of painting in the domestic setting. All epidemiological studies, except one, reported associations with some asthma-like symptoms. In children at least there was evidence that painting was associated with wheezing, but the study designs and the exposure assessment were unsuitable to differentiate between acute and longer terms affects. We found the epidemiological evidence to be weak and certainly insufficient to infer causality. **Conclusion:** The variable quality of the exposure assignment and heterogeneous study design makes it difficult to draw firm conclusions on whether domestic paint exposures cause or exacerbate asthma in children and/or adults.

Keywords

Asthma, chemical, indoor environment, paint, respiratory, VOC

History

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Background

Indoor exposures are risk factors for developing or triggering asthma [1] and are of concern because people living in developed economies spend more than 80% of their time indoors [2]. Redecoration and home renovation have been reported to make asthma worse, possibly through exposure to paint fumes [1], and concerns over possible acute effects of domestic paint fumes are not uncommon on asthma-related websites and in chat-room conversations (http://blog.asthma.org.uk/does_paint_trigger_y.html 01 March 2012).

Domestic paints comprise three main ingredients: a pigment (with or without a filler), a binder which acts to dry or to cure the paint, and a solvent to control viscosity and stability. Binders include a variety of resins, synthetic or natural, such as acrylics, vinyl-acrylics and vinyl acetate/ethylene, or alkyds (including propylene glycol), polyurethanes, polyesters, melamine resins, epoxy, or oils. There are two main categories of paint: water-based (WB) paints where water is the main solvent or diluent; and solvent-based (SB), or “oil-based” paints which produce rapid and high emissions of well-known non-polar organic solvents. WB paints

accounted for 95% of the indoor paints sold in Sweden in the 1990s [3], but a comprehensive estimate in other European/non European countries is not available. Newly applied paint, while it is drying and/or curing, can produce high local concentrations of indoor volatile organic compounds (VOCs) and their emission may persist for long periods through a “sink” phenomenon [4] (when a substance is absorbed onto a surface and then slowly released over a period of time). The contribution of domestic paint to the total indoor concentration of VOCs is variable, depending on the type of paint used, the nature of the surface to which it is applied, other sources of VOCs and household ventilation.

Following a request from a national asthma charity to advise on the respiratory effects of indoor paint exposure, we conducted an initial screen of the literature and found there were very few studies relating domestic paint emissions to acute symptoms in persons with asthma. Two relevant reviews on indoor exposures (including painting) in children were identified. Heinrich [1] systematically examined the literature relating the development of childhood asthma to indoor air exposures including both painting and redecoration activities and VOCs. A second review, focussed on domestic VOC emissions, identified four studies investigating asthma-related outcomes and indoor, residential chemical sources, including recent painting and renovation [5]. Whether the major groups of indoor chemical exposures possess

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allergy-promoting (adjuvant) effects was also evaluated in another review [6]. Outside the occupational setting, the specific effect of paint and paint related VOCs has not been widely investigated. To the best of our knowledge, a systematic summary of the association of domestic paint exposure with asthma has not been attempted previously.

The aim of this review is to identify and evaluate published experimental, occupational and epidemiological studies that have examined provocation or induction of asthma and asthma-related symptoms (particularly wheezing) in relation to exposure to domestic paints. We were particularly interested in determining whether any studies had addressed exposure to “painting” rather than a generic exposure to “house decoration/renovation” and whether VOCs were measured as well as a recording of indoor painting.

Methods

We used the online bibliographic database PubMed to identify relevant published studies up to May 2011. Our search terms were: (“paint” or “VOC”) and (“asthma” or “wheeze” or “cough” or “lung” or “FEV1” or “PEF” or “bronchial” or “airway” or “respiratory”). Bibliographies of relevant retrieved studies and recent reviews were hand-searched for additional publications. The abstract of each was screened and those not published in English, relating solely to animal work, or where respiratory symptoms were just one of many symptoms of ‘multiple chemical sensitivity’ were excluded. For full review we included studies that encompassed one or more of the following criteria:

- (1) Experimental (“chamber”) studies of controlled exposure in humans with assessment of respiratory effects (particularly asthma);
- (2) Occupational studies of respiratory health in house painters; studies of other occupational exposures – largely of spray painters – were excluded;
- (3) Epidemiological studies in children or adults of associations between adverse respiratory effects (particularly asthma) and indoor residential exposure to paint with or without related indoor VOC measurements. Eligible studies were organized in broad categories defined by population age (children vs. adults), health outcomes (asthma, asthma symptoms, lung function) and type of exposure (indoor painting, redecoration including painting, VOC measurements in relation to painting). Reported associations between health outcomes and risk factors were abstracted where possible, as well as adjustment variables where multivariate analyses were conducted. We classified exposure assessment as 1 (=weak), when a non-specific “re-decoration” exposure (including painting) or total indoor chemicals exposure (including painting) was considered; 2 (=sufficient), when specific information on indoor painting exposure was obtained; 3 (=good), when levels of VOCs were measured as well as a recording of indoor painting.

We decided not to carry out a meta-analysis, given the different types of study designs, lack of clarity in what the “exposure” (i.e. redecorating) actually represents, inclusion/exclusion criteria, study design and statistical methods.

Results

We identified 997 articles that met our search terms. Of these, 25 original articles and 2 reviews met our inclusion criteria.

Experimental studies

We identified three human experimental studies (Table 1), the first of which [7] is most pertinent to the issue of acute responses to paint fumes. Seventeen UK adults with asthma (all treated with bronchodilators and 14/17 taking inhaled steroids), who reported a worsening of their symptoms in response to paint (or other strong) odours were recruited to a double-blind, cross-over, chamber-based clinical study funded by a paint manufacturer. The details and effectiveness of the “blinding” were not described. On separate days, each volunteer was asked to paint a board, for 60 min, with either a “conventional” WB interior paint or a “VOC-free” alternative. Symptoms and lung function were measured before, during and for 10 min after each challenge, all participants having been instructed to withhold their bronchodilators 6 h prior to the challenge. Median symptom scores remained low during each exposure but tended to be higher during the WB paint session. The increases were statistically significant only for “wheeze” and “unpleasant taste.” “Unpleasant smell” scores increased significantly during the VOC-free painting session alone. Lung function improved during exposure to both types of paint; and changes in spirometry were unrelated to symptoms. The authors concluded that neither paint would represent a substantial hazard. This well-designed small cross-over study did not include people without asthma – and it is possible that similar effects may have been seen in adults without asthma. The remaining two experimental studies are of a component – propylene glycol (PG) – of WB-based paints. Wieslander et al. [8] studied 27 Swedish adults without asthma, most of them airline pilots and a fifth of them smokers, and exposed them to a PG mist for 1 min (geometric mean concentration of PG was 309 mg/m³); no referent exposure was used. During this, 16% developed a cough; none reported any difficulty in breathing. Sore or itchy eyes and dryness of the throat were the most commonly reported symptoms. In the second study [9], 20 Swedish men, divided equally into those with and without atopy, and 10 Swedish housepainters with irritant symptoms from WB paint were exposed, on separate occasions, to mixtures of glycol and glycol ethers with and without additional ammonia or, by way of reference, to clean air. None of the participants had physician diagnosed asthma. Each exposure lasted 4 h, the total exposures being higher than those which might be expected during and after normal painting. Any resulting symptoms from the active exposures were mild; only those relating to eye irritation were significantly greater than during the referent exposure. There were no consistent increases in respiratory symptoms and no changes in pulmonary function or measurements of nasal inflammation (myeloperoxidase, eosinophilic cationic, protein, lysozyme, albumin and interleukin-8). Only minor differences between the three groups of subjects were observed. The authors concluded that exposure to the studied components (and to ammonia) induces “mild irritation in eyes, nose and airways”.

Table 1. Experimental ('chamber') studies.

Reference	Population	Study design	N	Exposure	Important findings	Limitations
Beach 1997 [7]	UK adults with asthma and symptoms provoked by paint or other strong fumes	Cross-over	17	60 min painting with conventional versus VOC-free WB paint	Median increase in "wheeze" and "unpleasant taste" scores during exposure to conventional paint (ANOVA, $p < 0.01$). Median increase ^a in "unpleasant smell score during exposure to VOC-free paint." (ANOVA $p < 0.01$), but not with exposure to conventional paint. For almost all symptoms there was a greater increase from 0 to 60 min in median symptom scores with the conventional than with the VOC-free paint, but only for "breathlessness" these differences were statistically significant. Lung function measurements showed a small but significant increase during the use of both paints ($p < 0.05$): FEV1/FVC% baseline 69, conventional paint 70, VOC-free paint 72. 16% developed cough. None developed difficulty in breathing.	Successfully blinded? Under-powered? No assessment of late reactions
Wieslander 2001 [8]	Adult volunteers (no asthma) – most of them Swedish pilots	Single exposure	27	1 min propylene glycol mist, clean air as referent exposure		
Ernstgård 2007 [9]	Swedish men: 10 with, and 10 without, atopy; 10 housepainters with irritant symptoms from WB paint. None with asthma	Cross-over	30	4 hours exposure to (separately) glycol/glycol ethers with and without ammonia, clean air	Only minor symptomatic ("irritant") responses which tended to be more common after the glycol and glycol/ammonia exposures; significant only for eye and nose irritation and not for "breathing difficulty" ^a . No change in lung function, markers of inflammation in nasal lavages, and renal function in urine. Symptoms tended to be greater in non-atopic volunteers.	

^ano estimates reported in the paper (only Figures).

Occupational studies

Four occupational studies that fulfilled the inclusion criteria were identified (Table 2).

Among almost 9500 participants in the European Community Respiratory Health Survey those currently working as “non-spray (‘other’) painters” were at an increased risk of bronchial hyper-reactivity and asthma symptoms or medication use (OR (adjusted for age, sex, smoking status, and study centre) 2.54, 95% CI 1.04–5.28) compared to those currently employed as office workers [10]. The number of such painters was small ($n = 65$) but their apparent increase in risk was the highest for any occupational group other than farmers; no further information on their exposures was available. In Finnish construction workers [11] current asthma or current cough/wheeze were significantly increased for outdoor, but not indoor, painting (asthma: OR (adjusted for age, smoking and atopy) 4.68, 95% CI 1.36–16.11; cough/wheeze: OR 2.88, 95% CI 1.21–6.86), when compared to carpentry workers. There was a nearly three-fold risk of asthma for the painters who used WB paints daily, but no clear associations with respiratory symptoms except for cough or phlegm. Exposure to hardwood and soft wood dust and to agents used during woodworking (e.g. adhesives, paints, stains, varnishes, stripping fluids) has been associated with asthma and using “carpentry workers” as a comparator in this study may have underestimated the increased risk of “outdoor painting”.

The remaining two occupational studies were of Swedish house painters. In the first [3] 23 painters with asthmatic symptoms, 9 painters with other lower airway symptoms, and 12 painters without airway symptoms were identified. Only those with bronchial hyper-responsiveness (arguably a proxy for asthma) reported airway symptoms in relation to exposure to paints, glass fibre or spray filling. The authors did not compare painters with and without asthma but instead examined the role of atopy. In painters with atopy, a significant relation between work-related airway symptoms and the use of WB, but not SB paints, was observed ($p < 0.05$). In addition, 18 male painters, none with asthma, had measurements of lung function before and after a shift during which they worked only with WB paint. Small but statistically significant decrements in FEV1 (130 ml) and FVC (140 ml) across the shift were noted. Symptoms were not reported, and the clinical meaningfulness of these cross shift changes in FEV1 is uncertain because lung function data were not reported during a non-paint exposure period at work. In the second study, 175 Swedish housepainters with various levels of previous experience and most of whom worked with both WB (average time spent painting: 19 h per week) and SB paints (average 4 h per week) were followed for up to 3 years (1989–1992). The incidence of seven general airway symptoms, and three work-related symptoms, was analysed by means of a self-administered questionnaire at the beginning and end of the study. No information was provided regarding whether symptom questionnaires were completed during periods of high work exposure, low work exposure or vacations. Different airway symptoms were compared with clinical information obtained by a methacholine challenge test and dynamic

spirometry in a selected subgroup of 44 painters. A total of 18% reported a history of atopy, 5% asthma as an adult and 9% hay fever at the beginning of follow-up [12]. There was a significant increase in chest tightness or wheezing from cold air (6.4%), chest tightness or wheezing after catching a cold (9.9%), airway irritation on exposure to nonspecific irritants (17.4%), and an airway symptom index (25.6%) in house painters related to the estimated total exposure to VOCs, the main source being the limited use of SB paints. All these respiratory symptoms (except chest tightness when wheezing with a cold) were significantly related to lung function (FEV1). In contrast there was no increase in symptoms among those painters who worked only with WB products.

Epidemiological studies

In total we found 19 original epidemiological studies using our selection criteria. Two were excluded because they considered only household conditions (peeling paint/plaster/wallpaper) [13,14]; one because the focus was on a Chemical Odor Intolerance Index including paint and respiratory problems [15]; and six because their focus was on inflammatory markers [16], pneumonia [17], acute dermatitis [18], eczema and unspecified allergic symptoms [16], or sick building syndrome symptoms among which unspecific airways symptoms [19,20].

During the preparation of the manuscript for publication, a further three studies were identified. The 13 studies are summarized in Table 3 (children/adolescent: $n = 11$; adults: $n = 2$). Population size ranged between 153 [21] and 13 988 [22].

The study methods included birth cohort ($n = 4$) and case-control ($n = 2$) or cross-sectional ($n = 7$) designs; most were of European populations (three Swedish, two German, one Russian, one UK), two took place in Australia, three in China and one in Japan.

Exposure assessment was classified as “good” in only two studies, one in children [23] and one in adults [24], “sufficient” in five studies in children [25–29] and one in young female adults [21] and “weak” in the remaining five studies in children [22,30–33].

Two studies measured VOCs directly, showing higher level of total VOCs in the dwellings in which the wall was recently painted [23,24]. Significantly increased concentrations of aliphatic compounds (C8–C11), 2,2,4-trimethyl 1,3-pentanediol diisobutyrate (TXIB), and butanols were also observed in newly painted dwellings. Information on type of paint used was obtained in only two studies [24,25] neither of which showed any evidence of effect modification by paint type.

All studies, except three [27–29], reported at least one significant association, whereas none showed an association with lung function measures (evaluated in two studies [22,24]). None of these studies assessed the short-term effect of painting on respiratory symptoms in asthmatics or painting exposure as trigger of asthma symptoms, but rather assessed the association between the prevalence/incidence of asthma or asthma-related symptoms and painting exposure.

Table 2. Occupational studies (non-spray painters).

Reference	Population	N (response rate)	Study design	Important findings	Limitations
Wieslander 1994 [3]	Swedish, male house painters	18 (78%)	Cross-shift (WB paint only)	Small but significant falls in mean FEV ₁ and FVC	No referent exposure
		44 (77%)	Cross-sectional	No significant difference in use of WB or SB paint in those with and without NSBHR Airway symptoms at work in employees with/without BHR: WBP: 10% (BHR) versus 0% (no BHR) SBP: 13% (BHR) versus 0% (no BHR) Sanding dust: 32% (BHR) versus 23% (no BHR) Incidence of: asthma-like symptoms: 26% self-reported asthma: 1.2% airway irritation from WBP: 27% airway irritation from SBP: 26% exposure-response for total paint-VOC exposures no increase in symptoms in those working only with WBP OR for “other painters” (n = 65) 2.34 (1.04–5.28) among participants with BHR and asthma symptoms or medication Asthma: Indoor painting (new): OR 1.17 (0.42–3.27) Indoor painting (renovation): OR 1.14 (0.49–2.68) Outdoor painting: OR 4.68 (1.36–16.11) Cough/wheeze Indoor painting (new): OR 1.39 (0.77–2.52) Indoor painting (renovation): OR 1.31 (0.79–2.15) Outdoor painting: OR 2.88 (1.21–6.86)	
Wieslander 1997 [12]	Swedish male house painters	175 (69% follow up)	Prospective Cohort (3 years)		not an “inception cohort” no adjustment for other exposures (including smoking)
Kogevinas 1999 [10]	General population, 20–44 (ECRHS)	9476	Cross-sectional		Potential confounding by greater use of SB paints and other tasks (e.g. sanding) in outdoor work
Kaukiainen 2008 [11]	Finnish, male con- struction workers	1000 painters 1000 carpenters (c.65%)	Cross-sectional		

Table 3. Epidemiological studies (ordered by study design, exposure assessment and year of publica).

Reference	Population/setting	N (response rate)	Study design	Adjustment	Exposure (class)	Health outcome	Important findings	
							Asthma-related symptoms	Lung function
Trevillian 2005 [27]	Children assessed at age 7 years, Tasmania (Australia)	863 (93%)	Birth cohort	Number of siblings, <i>in utero</i> exposure to maternal smoking, air freshener use in infant's bedroom, cat or dog as a family pet during infancy, visible indoor mould, use of a plastic mattress cover in infancy, low maternal education at infant's birth, and paternal unemployment at infant's birth.	Questionnaire on infant bedroom painted in the last 12 months and bedding composite index (on exposure to synthetic items and sheepskin) (2)	Questionnaire on: - wheezing last 12 months - ever asthma	Recent painting in the bedroom did not significantly predict wheezing (adjusted OR 0.94 (0.40–2.21)). A greater increase in risk of subsequent wheezing according to type of bedding used was observed in the case of infants sleeping in recently painted bedrooms	na
Henderson 2008 [22]	ALSPAC children followed to age 8 years, UK	13 988	birth cohort	overcrowding in home, highest maternal education level, housing tenure, sex, exposure to environmental tobacco smoke, maternal history of asthma, maternal parity, maternal age at delivery, smoking during pregnancy, month of completion of chemicals questionnaire and maternal hours worked outside home	Questionnaire on chemical exposure (including painting) during pregnancy. Score for total chemical exposure calculated (CHCE) (1)	Questionnaire on: - Wheezing in several study periods (0–6, 6–18, 18–30, 30–42, 69–81 month) lung function - SPT for effect modification	Increased CHCE score (based on 1 z-score increase) was associated with wheezing in non-atopic children: - early- (<18 months): OR 1.41 (1.13–1.76) - intermediate- (18–30 months): OR 1.41 (1.13–1.76) - persistent: OR 1.47 (1.02–2.13) - late-onset (>=30 months): OR 1.69 (1.19–2.41) - not in atopic children	Increasing CHCE score was associated with decrements in FEV1 and FEF25–75%. Effect very small. Interaction test not sign
Diez 2003 [32]	vulnerable children (high IgE level cord blood, atopic parents, low birth-weight), fu 2 years, Germany	186	birth cohort	cord-blood-IgE 0.9 kU/L, birth weight 2500 g, male sex, double positive parental atopy anamnesis, smoking, keeping a pet and dampness	Questionnaire, including recent redecoration during 1st year of life (1)	Questionnaire on: - obstructive bronchitis - wheezing at age 1 and 2 year	Adjusted risks for redecoration during 1st year: - obstructive bronchitis OR = 4.1 (1.4–11.9) - wheezing OR = 2.4 (0.7–8.6)	na
Diez 2000 [31]	vulnerable (high IgE level cord blood, atopic parents, low birth-weight) new born children, fu 1 year, Germany	475	birth cohort	heating, gas cooking, size of the flat, new furniture and domestic animals	Questionnaire on restoration including painting at 6 weeks and 1 year. VOC measured (25 compounds) (1)	Questionnaire on: - wheezing (at 1 year) - pulmonary infections (at 6 weeks)	Response to “during pregnancy painted dwelling” was positive in 105 cases (39%) and showed correlation with concentrations of aliphatic compounds. Restoration factor for: - wheezing: OR 1.9 (1.1–3.5) - pulmonary infections (painting) OR 5.6 (1.3–24.0).	na
Rumchev 2004 [23]	Cases (admitted hospital asthma), 104 controls children up to 3 years, Australia	88 cases; 104 controls (90% cases; 70% controls)	Case-control	age, sex, atopy, socioeconomic status, smoking indoors, presence of air conditioning and house dust mites	VOC measured (2 weeks winter, 2 weeks summer), questionnaire on painting exposure (unspecified painting) (3)	Hospital admissions primary diagnosis asthma	Median concentration of total VOCs 54.9 mg/m ³ (max 622 mg/m ³) in house recently painted. Recent painting in house was reported for significantly more cases than for children without asthma (37 (22%) v 28 (14%), <i>p</i> < 0.05). Total VOC levels significantly	na

(continued)

Table 3. Continued

Reference	Population/setting	N (response rate)	Study design	Adjustment	Exposure (class)	Health outcome	Important findings	
							Asthma-related symptoms	Lung function
Emenius 2004 [25]	children aged 2 years of whom 181 cases (recurrent episodes of wheezing), nested in a birth cohort of 4089 children.	540 (75%)	nested case-control	gender, heredity of allergic diseases, maternal smoking, duration of any breastfeeding and building age	questionnaire, bedroom painted during 1st year of life, home inspection (2)	recurrent wheezing at age 1–2 (3 or more reported episodes of wheeze + use of inhaled steroids) to identify cases	($p = 0.045$) affected by recent painting Newly painted surfaces in the child's bedroom associated with an increased of recurrent wheezing: OR 1.7 (1.3–2.6).	na
Wieslander 1997 [24]	Sweden ECRHS, adults (20–44 years old). Sweden	562 (87%)	cross-sectional	age, gender, and current smoking	questionnaire (indoor paint last 12 months), VOC exp in a sample of 62 dwellings (3)	Questionnaire on: • shortness of breath last 12 months • Wheezing last 12 months • Current asthma (BHR, at least one symptom) SPT FEV ₁	Indoor painting associated with: • nocturnal breathlessness (OR 1.57 (1.05–2.36)) • daytime breathlessness (OR 1.16 (0.75–1.79)) • wheezing (OR 1.21 (0.83–1.76)) • current asthma (OR 1.56 (0.98–2.48)) Indoor concentration of total VOC, aliphatic compounds (C8–C11), butanols, and 2,2,4-trimethyl 1,3-pentanediol diisobutyrate (TXIB) but not formaldehyde were significantly elevated in newly painted dwellings. The risks for new painting were: • wheezing: OR 2.64 (0.52–13.5) • nocturnal breathlessness: OR 0.63 (0.03–11.9) • airway infection: OR 2.97 (0.59–15.0) • pollen allergy: OR 5.52 (0.58–52.3) • furry pet allergy: OR 6.59 (1.12–38.7) No results reported for asthma	No relation between indoor painting and atopy, serum ECP, serum IgE, PEF variability, or lung function
Takaoka 2011 [21]	female university students (mean age 21 years). Japan	153 (95.6%)	cross-sectional	Age, current smoking habit, parental asthma/allergy and other indoor exposure	Questionnaire on indoor painting during the last 12 months (2)	ECRHS questionnaire on: • asthma • physician diagnosed asthma • current asthma medication • asthma attack during last 12 months • wheezing or whistling during the last 12 months • shortness of breath during the last 12 months • airway infection • pollen allergy	ECRHS questionnaire on: • asthma • physician diagnosed asthma • current asthma medication • asthma attack during last 12 months • wheezing or whistling during the last 12 months • shortness of breath during the last 12 months • airway infection • pollen allergy	na
Zhao 2008 [29]	pupils aged 11–15 years. China	1,993 (90.2%)	cross-sectional	age, sex, parental asthma or allergy, home environmental factors, indoor (classroom) and outdoor pollutants (SO ₂ , NO ₂ , O ₃)	questionnaire, including recent home painting (last 12 months) (2)	ISAAC and ECRHS questionnaire on: • Cumulative asthma • Doctor-diagnosed asthma • Allergies • Respiratory symptoms (wheezing, whistling in the chest, breathlessness, infections) during last 12 months	The risks for new indoor painting were: • Cumulative asthma: OR 2.10 (0.54–8.18) • Wheezing: OR 0.99 (0.52–1.87) • Daytime attacks of breathlessness: OR 1.43 (0.97–2.11) • Nocturnal attacks of	na

Norback, 2007 [28]	pupils aged 9–20 years. China	2116 (90%)	cross-sectional	age, gender, rural childhood, current urban residency, exposure to ETS and recent redecoration at home	Questionnaire on indoor painting at home in the last 12 months (2)	Questionnaire on prevalence of: • self-reported asthma • doctor-diagnosed asthma • eczema • allergy (pollen, cat)	breathlessness: OR 1.96 (0.28–3.34) • Pet or pollen allergy: OR 0.66 (0.26–1.71) • Respiratory infection: OR 1.23 (0.84–1.80) The risks for new indoor painting were: • ever had asthma: OR 1.57 (0.51–4.81) • doctor's diagnosed asthma: OR 1.61 (0.37–7.03) • eczema: OR 1.02 (0.42–2.49) • pollen or cat allergy: OR 1.38 (0.62–3.07) The risks for recent (past 12 months) painting were: • current wheezing: OR 1.25 (0.99–1.58) • asthma: OR 1.09 (0.53–2.22) • any allergy: OR 1.25 (1.05–1.47)	na
Jaakkola 2004 [26]	children 8–12 years old. Russia	5951 (96–98%)	cross-sectional	age, gender, preterm birth, low birth weight, parental atopy, maternal smoking during pregnancy, expos- ure to environmental tobacco smoke at home, and other's and father's education.	Questionnaire, including recent painting past 12 months or earlier (2)	Questionnaire on: • Current asthma • wheeze last 12 months • any doctor diagnosed allergy	Home decorations in recent 2 years a risk • Persistent cough: OR 1.87 (1.61–2.17) • Persistent phlegm: OR 2.31 (1.88–2.84) • Doctor diagnosed asthma: OR 1.72 (1.45–2.04) • current asthma: OR 1.43; 95% CI 1.06–1.93 • current wheeze: OR 1.59 (1.29–1.97) • allergic rhinitis: OR 1.17 (1.01–1.35) Adjusted risk for "Painting and changing wallpaper in the bedroom of the child after birth": • allergic rhinitis (OR 1.69 (0.92–1.43) • any allergic disease (OR 1.25 (1.03–1.52) • asthma (OR 1.07 (0.74–1.54)	na
Dong 2008 [33]	children aged 6–13 years. China	10,784 (87.5%)	cross-sectional	age, gender, breastfeeding, living in the city, school- ing, house type, area of residence, number of rooms, distance to traffic, pollution source nearby the house, indoor coal use, current exposure to ETS, exposure to ETS in the first 2 years and during pregnancy, pet keeping, parents' educa- tion and parents' atopy	questionnaire, including home redecoration in the past 2 years (1)	Questionnaire on: • respiratory symptoms during last 12 months • doctor-diagnosed asthma • Current asthma • Doctor-diagnosed allergic rhinitis	Questionnaire on: • ever asthma • wheezing • bronchitis • allergic rhinitis • eczema last year, ever	na
Aberg 1996 [30]	children aged 7 years. Sweden	1649	cross-sectional	frequency of upper respira- tory tract infection, par- ental symptom of allergic disease, moisture inside windows, damp or mold damage, kind of ventila- tion, year renovation walls in the bedroom, number of siblings, cat and dog at home	Questionnaire, including painting or changing wallpaper in the bedroom of the child after birth (unspecified period) (1)	Questionnaire on: • ever asthma • wheezing • bronchitis • allergic rhinitis • eczema last year, ever	Adjusted risk for "Painting and changing wallpaper in the bedroom of the child after birth": • allergic rhinitis (OR 1.69 (0.92–1.43) • any allergic disease (OR 1.25 (1.03–1.52) • asthma (OR 1.07 (0.74–1.54)	na

Result: studies in children

Additional data were requested from one study group [27]. Four studies with exposure to painting (classified as sufficient) reported an association with wheezing in the last 12 months [25–27,29] (Table 3). Among these studies only Emenius et al. [25] reported a significant association (OR 1.7 95% CI 1.3–2.6). An association of exposure with “current asthma” was reported in only one of the studies with sufficient exposure assessment (OR = 1.09 95% CI 0.53–2.22 [26]), whereas a non-significant association with cumulative asthma was reported in two studies (OR 2.1 95% CI 0.5–8.2 [29]; OR 1.6 95% CI 0.5–4.8 [28]). One further study in pre-school children [23] reported an unadjusted analysis showing that painting exposure (in the last 12 months) was more common among children admitted with asthma than controls (22% vs. 14%, $p < 0.05$). However, this was not explored further since the main focus of the report was the relationship of VOCs (from all sources) with asthma.

Four papers, all of which studied children reported an association between wheezing and redecoration and renovation or total indoor chemicals exposure, in which the questions used to assess exposure explicitly included painting (i.e. we considered the exposure assessment as “weak”) (OR 1.6 95% CI 1.3–2.0 [33]; OR 1.5 95% CI 1.0–2.1 [22]; OR 2.4 95% CI 0.7–8.6 [32]; OR 1.9 95% CI 1.1–3.5 [31]; Table 3). An association of exposure with asthma was reported only in two studies (OR 1.4 95% CI 1.1–1.9 [33]; OR 1.1 95% CI 0.7–1.5 [30]).

Results: studies in adults

There was one study in adults with “good” exposure measures [24]. Thirty-two percent of participants in the Swedish arm of the European Community Respiratory Health Survey, reported their home had been painted during the past year and this was associated with an increased prevalence of nocturnal breathlessness (OR = 1.6, CI 1.1–2.4) and (borderline) current asthma (OR = 1.6, CI 1.0–2.5), there was no increase in wheezing and daytime breathlessness. Blood eosinophil counts were significantly higher in those living in newly painted homes, and significantly related to a semi-quantitative “indoor painting index”. No association of painting with atopy, eosinophil cationic protein, IgE, peak flow variability or lung function was observed.

The second study conducted on young female adults reported a significant association between painting and pet allergy (OR = 6.6; CI 1.1–38.7), but no significant associations were detected for wheezing or nocturnal breathlessness [21].

Discussion

Even though we identified 20 original studies that examined the potential health effects of domestic paint exposure on respiratory disease the variable quality of the exposure assignment and heterogeneous study design makes it difficult to draw firm conclusions on whether such exposures cause or exacerbate asthma in children and/or adults. Very few population-based studies have examined the effects of painting rather than of redecoration/renovation, and only two

studies directly measured indoor VOCs during a period of renovation/redecoration that included painting. Of this latter group only one [23] related these measurements to asthma development or exacerbations. Causal relationships of asthma with paint exposure require longitudinal studies designed to answer this question – but all population observational studies are likely to run into difficulties of confounding by other redecoration activities.

While the immediate or near-immediate provocation of asthma symptoms by paint fumes in the home is a topic for discussion in informal patient forums, published research on the matter is surprisingly scanty. Most of the relevant published material is concerned with the longer term association of symptoms with painting/redecorating – any observed effects may be related to effects of other exposures occurring at the time the painting is done (i.e. other redecoration activities such as installation of linoleum floor, particleboard, new furniture, wall coverings or suspended ceilings), or from prolonged emissions from the painted surface following painting, or both. Furthermore, redecoration/renovation may occur at times of other major life events and changes in lifestyle – such as moving home, changing occupation or the birth of children. The occupational literature is tantalising in suggesting that work as a (non-spray) painter increases the risk of asthma but it provides no detail that can be applied to the domestic setting [3]. All the occupational studies, except one [12], used a cross-sectional study design and they may have missed workers who left the job because of sensitivity to paints or solvents. Thirteen relevant epidemiological studies of associations between respiratory effects (with a focus on asthma) in adults or children and indoor residential exposure to paint with or without indoor VOC measurements were identified. All but three [27–29] reported associations with some asthma-like symptoms but none found evidence using objective measures of lung function and only five studies provide evidence specifically in relation to painting in the home at some point in the past. In children at least there was evidence that painting was associated with wheezing, but the study designs and the exposure assessment were unsuitable to differentiate between acute and longer terms affects. We found the epidemiological evidence to be weak and certainly insufficient to infer causality.

There are numerous sources of VOCs in the home (pressed wood products, furniture fabrics, cleaning, disinfecting, cosmetic, and hobby products, adhesives, office equipment such as printers, correction fluids and permanent markers, and heating and cooking fuels) and the literature on their health effects is large [6]. A large number of epidemiological studies have explored the relationship on the effects of indoor VOC many demonstrating an association with asthma or related symptoms in children [34]. Domestic paints produce fumes – many of them VOCs – and their emissions may persist long after any odour has gone. European manufacturers are bound by law to produce paint types that contain far lower concentrations of VOC than previously and in many countries low-VOC paints have become the mandated norm. While measures to improve indoor air quality are to be welcomed, we found little evidence that the level of VOC in the paint was associated with symptoms. Beach et al. [7] addressed this

directly (by examining acute effects in a small group of volunteers with asthma exposed to conventional and to VOC-free paint) and although the “conventional” paint was associated with more symptoms the differences were small and unaccompanied by any objective changes in lung function. Of the 13 relevant epidemiological studies none includes comparison of low-VOC paints with other type. Two of the studies confirm the expected association of increased specific and total VOCs in homes that have recently been painted, but even using proxy measures of increased VOC exposure (e.g. “recently painted home”) the relationships are not clear [23,24].

Propylene glycol and glycol ethers (PGEs) are considered to be good solvents, with relatively low volatility and are used in WB paints, varnishes, cleaning fluids and other household products. Compared to SB paint, the proportion of glycol ethers and propylene glycol in the total VOC emissions from WB paint is higher. The health effects of PGEs have received some attention following the recent publication of a study that investigated levels of eight classes of VOCs in young children and suggested exposure may be related to allergic disease, including asthma [35]. One hundred ninety-eight cases (with at least two of wheeze, eczema or hayfever) and 202 controls (symptom free) were identified using a respiratory questionnaire for further investigation including home sampling and inspections; air samples were taken in the bedroom in which the child slept most of the time and measured for VOCs. Of the eight classes of VOCs detected, PGEs were significantly higher in case homes than in control homes, the differences being seen for all conditions (asthma, rhinitis, eczema). This study requires replication in another population or setting and to be conducted in such a manner that the exposure is measured prior to the development of symptoms. Longitudinal cohort studies of highly exposed versus unexposed workers would best determine any relationships between exposure and disease.

We have focussed on respiratory symptoms as the outcome of interest – but we are aware of one study that examined nasal biomarkers and WB paint exposure [36] which suggested that markers of nasal inflammation (myeloperoxidase, eosinophilic cationic protein) may be related to paint emissions. The authors also found associations between biomarkers and measured exposure to specific compounds, including propylene glycol, 2-phenoxyethanol, sum of aliphatic glycol ethers, and one MVOC (1-octen-3-ol). These findings could suggest that lower airway inflammation may also occur with such exposure.

Painting is often just one component of major redecoration and renovation in homes, and is often accompanied by increases in exposure to other agents (e.g. wood and plaster dust, paint strippers, carpet fixative). In most studies it is difficult, if not impossible, to separate the health effect of each exposure. We cannot rule out that any positive associations arise from residual confounding by other decorating/renovating activities or the provision of new furnishings. Similarly, those who work as “domestic” painters may be exposed to a range of other agents associated with home redecoration that might exacerbate asthma. Evidence that implicates paint, and paint alone, however, does come from the paint challenge studies of Beach et al. [7], which provides

limited evidence that paint exposure may contribute to the burden of work aggravated asthma.

Conclusion

From this review, we conclude there is inadequate evidence to determine if home or work-related exposure to domestic paints either causes asthma or aggravates pre-existing asthma in exposed individuals.

With such uncertainty we broadly agree with the conclusion of a previous systematic review which states: “we can still conclude that there is a need for extensive ventilation for several months after renovation and redecoration. This conclusion is more or less derived as a precautionary principle” [1]. With all these uncertainties, the public health message remains that people should use all paints in well-ventilated rooms. This is easier to achieve in some homes and in some climates than in others, and measures to reduce exposures may be warranted (e.g. the avoidance of painting on surfaces that absorb the paint and then act as a source of low grade emissions over the following months). Low emission paints have been developed, and are widely promoted in some parts of Europe, but hard evidence for their health value is not available. The necessary studies are difficult to perform, requiring substantial resource for identification of participants who are going to redecorate their homes, measurement of painting activity (and of other redecoration activities) and measurement of acute and long-term symptoms. Notwithstanding, further research with more rigorous exposure measures and different design is needed to address popular concerns over the acute effects of domestic paint exposure, and to strengthen the evidence base for assessment of acute or long-term effects of domestic paint. Such studies will help the development of sound public health policy and advice to improve indoor air quality.

Declaration of interest

The authors declare no conflicts of interest. The authors alone are responsible for the content and writing of this article. The study was supported by a grant from Asthma UK.

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