



Non-IgE-mediated and irritant-induced work-related rhinitis

Andrea Siracusa^a, Ilenia Folletti^a, and Gianna Moscato^b

Purpose of review

Recently there has been growing interest in non-IgE-mediated and irritant-induced occupational rhinitis due to old and new low-molecular-weight and irritant agents. The purpose of this review is to summarize the scientific evidence on agents and work activities responsible for non-IgE-mediated and irritant-induced occupational rhinitis and work-exacerbated rhinitis published in 2011 and 2012.

Recent findings

Several epidemiological, surveillance and experimental studies, case reports and reviews showed that workers exposed to drugs, wood dust, chemicals, metals and biocides are at high risk of non-IgE-mediated and irritant-induced occupational rhinitis; among activities at risk are healthcare, antibiotic manufacturing and cleaning workers. Work-exacerbated rhinitis has not been specifically studied, but it is reasonable to expect that it is frequently associated with work-exacerbated asthma. Recently, work-related anosmia/microsmia, nasal polyps and sinusitis have also been described. Reducing or eliminating workplace exposure to the specific agent has been confirmed to be effective in preventing symptoms of nonallergic occupational rhinitis.

Summary

In consideration of the relevance of non-IgE-mediated and irritant-induced work-related rhinitis, physicians should recognize work-related rhinitis symptoms due to old and new low-molecular-weight and irritant agents. The mechanisms of non-IgE-mediated and irritant-induced occupational rhinitis remain largely unclear and need to be studied further. Substitution of responsible agents, reduction or elimination of exposure at the workplace should be enforced as effective measures.

Keywords

case reports, epidemiology, irritants, non-IgE-mediated, occupational rhinitis

INTRODUCTION

Work-related rhinitis (WRR) is two to four times more frequent than occupational asthma [1] and includes occupational rhinitis (Ocr) and work-exacerbated rhinitis [2]. Ocr has recently been classified as allergic or nonallergic [2]. Allergic Ocr is caused by immunologically mediated hypersensitivity reactions characterized by a latency period, which may be IgE-mediated or not. If IgE-mediated, it is usually due to high-molecular-weight and some low-molecular-weight (LMW) agents. If non-IgE-mediated, it is due to LMW chemicals for which the allergic mechanism has not yet been characterized [2]. Nonallergic, irritant-induced Ocr, a type of Ocr without a latency period, is caused by an irritant, nonimmunological mechanism. A single exposure to high levels of irritants in the workplace can give rise to reactive upper airway dysfunction syndrome (RUDS), an infrequent nonallergic Ocr,

whereas multiple exposures to irritants may cause the most common form of irritant-induced Ocr [2]. In the past decade studies showed a high prevalence of irritant-induced Ocr in working populations exposed to one or more irritant agents [3,4] and 'new' irritants or LMW agents, in which an immunologic mechanism was not demonstrated, responsible for Ocr have been described [5^a,6^a,7^a]. Few data are available for work-exacerbated rhinitis [8].

^aOccupational Allergology Unit, Department of Clinical and Experimental Medicine, University of Perugia, Perugia and ^bAllergy and Immunology Unit, Fondazione Salvatore Maugeri, Institute of Care and Research, Scientific Institute of Pavia, Pavia, Italy

Correspondence to Andrea Siracusa, MD, Borgo 20 giugno 87, 06121 Perugia, Italy. Tel: +39 075 32781, 39 0339 725 4969; fax: +39 0744 205 762; e-mail: asiracusa70@gmail.com

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KEY POINTS

- Recent studies have expanded knowledge on new irritant and low-molecular-weight agents and work activities causing non-IgE-mediated and irritant-induced occupational rhinitis and other work-related nasal symptoms.
- Non-IgE-mediated and irritant-induced work-related rhinitis is frequently reported by healthcare, antibiotic manufacturing and cleaning workers.
- Main new agents include drugs, wood dust, chemicals, metals and biocides.
- Reduction or avoidance of exposure should be enforced as effective measures.

We have used PubMed and OvidSP with the search terms (work-related or occupation* or 'occupational exposure') and (rhinitis or 'nasal irritation*' or 'nasal symptom*'). We found 42 papers, including epidemiological, surveillance and experimental studies, case reports and reviews.

The purpose of this review is to summarize and discuss the scientific evidence on new agents and work activities responsible for non-IgE-mediated and irritant-induced OcR, and work-exacerbated rhinitis that have been published between 2011 and 2012.

REVIEWS

Eight reviews reporting data and/or discussion on OcR were published between 2011 and 2012. Sublett and Bernstein [9] summarized the knowledge on OcR, mentioning possible mechanisms of non-allergic WRR. Shusterman [10] reported exposure to chlorine gas in pulp mill workers, chronic exposure to hexavalent chromium from welding fumes or metal plating operations and exposures to the World Trade Center (WTC) disaster (11 September 2001) as possible causes of RUDS/irritant WRR. Higgins and Reh [11] discussed the role of occupational irritants in inducing WRR.

A comprehensive review of new deleterious agents causing occupational asthma reported several cases of OcR due to LMW agents where an immunologic mechanism was not demonstrated [7[■]]. Among such agents were several drugs (colistin, vancomycin, lasamide and precursors, sevoflurane and isoflurane, aescin, 7-amino-3-thiomethyl-3-cephalosporanic acid and mitoxantrone), wood dust (Chengal, Tali and Jatoba) and a disinfectant (peracetic acid–hydrogen peroxide mixture). The authors discussed the possible onset of asthma in workers with repeated low-dose exposure to one or

more respiratory irritants, such as pulp and paper mills exposed to chlorine, meat wrappers, workers exposed to formaldehyde, aluminum smelters exposed to gaseous fluorides, hydrofluoric acid and sulfur dioxide, workers exposed to machining fluids, sulfite millers, workers exposed to the WTC collapse or cleaning products [7[■]]. Similarly, we can expect the possible onset of low-dose irritant-induced OcR after repeated exposure to the above agents/activities, although no data are available.

Pralong *et al.* [6[■]] reviewed new LMW agents causing occupational asthma/OcR with a latency period reported between 2000 and 2010, and found 39 publications describing 41 new LMW agents such as drugs, wood dust, chemicals, metals and biocides, causing 62 cases of occupational asthma and 33 OcR. Despite the presence of a latency period, suggesting an underlying immunologic mechanism, specific skin prick tests (SPTs) or serum IgE were positive in 24% of cases of occupational asthma and in 27% of OcR, and an IgE-mediated mechanism was shown in only 20% of the newly described agents.

Three studies reviewed specific work agents/activities. In antibiotic manufacturing workers 17 case reports describing 21 cases of OcR were reported [12[■]]. Specific SPT or IgE was positive in 43% of cases, negative in 38% and not reported in 19%. Negative SPT or IgE was found in occupational asthma/OcR due to ampicillin (four patients), amoxicillin, cefmetazole, cefadroxil, vancomycin and colomycin (one patient each).

The effect of methyl methacrylate as possible respiratory sensitizer or irritant was reviewed [13[■]]. Epidemiologic studies in exposed workers reported irritation of eyes, nose and upper respiratory tract associated with short-term peak exposure. The prevalence of OcR ranged from 1 to 10%. The authors argued that methyl methacrylate is a respiratory irritant and not a sensitizer.

An invited commentary reviewed the consequences of respiratory exposures in the farm environment [14]. Farmers often report smell impairment, nasal polyps and hyperemia of the nasal mucosa, indicating the effect of irritants in their workplace. In contrast, other studies show that growing up on a farm seems to have a protective effect on upper and lower airways, leading to a lower prevalence of allergic rhinitis and asthma.

Summarizing, there is a growing interest in non-IgE-mediated and irritant-induced occupational asthma and OcR due to new LMW and irritant agents.

EPIDEMIOLOGY AND RISK FACTORS

Cross-sectional studies in working groups published in the past 2 years estimated the prevalence of nasal

symptoms, such as stuffy, runny or blocked nose, sneezing, rhinorrhea, bleeding and nasal congestion in 3–74% of workers in different settings and exposed to LMW and irritant agents (Table 1) [15[■],16,17,18[■],19,20,21[■],22[■],23–26].

A nonsignificant higher prevalence of physician diagnosis of rhinitis was found in USA greenhouse workers exposed to fungi, bacteria, endotoxin and β -D-glucan than in office workers [15[■]]. Three rhinitis symptoms (frequent sneezing, stuffy and runny nose) were more frequent in employees of a water-damaged school exposed to mold contamination and *Cladosporium* than in controls [16]. A high risk of nasal congestion and runny nose was associated with exposure to second-hand smoke in flight attendants, restaurant, shopping mall and hotel employees [17,18[■]]. A high prevalence of work-related nasal symptoms was found in nail technicians exposed to acrylates and in brass foundry workers exposed to methyl isocyanate, isocyanic acid and formaldehyde [19,20]. In workers employed in on-board fish processing, filleting of fish was associated with a high risk of work-related respiratory symptoms such as running nose, frequent sneezing and cough with phlegm [21[■]]. In dental healthcare workers work-related nasal and ocular symptoms were strong predictors of work-exacerbated asthma (WEA) [22[■]]. Other occupations with a high prevalence of work-related nasal and ocular symptoms were early manufacturing process of tea (nonsignificant difference from office workers) [23] and manufacturing of hygiene products; in the latter the prevalence of nasal crusts and nose bleeding was higher in workers exposed to paper dust than nonexposed individuals [24]. A nonsignificant increased risk of rhinitis symptoms was found in South Korean farmers applying paraquat (prevalence 2.8%) compared with non-paraquat-applying farmers (prevalence 1.4%) [25]. The prevalence of runny nose was 32% in Iranian lifeguards and 10% in matched controls and nasal symptoms exacerbated after exposure to chlorine at work [26].

In a cross-sectional population-based study [27] a high risk of chronic rhinitis with nasal congestion and runny nose was found in workers with occupational exposure to gas, fumes and dust.

Two longitudinal studies [28,29] in working groups demonstrated a risk of rhinitis symptoms in sugar cane refinery workers (exposed to dust/bagasse) and wood workers (exposed to airborne fungi/wood dust). In the former, prevalence of rhinitis increased from 17 to 40% during harvesting season and did not change in control individuals [28]. Finally, a longitudinal population-based study [30] showed a high prevalence of sino-nasal

symptoms in migrant day laborers, probably due to dust and irritant exposure.

Level of exposure was confirmed as the main risk factor of non-IgE-mediated and irritant-induced WRR and was estimated by a questionnaire (Table 1). A dose–response relationship between nasal symptoms and exposure to isocyanic acid, methyl isocyanate, formaldehyde and total dust was shown [20].

Some studies [17,18[■],19,25,26] showed a non-IgE-mediated and/or irritant-induced mechanism associated with one agent, such as chemicals or second-hand smoke (Table 1). However, in most cases, exposure to multiple irritant and sensitizing agents was shown, making mechanisms more difficult to interpret (Table 1) [15[■],16,20,21[■],22[■],23,24,27–30]. In two studies [22[■],23], an IgE-mediated mechanism was demonstrated. Sensitization to latex (7%) or tea (1%) was much lower than the prevalence of work-related nose symptoms, probably due to the low sensitivity of allergologic tests (as was previously shown for latex) and/or to the prevalent exposure to LMW/irritant/ill-defined agents.

CASE REPORTS

In the past 2 years, 7 papers including 18 case reports on agents causing non-IgE-mediated and irritant-induced OcR have been published (Table 2) [12[■],31,32[■],33–35,36[■]]. An IgE mechanism was never demonstrated, although in some cases an immunological mechanism cannot be excluded [12[■],31,32[■],36[■]]. Six cases of work-related asthma and three WRR were reported in healthcare workers exposed to detergent enzymes for cleaning medical instruments [31]. Specific IgE to biological enzymes (Maxatase, Savinase, α -Amylase and Alcalase) were negative in all cases. Enzymes, apart from sensitization, may cause irritative symptoms. Removal from exposure improved or eliminated respiratory symptoms.

Ten cases of OcR, eight of which were associated with occupational asthma, due to ethylenediamine tetraacetic acid (EDTA)-containing detergents or disinfectants, were reported in healthcare or cleaning workers who used spray formulations of cleaning products [32[■]]. The diagnosis was based on WRR symptoms and positive nasal provocation tests.

A bricklaying lecturer developed after 8 years of low-dose exposure to irritants work-related runny eyes and nose, and 4 years later symptoms of occupational asthma [33]. Working outdoors completely prevented work-related symptoms.

A 52-year-old woman developed work-related asthma and rhinitis 2 years after starting packaging

Table 1. Epidemiology of non-IgE-mediated and irritant-induced occupational rhinitis in cross-sectional studies in working groups (2011–2012)

Reference/ country	Patients (n): exposed/ controls	Participation rate (%)	Mean age (years)	Agents	Duration of exposure	Prevalence in exposed workers (%)	Prevalence in nonexposed workers (%)	Exposed vs. nonexposed workers: OR (95% CI) unless otherwise stated	Symptoms	Risk factors/ mechanism
Adhikari <i>et al.</i> , 2011/USA [15 ^a]	35 greenhouse workers/ 14 office employees	NR	40	Fungi, bacteria, endotoxins and (1 → 3)-β-D-glucan	NR	21	7	$P = 0.40$	NR	None/IgE-mediated?, non-IgE-mediated?, irritant-induced?
Thomas <i>et al.</i> , 2012/USA [16]	95 water damaged school employees/ 110 comparison school employees	80	46	Mold contamination, <i>Cladosporium</i>	4	Runny nose: 25 Stuffy nose: 29 Frequent sneezing: 20	Runny nose: 7 Stuffy nose: 10 Frequent sneezing: 4	Runny nose 3.9 (1.7–8.6) Stuffy nose 3.1 (1.6–6.1) Frequent sneezing 5.2 (1.8–15.0)	Runny and stuffy nose, frequent sneezing	Mold contamination/ IgE-mediated?, non-IgE-mediated?
Beatty <i>et al.</i> , 2011/USA [17]	362 flight attendants/ NHANES 2005–2006 participants	NR	58	Second-hand smoke	17	22	NR	2.1 (1.4–3.2)	Nasal congestion	Second-hand smoke/ irritant-induced
Zheng <i>et al.</i> , 2011/China [18 ^a]	2500 restaurant, shopping mall and hotel employees/ 206 kindergarten employees	90	27–33	Second-hand smoke	NR	Runny nose: restaurants 32, shopping malls 25, hotels 31	Kindergartens 15	Runny nose: restaurants 2.2 (1.7–3.3), shopping malls 1.8 (1.1–2.9), hotels 2.0 (1.3–3.1)	Runny nose	Workplace exposure to second-hand smoke/irritant-induced
Harris-Roberts <i>et al.</i> , 2011/UK [19]	71 nail technicians/ 64 office employees	100	30	Methyl methacrylate monomer and ethyl methacrylate	6.4	21	3	6.2 (1.3–30.7)	NR	Exposure to ethyl methacrylate/ non-IgE-mediated and/or irritant-induced
Löfstedt H <i>et al.</i> , 2011/Sweden [20]	43 foundry workers/ 69 nonexposed workers	90	44	ICA, MIC, formaldehyde and total dust	8	74	39	Nasal sx: OR 4.6 (2.0–10.7)	Nasal sx (and signs)	Dose-response relationship between nasal sx and exposure to ICA, MIC, formaldehyde and total dust/irritant-induced
Shiryaeva <i>et al.</i> , 2011/Norway [21 ^a]	122 trawler fishermen/ 118 merchant seafarers	99	46	Onboard fish processing and bioaerosols	Runny nose in trawler fishermen: 30, in merchant seafarers: 29	30	29	In filleting fish workers: runny nose 3.0 (1.2–7.7), frequent sneezing 3.4 (1.0–12.7)	Runny nose, sneezing	Filleting of fish/ IgE-mediated?, non-IgE-mediated?, irritant-induced?
Singh <i>et al.</i> , 2012/South Africa [22 ^a]	454 dental healthcare workers/ND	25	36	Latex, acrylates and disinfectants	10	WR ocular-nasal sx: 14	ND	ND	Runny, blocked and stuffy nose	WR ocular-nasal sx: predictor of WEA ($P < 0.001$)/ IgE-mediated, non-IgE- mediated and irritant- induced
Shieh <i>et al.</i> , 2012/Taiwan [23]	91 tea workers engaged in early manufacturing process/32 office employees	93	41	Inhalable and respirable dust, and coarse fraction	NR	29	28	Rhinorrhea/sneezing: $P < 0.05$	Nasal catarrh, rhinorrhea, sneezing	Ball-rolling workers had a higher prevalence of acute nasal sx/IgE- mediated, non-IgE- mediated and irritant- induced
Holm <i>et al.</i> , 2011/Sweden [24]	857 workers manufacturing hygiene products/186 nonexposed workers	52	45	Superabsorbent polymer and paper dust	NR	47	38	Exposure to paper dust: nasal crusts 1.4 (1.01–2.1); nose bleeding 1.7 (1.2–2.4)	Nasal blockage and crusts, nose bleeding	Exposure to paper dust/ irritant-induced
Cha <i>et al.</i> , 2012/ South Korea [25]	2508 farmers applying pesticides/374 farmers nonapplying pesticides	NR	NR	Paraquat	NR	2.8	1.4	2.0 (0.8–5.1)	NR	Exposure to paraquat/ irritant-induced
Boskabady <i>et al.</i> , 2012/Iran [26]	38 lifeguards/38 controls	100	31	Chlorine	13	32	10	Runny nose: 4.9 (1.1–13.6)	Sneezing and runny nose	Exposure to chlorine/ irritant induced

CI, confidence interval; ICA, isocyanic acid; MIC, methyl isocyanate; ND, not done; NR, not reported; OR, odds ratio; sx, symptoms; WEA, work-exacerbated asthma; WR, work-related.

Table 2. Agents causing non-IgE-mediated and irritant-induced occupational rhinitis: case reports (2011–2012)

Reference/country	Patients (n)	Occupation/activity	Agent	Age (years)	Diagnostic approach	Duration of exp. before work-related rhinitis (years)	Prevention	Other observations	Mechanism
Díaz Angulo <i>et al.</i> , 2011/Spain [12 [■]]	1	Packaging of erythromycin	Erythromycin ethylsuccinate	52	WRS	2	Avoidance of workplace exposure to granulated erythromycin	Specific IgE for erythromycin ethylsuccinate were negative	Non-IgE-mediated
Adisesh <i>et al.</i> , 2011/UK [31]	3	Healthcare workers	Detergent enzymes for cleaning medical instruments	46–56	WRS	3–6	Workplace exposure limits must not be exceeded	Specific IgE for Maxatase, α Amylase and Alcalase were negative	IgE-mediated? Irritant-induced?
Laborde-Castérot <i>et al.</i> , 2012/France [32 [■]]	10	Cleaners and healthcare workers	Ethylenediamine tetraacetic acid (EDTA)	46	WRS and specific nasal provocation tests	NR	/	/	Irritant? Pharmacological? Non-IgE-mediated?
Burge <i>et al.</i> , 2012/UK [33]	1	Bricklaying lecturer	Sand and lime (calcium carbonate) dust	49	WRS	8	Job change with outdoors work teaching slab laying	/	Irritant-induced
Burton <i>et al.</i> , 2011/UK [34]	1	Craft and design teacher	Medium-density fibreboard	54	WRS and specific inhalation challenge	10	Job change with no exp. to medium-density fibreboard	/	Irritant-induced
Nam <i>et al.</i> , 2012/Korea [35]	1	Pepper mill worker	Capsaicin	44	WRS and specific nasal provocation tests	4	Job change with no exp. to capsaicin	Skin prick tests to capsaicin were negative	Irritant-induced and/or pharmacological
Pala <i>et al.</i> , 2011/Italy [36 [■]]	1	Hairdresser	Ammonium persulfate	25	WRS and specific inhalation challenge	5	/	Skin prick tests to ammonium persulfate were negative	Non-IgE-mediated

WRS, work-related symptoms.

erythromycin [12[•]]. Serum IgE to erythromycin ethylsuccinate was not found. Avoiding further exposure to granulated erythromycin improved her work-related symptoms.

In three other case reports (a craft and design teacher worker exposed to medium-density fibre-board, a pepper mill worker exposed to capsaicin and a hairdresser exposed to ammonium persulfate) the diagnosis of Ocr was based on work-related symptoms and specific inhalation challenges [34,35,36[•]]. The design teacher worker also had occupational asthma and specific IgE to soft and hard wood were negative [34]. In the pepper mill worker SPT to capsaicin was negative [35]. In the hairdresser Ocr was associated with work-related cough and SPT to ammonium persulfate was negative [36[•]]. The latency of specific exposure before Ocr onset was between 4 [35] and 10 years [34]. Job change with no exposure to the specific agent improved symptoms of Ocr [34,35].

Summarizing, in the past 2 years, several relatively new agents caused Ocr after a latency of 2–10 years. In most cases reduction or avoidance of exposure was effective.

WORK-EXACERBATED RHINITIS

Work-exacerbated rhinitis has not been specifically investigated and few data are available on the association between WRR symptoms and WEA [8,22[•]]. WEA is associated with occupational exposure to sensitizers and nonsensitizing agents such as dusts, abnormal temperature or poor indoor air quality, physically strenuous work, chemicals and irritants, and occurs in more than 20% of adults with asthma [37]. In adults rhinitis is around four times more frequent than asthma and Ocr due to the fact that high-molecular-weight agents tend to be 1.2–3.3 times more prevalent than occupational asthma [38]. Moreover, there is some evidence that WRR symptoms are highly associated with WEA [8,22[•]]. At least one work-related nasal symptom was reported by 83% of patients with WEA [8] and work-related ocular–nasal symptoms were a strong predictor of WEA [odds ratio (OR) 6.7, 95% confidence interval (CI) 2.4–19.1] [22[•]]. In conclusion, we may speculate that work-exacerbated rhinitis is frequently associated with WEA. If so, then work-exacerbated rhinitis would affect a substantial proportion (~20%) of working adults with rhinitis. Unfortunately, data are missing.

OTHER WORK-RELATED NASAL INVOLVEMENT

Three studies dealt with the work-related nasal symptoms other than Ocr. Altman *et al.* [39] evaluated

olfaction in 99 WTC-exposed patients and in 99 matched normal individuals by the University of Pennsylvania Smell Identification Test. Exposure to WTC air pollution was associated with a decrease in the ability to identify odors ($P=0.003$). Three per cent and 15% of exposed workers had anosmia and severe microsmia, respectively.

In a case-control study [40] of 100 patients with nasal polyps admitted to hospital and 102 controls from the general population, 23% of patients with nasal polyps and 9% of controls reported an evident occupational exposure ($P=0.003$); 27% of patients and 16% of controls had a long occupational exposure ($P=0.001$).

Another case-control study [41] showed that patients with rhinosinusitis who needed surgery, especially repeated surgical interventions, were more likely to report a relevant occupational exposure and in 51% of cases irritants were involved.

EXPERIMENTAL STUDIES

In 2011–2012 few experimental studies evaluating the effect of inhalation of various irritants on upper and lower airways were published. The effect of inhaled 2,3-pentanedione was evaluated on rats [42]. Previously, a close structural relative of 2,3-pentanedione, diacetyl (2,3-butanedione), was used in microwave popcorn production for the flavor and aroma of butter and was associated in exposed workers with the risk of eye, nose or throat irritation, airway obstruction and severe bronchiolitis obliterans [43[•]]. The inhalation of 318 ppm for 6 h in rats caused an epithelial injury in the upper nose, such as the loss of olfactory neurons, apoptosis and necrosis.

Acute inhalation of allyl alcohol was evaluated in rats [44]. Mild, reversible nasal inflammation was found at the lowest exposure levels (50 ppm/1 h, 20 ppm/4 h or 10 ppm/8 h), whereas severe, irreversible nasal olfactory epithelial lesions were found at high exposure levels (50 ppm/8 h).

In the early to mid-1990s 1-bromopropane was used as an intermediate in the production of pesticides, quaternary ammonium compounds, flavors and fragrances, pharmaceuticals, and other chemicals. A recent USA National Toxicology Program Technical Report evaluated inhalation of 2-year exposure of 1-bromopropane in rats [45]. In the nose there was an increase of chronic inflammation, respiratory epithelial hyperplasia and metaplasia of the olfactory epithelium.

In 41 male volunteers exposure to formaldehyde concentration of 0.7 ppm for 4 h and of 0.4 ppm for 4 h with peaks of 0.8 ppm for 15 min did not cause adverse effects related to irritation, and no

differences between hyposensitive and hypersensitive individuals were observed [46].

MANAGEMENT AND PREVENTION

The management of OcR has two main objectives: preventing or reducing nasal symptoms and decreasing the risk of occupational asthma onset [2]. Improvement/disappearance of symptoms can be obtained by avoidance or at least reduction of exposure. Pharmacologic treatment is another option. The effectiveness of reducing or eliminating workplace exposure to the specific agent has recently been confirmed. In four case reports job change with no exposure to the responsible agent was effective in the improvement or resolution of WRR symptoms [12[■],33–35]. In one case, OcR symptoms improved after a job change associated with antihistamines and intranasal corticosteroids treatment [35].

A Chinese study [18[■]] evaluated the association between respiratory symptoms and second-hand smoke exposure among different working populations in Shanghai. Compared with employees from kindergartens, who had the lowest exposure to second-hand smoke, nonsmoking workers from restaurants, shopping malls and hotels had a higher prevalence of upper and lower respiratory symptoms.

Implementation of exposure controls was evaluated among workers exposed to a butter-flavoring chemical (diacetyl) at a microwave popcorn plant [43[■]]. Ventilation and isolation of the production process resulted in a three-fold reduction of diacetyl air concentrations. In workers with past high exposure nasal symptoms declined and new workers had fewer upper and lower respiratory symptoms than workers with past high exposure.

CONCLUSION

Recently, there has been a growing interest in non-IgE-mediated and irritant-induced occupational rhinitis in many work activities. Moreover, several old and new irritants and LMW agents, in which an immunologic mechanism was not demonstrated, have been described in epidemiological, surveillance and experimental studies, case reports and reviews. Among new agents are drugs, wood dust, chemicals, metals and biocides, and among activities healthcare, antibiotic manufacturing and cleaning workers were more frequently reported. In most cases reduction or avoidance of exposure was effective. A few studies evaluated other work-related nasal involvement, such as anosmia/microsmia, nasal polyps and sinusitis. The mechanism of

non-IgE-mediated and irritant-induced occupational rhinitis in most cases remains undetermined.

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Conflicts of interest

There are no conflicts of interest.

REFERENCES AND RECOMMENDED READING

Papers of particular interest, published within the annual period of review, have been highlighted as:

- of special interest
- of outstanding interest

Additional references related to this topic can also be found in the Current World Literature section in this issue (pp. 218–219).

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