

Part I

The personal environment

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Cosmetics and personal care products in lung diseases

Howard M. Kipen

UMDNJ–Robert Wood Johnson Medical School, Piscataway, NJ, USA

1.1 Introduction: historical context of cosmetics and respiratory illness

Cosmetics may be associated with respiratory illness through two different but overlapping mechanisms. One is via causation of pathological disease, most prominently related to allergen-mediated mucosal and airway responses. The second mechanism is through symptoms and illness behavior associated with odors from the cosmetics. The extent to which these symptoms may also interact with mucosal irritant properties of the agents makes differentiation between airway pathology and symptoms unrelated to airway pathology at times problematic. This chapter will describe the data supporting different disease mechanisms and appropriate clinical and preventive responses.

A wide range of individuals, rather than typically ‘healthy workers’, regularly come into contact with personal care products such as soaps, perfumes and hair products. Many of these products are designed to announce their presence to those nearby (perfume odors), and they encompass a diverse array of chemical substances. Odor-driven responses may be from the essential product, such as a perfume essence, or added material contained in a mix, such as fragrances added to a hairspray or after-shave. While behavioral effects of agents such as perfumes are intentional and legendary, the association of physical pulmonary conditions with cosmetic products was not reported until the late 1950s. Around 1960 a series of cases reporting a ‘storage disease’ (thesaurosis) or pneumonitis (‘hairspray lung’) were published. However a prevalent condition of the pulmonary parenchyma was never established (possibly due to various

changes in hairspray formulations) and all subsequent concern with respiratory effects of cosmetic and personal care agents has centered on the airways, particularly asthma. The first report of allergic occupational asthma in hairdressers is attributed to Jack Pepys [1] in 1976. The remainder of this chapter will consider both allergic airway disease from cosmetics and personal products and the more complex nonallergic responses to odors.

1.2 Epidemiological context

1.2.1 Occupational exposure to cosmetics and personal care products

Data from the USA reveal the substantial size of the workforce involved in cosmetology. According to the US Bureau of Labor Statistics, barbers, cosmetologists and other personal appearance workers held about 790,000 US jobs in 2004. Of these, barbers, hairdressers, hairstylists and cosmetologists held 670,000 jobs; manicurists and pedicurists 60,000; skin care specialists 30,000; and shampooers 27,000. Because most of the relevant scientific literature pertains specifically to hairdressers, this term will be used for the remainder of the chapter. There is no available data on the number of individuals involved in the perfume industry.

Although methods for ascertainment differ greatly between countries, the burden of airway disease in hairdressers has been quantitated in many different nations. Methodologies of varying rigor, including some that are population-based, have documented apparent excesses of asthma and respiratory symptoms relative to the general population among hairdressers working in Sweden, France, Germany, Belgium, Norway, Turkey and Italy.

A 2002 questionnaire study of all active Swedish hairdressers showed an asthma incidence rate ratio of 1.6 in never smokers, comparable to the effect of smoking alone in the same group. There was also a nonsignificant excess risk of asthma for self-reports of more frequent exposure to bleaching agents or hairsprays. Interestingly, there was no effect modification by reported atopy and no dose-response relationships for use of persulfates, at variance with much of the clinical data cited below that emphasize the role of persulfate exposure.

Iwatsubo and colleagues [2] found no increased respiratory symptoms among hairdressing apprentices compared with office apprentices, but there was a significant decline in FEV₁ and FEF₂₅₋₇₅ (forced expiratory flow), not linked to any specific hairdressing activities. Other studies from France are based on the voluntary national physician reporting program for occupational asthma (Observatoire National des Asthmes Professionnels). French asthma incidence rates for hairdressers are 308/million, placing hairdressers at the third highest risk for occupational asthma after bakers and pastry makers (683/million) and car painters (326/million).

In Belgium questionnaires completed by hairdressing students showed that 14.1% had already had asthma and 26.7% reported wheezing over the past 12 months. A 1996 study estimated that the burden of work-related asthma in Turkish hairdressers was 14.6%. In Italy about half of a group of hairdressers referred for work-related respiratory symptoms were found to have occupational asthma by specific inhalation challenge, along with a strong association with occupational rhinitis.

1.2.2 Non-occupational exposure to cosmetics and personal care products

In a Danish nonoccupational population-based study that included methacholine challenge and skin prick testing it was found that there was no relationship between perfume-associated significant symptoms and atopy, serum ECP or FEV1. However, 42% of subjects reported ocular or airway symptoms from exposure to fragrance, and these 42% were 2.3 times as likely to have bronchial hyperreactivity (BHR) as those without symptoms, suggesting a link between fragrance responses and this defined physiological vulnerability. The fact that 30–40% of those who reported respiratory symptoms in this population-based study had a positive BHR test suggests the possible import of fragrance-induced symptoms, although physiological studies in vulnerable or symptomatic individuals, discussed below, suggest that these relationships are quite complex.

Reported provocation of symptoms by environmental chemicals, prominently including perfumes and cosmetics, typically detected by odor, has been shown to be common, averaging about 10–20% of random samples with a range of 10–60% of more specific subpopulations, asthmatics being a prominent subgroup. A more extreme form of such reported sensitivity to chemicals is multiple chemical sensitivities (MCS) or idiopathic environmental intolerance (IEI). In this case the sensitivity to odors affects behavior and social interactions, becoming potentially disabling. No clear physiological abnormalities or explanations have been discovered. Although many clinicians and researchers favor psychological mechanisms for such odor-induced symptoms, there is substantial disagreement.

Of particular interest to pulmonologists, individuals fitting the description of MCS seem to have a high rate of pulmonary symptoms. Although data come from clinical series, when compared with age- and sex-matched controls, MCS individuals reported on questionnaires from 1.5 to over 10 times the rate of upper and lower respiratory symptoms, and as suggested above, individuals with asthma report higher rates of provocation by cosmetics and personal care products.

1.3 Description of exposures

1.3.1 Major work processes

Hairdressers, besides cutting and shampooing hair, are involved in permanent wave applications and rinsing, in applications of neutralizing agent, in preparing, applying and rinsing hair color, and in preparing, applying and rinsing hair bleaches. Mixing of bleaching powder takes 2–5 minutes per treatment, and it is thought that most exposure to persulfates occurs in this phase, often done in a back room of the salon, rather than during application in the salon *per se*.

1.3.2 Occupational exposures

Hair dressers have three main classes of workplace exposures:

1. para-phenylamine diamine based dyes, generally associated with delayed hypersensitivity contact dermatitis;

2. henna (vegetable dye), a rare cause of occupational asthma; and
3. lacquers and bleaching agents with persulfate salts, known to cause dermatitis, rhinitis and asthma.

We focus on the latter for this respiratory disease text.

There are three categories of hair-dye formulation used respectively for temporary, semi-permanent and permanent hair coloring. The latter are also known as oxidative dyes and are resistant to shampooing. The permanent dyes almost invariably contain ammonium, potassium and sodium persulfates. Persulfate salts are reactive, low-molecular weight compounds widely used in many industries, but particularly cosmetics. The persulfates ($\text{H}_2\text{S}_2\text{O}_8$) are mixed with an oxidant (H_2O_2) immediately before use. Improved hair penetration is achieved with the addition of ammonia releasers such as ammonium chloride or ammonium phosphate. Permanent waving chemicals can be either alkaline or slightly acidic aqueous solutions. They contain thioglycolic acid or hydrogen peroxide, with ammonia added to enhance hair penetration. Thus, potent irritants/oxidants including ammonia, hydrogen peroxide (H_2O_2) and persulfates ($\text{H}_2\text{S}_2\text{O}_8$) are commonly found in the hairdressing environment.

Hair bleaching agents are generally felt to be the most common cause of occupational asthma in hairdressers; however not all studies report that duration of exposure was significantly greater in those who became sensitized. They are the leading causes implicated in specific occupational asthma reports from France and Italy.

1.3.3 Perfumes and nonoccupational exposures

Perfumes are blends of odiferous ingredients made from a diluent (commonly ethanol) and mixtures of up to 3000 natural and synthetic fragrance ingredients including volatile oils and aldehydes, potential irritants and sensitizers. Because many of the ingredients are volatile, exposure is widespread, either intentionally or incidentally in proximity to users. Cleaning agents for home or commercial use are associated with asthma, and also contain perfume agents as well as cleaning agents that may be respiratory irritants or sensitizers.

1.3.4 Quantitation of exposures in hair salons

In a Swedish study exposures to persulfates during mixing were associated with personal exposures of 35–150 μg persulfates/ m^3 and mixing area exposures ranged from 23–50 μg persulfates/ m^3 . In a study of exposure in French salons, H_2O_2 showed mean personal exposure levels of 51 $\mu\text{g}/\text{m}^3$, NH_3 was 900 $\mu\text{g}/\text{m}^3$ and persulfate was 190 $\mu\text{g}/\text{m}^3$. These values are below applicable workplace standards, although many deficiencies in ventilation were noted in this study and would seem to be common in the industry.

1.3.5 Exposure history: practical advice and pitfalls

It is important to understand the layout of a salon, including any separate rooms in which mixing of hair products takes place. Specific questions about windows or mechanical ventilation are important. Although ventilation in salons is often reported as substandard, in the rare instances when exposures have been measured, they have been typically less than applicable threshold limit values (TLV) (H_2O_2 , NH_3 and $\text{H}_2\text{S}_2\text{O}_8$) on either side of the Atlantic. This may reflect that the salons studied were not completely representative of all salons. Of course, for individuals who have become sensitized, adherence to threshold limit values cannot be relied upon to prevent future reactions.

1.3.6 Documentation of exposure and biomonitoring

Exposure monitoring in salons is not commonly performed, and measures of persistent body burden do not exist and are probably not appropriate to the natural history of the relevant conditions. Moscato [3] reports that, although some hairdressers with asthma have positive skin tests to persulfate, it is not a reliable test of sensitization, because many individuals with disease and apparent exposure have negative tests. As with other prominent causes of occupational asthma, especially for low molecular weight antigens, the available skin test is not clearly immunologically (IgE) mediated. One caveat is that anaphylaxis to persulfate skin testing has been reported.

1.4 Respiratory diseases associated with exposure to cosmetics and personal care products

1.4.1 Occupational asthma

Occupational asthma in hairdressers is felt to arise most commonly from sensitization to persulfate salts, although there are case reports with henna as the sensitizer. Pulmonary function test changes and development of asthma are reported during apprenticeship, although latencies of up to 10–15 years appear in the literature. Most published descriptions of occupational asthma in hairdressers is of the allergic sensitization variant; however, there are a limited number of publications describing more immediate responses apparently independent of sensitization. The immunological basis of the sensitization has not been elucidated.

One provocative study implicated hairsprays as triggers of pre-existing asthma. Schleuter and colleagues [4] studied immediate responses to hairspray in 1979. They reported a 10–20% decrease in mid flows in eight asthmatics, with no response in 13 healthy subjects to a 20 second spray of two hairsprays. The investigators attributed this bronchoconstriction response to the perfume content of the hairspray rather than the plasticizer, diethyl phthalate. However, this and other phthalates in indoor air from building products have been subsequently epidemiologically implicated in asthma induction, and they are still prevalent in hairspray at concentrations of up to 3%. Further examination of a potential role for phthalates in respiratory irritation and

asthma is warranted, in both occupational and nonoccupational settings. For more information on the use of phthalates in cosmetics see: http://www.safecosmetics.org/docUploads/NotTooPretty_r51.pdf

1.4.2 Responses to odors

Cone and Shusterman [5] discussed the health effects of indoor odors. They emphasized variability in the human odor response, and that perfumes are a commonly reported exacerbating agent for asthma. The citation supporting the relationship between perfume and asthma derives from a commonly cited convenience sample of 60 asthmatics specifically recruited by Shim and Williams [6] with sensitivity to odors in mind. They documented that physiological responses to odor provocation could occur; and that atropine, beta agonists and cromolyn abrogated responses in three out of four subjects tested. However, subjects were not blind to test exposures and thus the response could have been perceptual rather than irritant. In fact, they raised the possibility of behavioral sensitization to odorants. The differentiation between irritant/allergic airway effects as opposed to behaviorally or perceptually mediated effects is a recurring theme when considering the human (respiratory) response to cosmetics and personal care products. This differentiation is more frequently an issue in general environmental contexts rather than occupational contexts.

Relationships have been documented in individuals among asthma symptoms, hay fever and chemical odorants. A number of well-controlled studies have shown that perfume stimuli induce respiratory symptoms in asthmatics but not always with accompanying physiological change. Millquist [7–9] exposed nonasthmatics, with a history of respiratory symptoms (but no airway obstruction) following nonspecific irritating stimuli, to perfume. She elicited respiratory symptoms (as well as hoarseness, eye irritation, headache and fatigue) without airway obstruction. The symptomatic responses persisted even after using a carbon filter to block odor. In a subsequent study of ocular exposure to perfume, she again elicited asthma symptoms, even in the absence of hyperventilation, as documented by stable end-tidal CO₂. A sensory mechanism, possibly via the trigeminal nerve, was hypothesized, and this integrative approach is a promising avenue for further exploration of individual responses, as well as for therapy. Lastly, she exposed 10 asthmatics (all with provocative concentration for 20% fall in FEV₁ < 2 mg/ml) to a commercial perfume and found no change in FEV₁ compared with a saline exposure and no increase in symptoms.

Similarly Opiekun *et al.* [10] studied mild and moderate asthmatics following a 30 minute controlled exposure to a prototypical fragranced air-sanitizing product. They found increased nasal symptoms, but there were no explanatory physiological changes in nasal mucosal swelling (measured by acoustic rhinometry), no ocular hyperemia, and no significant changes in FEV₁ (other spirometric values were not reported) at 5 or 30 minutes after exposure. These investigations document that both asthmatics and nonasthmatics can respond to perfumes with respiratory symptoms, yet no significant bronchoconstriction. Making this determination between physiological airway responses and perceived respiratory distress can be challenging for the clinician.

There are reports of those with immediate asthmatic (symptom) responses to perfume; however no analytic epidemiology addresses this issue *per se*. There is

substantial literature on how professional cleaners/janitors and users of cleaning sprays have increased asthma morbidity; however this may be more associated with some of the cleaning agents as opposed to the scents and is addressed elsewhere in this book. Attempts to separate the effects of the alcohol vehicle from the active perfume ingredients have suggested that both may play a role in production of spirometric effects and symptoms, with more severe and atopic asthmatics showing greater responses to perfume challenge.

1.4.3 Unexplained symptoms and psychophysiological responses

As suggested in Millquist's work above, many individuals suffer from episodic respiratory symptoms, sometimes triggered by environmental exposures, but do not meet diagnostic criteria for asthma or other conditions: they do not have bronchospasm. Prominent among reported triggering exposures are cosmetics, with frequently described exposures including cosmetic counters at department stores, churches and office or classroom environments where coworkers use perfumes and other cosmetics. The limited epidemiology has been described above, but there are a number of pertinent clinical studies that have been carried out and suggest the importance of odor-triggered neural mechanisms as explanations for these symptoms.

Van den Bergh suggested learned responses to odors of a Pavlovian nature that can be conditioned or deconditioned. A group in Toronto found that panic symptoms could be triggered by standardized stimuli much more readily in those with unexplained symptoms and suggested a relationship between unexplained symptoms, panic attacks and hyperventilation. Although this has not been studied in asthmatics, and does not directly concern perfume scents, it provides a potential mechanistic underpinning to understand individuals with complaints of respiratory distress attributed to scents, and suggests the design of behaviorally based therapeutic strategies where pathological pulmonary disease has been excluded.

1.5 Diagnosis and management of occupational asthma in hairdressers

There are no randomized trials to guide diagnosis or management of occupational asthma in hairdressers. Diagnosis of occupational asthma in hairdressers is not always straightforward due to the lack of reliable markers of sensitization to persulfate salts as discussed above, but general methods have been reviewed [11]. Both immediate and delayed symptom responses are reported. Because an underlying IgE mechanism is not reliably demonstrated, immunological tests by skin prick or serum-specific IgE lack both sensitivity and specificity. Thus, reliable confirmation of clinical suspicion relies on specific inhalation challenge testing. Various techniques have been described, although such challenges are not in widespread use in many areas of the world, particularly the USA.

Treatment of allergic occupational asthma is via standard protocols with avoidance of exposure at the top of the list. Once a diagnosis of occupational asthma in a hairdresser, usually to persulfate salts, has been made, exposure reduction or

elimination is the most desirable therapeutic alternative. Use of respiratory protection is described but without apparent success, and improved hygiene of salons is often difficult to accomplish. In one study of eight cases, mean exposure duration prior to diagnosis was 15 years and mean duration of symptoms before diagnosis was 38 months, suggesting that improved surveillance could be a key to reducing morbidity.

1.5.1 Medical management of reactions to scented products

Once physiological responses to environmental or occupational exposures have been excluded, a more difficult set of management challenges faces the practitioner. Pulmonary medications have little relevance unless there is comorbid asthma. Speech therapy or behavioral approaches may be useful for upper airway (vocal cord) dysfunction, which may be triggered by irritants and possibly nonirritating odors.

Psychotherapy, anxiolytic medication, cognitive-behavioral therapy (CBT) and biofeedback have all been tried clinically, and have shown responses for individual cases in resolving respiratory and other symptoms associated with odiferous stimuli. More rigorous randomized trials have been conducted in broader groups of somatizing patients, and shown significant, 20–40%, improvement in symptoms and limitations, with courses of cognitive-behavioral therapy.

Blind referrals to mental health practitioners are often ineffective. The referring pulmonologist must clearly communicate that organic lung disease has been excluded, freeing the mental health practitioner to concentrate on reducing symptomatic responses, possibly even in the face of continued exposure to moderate levels of nonsensitizing cosmetics.

The ideal CBT takes place in the setting of a physician's office, as some patients with these symptoms are reluctant to view their symptoms as psychological. It is sometimes useful to convey to the patient that they need to demonstrate the power of 'mind over matter', developing their mental strength to overcome as yet unidentified, but not life-threatening, problems in their body.

1.5.2 Other illnesses

Upper extremity musculoskeletal complaints are associated with work as a hairdresser, and can largely be addressed through client chairs that are adjustable in height. Use of nail cosmetics in nail salons is gaining increasing popularity worldwide. Although a number of irritant compounds are used, there are no reports of respiratory disease in the literature. Ethyl methacrylate, formerly used in artificial nail processing, has been linked to asthma. Its use is largely discontinued.

1.5.3 Medicolegal and compensation

Individual countries and states vary in their system of compensation and requirements. In those places where specific inhalation challenge is a component of compensation evaluation, this bodes well for specific identification of cases, allowing for appropriate

compensation. In the USA, where specific challenge testing is not common, less direct evidence probably leads to less efficient, and likely more contentious, determinations.

1.5.4 Public health

Some of the epidemiology has indicated an increased prevalence of asthma among hairdressing apprentices. In one study of hairdressers there was a mean of 38 months between symptom onset and diagnosis, accounting for fairly poor outcomes with persistent symptoms and a decline in FEV₁, despite cessation of exposure. This emphasizes the importance of surveillance and early recognition of occupational disease if there is to be any confidence of avoiding long-term impairment. Development of nonsensitizing products for hair bleaching is clearly a goal.

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