



## REVIEW ARTICLE

## Allergic contact dermatitis in children: which factors are relevant? (review of the literature)

Flora B. de Waard-van der Spek<sup>1</sup>, Klaus E. Andersen<sup>2</sup>, Ulf Darsow<sup>3</sup>, Charlotte G. Mortz<sup>2</sup>, David Orton<sup>4</sup>, Margitta Worm<sup>5</sup>, Antonella Muraro<sup>6</sup>, Peter Schmid-Grendelmeier<sup>7</sup>, Ramon Grimalt<sup>8</sup>, Radoslaw Spiewak<sup>9</sup>, Odilija Rudzeviciene<sup>10</sup>, Carsten Flohr<sup>11</sup>, Susanne Halken<sup>12</sup>, Alessandro Fiocchi<sup>13</sup>, Luis M. Borrego<sup>14</sup> & Arnold P. Oranje<sup>15</sup>

<sup>1</sup>Department of Dermatology, Erasmus MC University Medical Center Rotterdam and KinderHaven Havenziekenhuis, Rotterdam, The Netherlands; <sup>2</sup>Department of Dermatology and Allergy Centre, Odense University Hospital, University of Southern Denmark, Odense, Denmark; <sup>3</sup>Department Of Dermatology and Allergy Biederstein, Technische Universität München, ZAUM-Center for Allergy and Environment, Munich, Germany; <sup>4</sup>Department of Dermatology & Allergy, Amersham Hospital Whielden Street, Amersham Bucks, UK; <sup>5</sup>Department of Dermatology and Allergy, Charité - Universitaetsmedizin Berlin, Berlin, Germany; <sup>6</sup>Department of Pediatrics, Padua General University Hospital, Padua, Italy; <sup>7</sup>Department of Dermatology, Allergy Unit, University of Zürich, Zürich, Switzerland; <sup>8</sup>Department of Dermatology, University of Barcelona, Barcelona, Spain; <sup>9</sup>Department of Experimental Dermatology and Cosmetology, Jagiellonian University Medical College, Krakow, Poland; <sup>10</sup>Faculty of Medicine Clinic of Children's Diseases, Vilnius University Children Hospital, Vilnius, Lithuania; <sup>11</sup>Department of Paediatric Dermatology & Allergy, St John's Institute of Dermatology, Guy's and St Thomas' Hospital NHS Foundation Trust and King's College London, London, UK; <sup>12</sup>Department of Pediatrics, Odense University Hospital, Odense, Denmark; <sup>13</sup>Division of Allergy, Pediatric Hospital Bambino Gesù, Rome, Vatican City, 00165, Italy; <sup>14</sup>Department of Allergology/Immunology, Hospital CUF Descobertas, Lisbon, Portugal; <sup>15</sup>Departments of Dermatology and Pediatrics, Erasmus MC-Sophia Children's Hospital and KinderHaven Havenziekenhuis, Maastricht Ziekenhuis Rotterdam, The Netherlands

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### Correspondence

Flora B. de Waard-van der Spek MD, PhD, Department of Dermatology, Erasmus MC, PO Box 2040, 3000 CA Rotterdam, The Netherlands.

Tel.: +31 10 7034880

Fax: +31 10 2021274

E-mail: f.dewaard@erasmusmc.nl

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### Abstract

Allergic Contact Dermatitis (ACD) in children is increasing. Sensitization to contact allergens can start in early infancy. The epidermal barrier is crucial for the development of sensitization and elicitation of ACD. Factors that may influence the onset of sensitization in children are atopic dermatitis, skin barrier defects and intense or repetitive contact with allergens. Topical treatment of ACD is associated with cutaneous sensitization, although the prevalence is not high. ACD because of haptens in shoes or shin guards should be considered in cases of persistent foot eruptions or sharply defined dermatitis on the lower legs. Clinical polymorphism of contact dermatitis to clothing may cause difficulties in diagnosing textile dermatitis. Toys are another potentially source of hapten exposure in children, especially from toy-cosmetic products such as perfumes, lipstick and eye shadow. The most frequent contact allergens in children are metals, fragrances, preservatives, neomycin, rubber chemicals and more recently also colourings. It is very important to remember that ACD in young children is not rare, and should always be considered when children with recalcitrant eczema are encountered. Children should be patch-tested with a selection of allergens having the highest proportion of positive, relevant patch test reactions. The allergen exposure pattern differs between age groups and adolescents may also be exposed to occupational allergens. The purpose of this review is to alert the paediatrician and dermatologist of the frequency of ACD in young children and of the importance of performing patch tests in every case of chronic recurrent or therapy-resistant eczema in children.

Allergic contact dermatitis (ACD) in children is increasing (1). Sensitization to contact allergens may already begin at an early age. The data on prevalence of contact allergy among

children visiting dermatology clinics vary between 15% and 71% (2–5). Patch testing is the gold standard diagnostic test (6, 7).

The different patients' populations are difficult to compare because their numbers vary from 53 to 1023 patients, and there is a considerable variation in age and sex distribution, patch test materials, methodology, and selection of patch test allergens. Moreover, there are regional differences in exposure to different allergens. The most common contact allergens in children are metals (nickel, cobalt, and chromate), fragrances, preservatives, neomycin, rubber chemicals and p-tert-butylphenol-formaldehyde resin (2, 8–12).

In this review, we will present epidemiological data, and factors that may influence early sensitization to contact allergens in children. The relationship between contact sensitization and atopic dermatitis, and the role of emollients and skin care products, natural remedies, perioral and perianal dermatitis, juvenile plantar dermatosis, shoes, shin guards, tattoos, textiles, diapers, and toys will be discussed.

## Epidemiology

During the last 30 yr, several studies have reported a considerable number of children with contact allergy and allergic contact dermatitis (ACD) confirming that allergic contact dermatitis is common in children and may cause a significant clinical problem (1, 2).

The incidence and prevalence of contact allergy and ACD in the general population of children are largely unknown because only a few systematic studies in unselected populations were undertaken (2). A point prevalence of contact allergy of 15.2% was found among 1146 8th-grade school children in Odense, Denmark (8). The point prevalence of allergic contact dermatitis was 0.7%, and the lifetime prevalence of ACD was estimated at around 7%. Other studies reported the point prevalence of contact allergy to be 13.3–23.3% in unselected children aged 5–14, 7–12 and <18 yr, respectively (9–11). However, the relevance of positive patch test results was not provided, and therefore, an accurate estimate of allergic contact dermatitis could not be given, the percentages can be too high. A study in 321 very young children showed a high prevalence of contact sensitization. Two hundred children (62.3%; 102 girls and 98 boys aged 3–36 months [mean age 27 + 5.6 months]) developed at least one positive reaction. The most frequent patch test reactions were to metals, cocamidopropyl betaine, neomycin, and methylchloroisothiazolinone/methylisothiazolinone (7). In a recent review, sensitization rates of 26.6–95.6% in selected groups of children with suspected ACD was reported (1), which is higher than the prevalence found in similar material earlier. The associated relevance was 51.7–100%. Neither sex nor the presence or the absence of atopic dermatitis seemed to influence the risk of ACD in children.

The sensitization rate is increasing with age as the environmental exposures accumulate. Many studies reported on prevalence increasing with age or on similar prevalence rates in the different age groups (10, 11). However, a few studies reported an increased rate of contact allergy among younger children (13, 14). The majority of studies indicated a female predominance in contact allergy and ACD (2, 8, 15). Especially, nickel allergy has been found to be more common

among girls (2). Nickel is also one of the most frequent contact allergens in patients with atopic dermatitis. However, young children are not as often patch tested as the older ones and adults. Belloni-Fortina et al. (7) evaluated contact sensitizations in patients younger than 3 yr of age with suspected contact dermatitis and found no statistically significant differences between children younger and older than 24 months of age. Contact sensitization is not rare in young children, clinical relevance needs to be studied further.

The prevalence of contact allergy in clinics over time was evaluated in a couple of studies. The overall impression was that contact allergy has become more frequent in recent years (13, 16). Both studies reported that the rate of sensitizations to different allergens varied largely over time, and therefore, periodic evaluation of patch test results was necessary to update the test series.

A more frequent exposure to allergens at a younger age, for instance, through ear piercing and the use of cosmetic products seems at least partly responsible for the observed increase in prevalence. Improved diagnosis of ACD and increased use of patch tests in children resulting in improved detection of contact allergies may be other reasons for this observation.

## ACD and filaggrin

Recently, the key role of the protein filaggrin (FLG) in maintaining an effective skin barrier was demonstrated (17). Carriage of FLG loss-of-function mutations showed a strong association with atopic dermatitis and lack of FLG expression may also predispose to some forms of ACD by allowing easier contact of haptens with epidermal antigen-presenting cells. The few studies, to date, on the association between FLG mutations and ACD indicated on an increased risk of nickel sensitization and reported reactions to jewelry, in particular in women and in the presence of concomitant AD (18). However, no association with sensitization to other haptens was observed, and overall, the role of FLG mutation carriage in ACD etiology appears less important than in AD. It is also likely that other genetic factors play a role in the development of ACD (19, 20).

## Factors inducing contact allergy

Factors that may influence early sensitization in children are atopy, in particular atopic dermatitis, and other factors inducing skin barrier defects and contact at an early age with several haptens that are able to sensitize children.

## Atopic dermatitis

Allergic contact dermatitis is not rare in children with atopic dermatitis (AD) (21–23). The relationship between atopy and ACD is poorly understood. Whether patients with AD are more prone to ACD than non-atopic individuals remains controversial (24). The role of contact allergy in AD patients is frequently underestimated. Studies have indicated that there is a similar prevalence of ACD in patients with AD and

non-atopics (12, 14, 16, 23, 25–27). In the past, there was evidence to support lower rates of ACD in atopics (28). A higher rate of false-positive reactions was reported in atopic individuals (29, 30).

Preventive measures from an early age should be introduced to avoid contact with nickel-containing objects, perfumed cosmetics, and products or topical medication including lanolin and neomycin in AD patients (31).

Contact sensitization may worsen the skin condition and influence the course in patients with atopic eczema. Moreover, sensitized atopic subjects may respond to very low concentrations of contact allergens because of their impaired skin barrier function and hyper-reactivity to irritant stimuli enhancing contact reactions (32).

In a recent study of 101 patients with suspected allergic contact dermatitis (48 males and 53 females, aged 6–18 yr, mean 11.7 yr), at least one positive patch test reaction was noted in 89% of atopic patients and in 66% of non-atopic patients. EASI (Eczema Area and Severity Index) scores higher than 10 correlated with a higher probability of finding more than three positive patch tests in one patient. The most common sensitizations were to nickel sulfate (31%), followed by wool alcohols (18%), p-tert-butylphenol-formaldehyde resin (PTBF resin) (15%), and *Myroxylon pereirae* (12%). Statistically significant differences between atopic dermatitis and non-atopic patients were observed, with 20% (n = 11) of the atopic patients showing positive responses to *Myroxylon pereirae* and 19% (n = 10) of those with atopic dermatitis having reactions to fragrance mix 1 (33). However, no difference in the rates of sensitization or ACD in atopic dermatitis children as compared with non-atopics were reported in other studies (12–14, 16, 23, 26, 27).

Antiseptics and emollients seem to be the most frequent causes of contact allergy to topical treatment in children with atopic dermatitis. Chlorhexidine is one of the allergens that is often used leading to skin problems (34). Topical corticosteroids may be sensitizing and, if they are, the patch test reactions are delayed and must be read after 7 days or even later (34–36). Bufexamac is a non-steroidal anti-inflammatory drug that has been used in topical drug specialities. In patients with atopic dermatitis differences in sensitization rates to bufexamac were observed, probably due to difference in exposure to bufexamac (31, 34). Because of the (relatively low) risk of serious contact allergic reactions and erythema-multiforme-like reactions after topical administration of bufexamac, and a very limited evidence for the effectiveness of bufexamac, the European Medicines Agency's Committee for Medicinal Products for Human Use (CHMP) concluded that the benefits of the bufexamac-containing medicines do not outweigh its risks and recommended that they should be taken off the market across the European Union (37).

Atopic patients are at a significant risk of developing contact dermatitis, especially of the hands, when exposed to occupational irritant factors, that is, chemicals, water or soil. Preventive strategies should be developed and optimized to reduce the incidence of occupational dermatitis in AD patients (38, 39).

### Emollients and skin care products

Emollients have been used for many years, especially in atopic dermatitis, and are considered as the mainstay of maintenance therapy. They are also used as an additional in-between therapy in ACD. Hydration of the skin is usually maintained by at least twice daily application of moisturizers with a hydrophilic base. Barrier preparations can also have the form of bath oils or shower gels, emulsions or micellar solutions. There is limited evidence for the benefits of using emollients. A randomized controlled trial by Grimalt showed that the correct use of emollients reduced the amount of corticosteroids necessary for effective treatment (40). Certain moisturizers could improve skin barrier function in atopics and reduce their skin susceptibility to irritants (41). However, data for patients with ACD are lacking. Regimens for basic/maintenance therapy are still awaiting validation based on systemic reviews and a Cochrane review is in preparation (42).

An emollient consists of a carrier-containing lipophilic (natural oils or waxes, synthetic mineral oil compounds) and/or hydrophilic (water, moisturizer, and gels) components, and other ingredients (a moisturizer, emulsifiers, and preservatives). Urea-based products are suitable for use in children older than 3 yr. In infants and young children, these products often cause stinging or burning sensations. Glycerin seems better tolerated in younger children (41). Products with a high content of propylene glycol can cause irritant contact dermatitis, allergic contact dermatitis, and non-immunologic contact urticaria, and should not be used in children younger than 2 yr (43).

**Table 1** Some ingredients of emollients and their potential risks

Ingredient		Comments
Emulsifier	Wool wax and wool wax alcohols	Contact sensitizers
	Cetyl-stearyl alcohol	Contact allergen and irritant
Moisturizer	Glycerin	Well tolerated
	Urea	Young children: stinging Suitable for children aged 3 yr and older
Humectant, Solvent	Propylene glycol	Irritant contact dermatitis, allergic contact dermatitis, and non-immunologic contact urticaria
Preservatives	Diazolidinyl urea	Irritant contact dermatitis, allergic contact dermatitis, and non-immunologic contact urticaria
	Imidazolidinyl urea	Contact sensitizers
	Quaternium 15	
	2-bromo-2-nitropropane-1,3-diol	
	Formaldehyde releasers, MCI, MCI/MI	
Fragrances		Contact sensitizers

MDBGN, methylidibromoglutaronitrile; MCI, methylchloroisothiazolinone; MCI/MI methylchloroisothiazolinone, methylisothiazolinone.

Many of the additives mentioned are potential contact sensitizers (44). (Table 1)

There is some evidence that large preventive use of emollients containing allergens such as peanut (45) or oat (46) may induce allergic eczema and increase the risk of skin sensitization and allergy.

Most cases of ACD to skin care products are caused by leave-on cosmetics. The risk of developing ACD from rinse-off products such as soaps, shampoos, and shower foams has been less studied; however, they seem a rare cause of dermatitis in children (47). Formaldehyde releasers, methylidibromoglutaronitrile (MDBGN), cocamidopropyl betaine, and rarely methylchloroisothiazolinone/methylisothiazolinone (MCI/MI) may be the culprits. These preservatives are added to water-containing cosmetics (personal care products and toiletries) to prevent the growth of microorganisms (48–50).

The results of patch tests to own cosmetics must be interpreted with caution in the view of the potential risk for false-positive reactions, especially in case of rinse-off products, which in daily use remain on the body for a very short period of time. For patch testing rinse-off products, a 1% dilution in water is commonly recommended. In cases of doubt, a dilution series in a reasonable concentration range is recommended. It is usually safe to patch test leave-on products as is. When testing to preservatives petrolatum-based patch test, preparations seem statistically significantly more sensitive than aqueous solutions of the same allergens (51).

There is a need for more detailed description of all the relevant sensitizers including denomination of the causative products. Ingredient labeling on cosmetics is very important to help identifying possible allergens in products.

### Natural remedies

The use of natural remedies is increasing. These products are generally considered safe. However, some of these products contain potential sensitizers and may induce allergic contact dermatitis (52). Herbal therapies have been used for centuries. The commercial production of tea tree oil, extracted from *Melleuce alternifolia* Cheel, has increased. One should be aware of the sensitizing effects of tea tree oil. This oil has to be kept in the dark, and 'older' tea tree oil becomes a strong sensitizer due to oxidation. Another example is balsam of Peru, a resin of exotic plant *Myroxylon pereira*, which is a quite common component of natural remedies, next to its use as fragrance and food flavoring agent. Its sensitizing properties have been long known. Propolis (bee glue) allergy is seen with increasing frequency in individuals who use propolis in biocosmetics and for self-treatment (53–56). Other natural remedies reported to cause of ACD in children include Marigold (*Calendula officinalis*), and carnauba wax (*Copernicia prunifera*) (57, 58). Apart from the specific hypersensitivity, Marigold belonging to the *Compositae* family, is also known to cause irritant as well as phototoxic reactions. Sesquiterpene lactones (SL) are the main allergenic components in *Compositae* plants, and the SL-mix is a useful screening allergen for *Compositae* dermatitis, which may appear as hand eczema in young children (38). It is important to always ask about use of

natural remedies parallel to prescribed treatment, which may sustain the symptoms of ACD in children (52).

### Perioral and perianal dermatitis

Perioral dermatitis, a common skin disease in young women, has also been occasionally reported in children (59). Most perioral dermatitis cases in children are associated with lip licking or inhalation steroids (41, 60, 61). In a single report, perioral dermatitis in eight children was linked to the use of physical sunscreens with a high sun protection factor (probably by micropigments) (62).

Perioral dermatitis based on contact allergy is diagnosed after exclusion of the earlier mentioned common causes. Dental fillings, toothpaste, and rosin in chewing gum were reported as causes of sensitization (63–66).

Perianal dermatitis is probably the most common cutaneous disorder of the genito-anal area. Studies on the epidemiology and causative factors are rare and in children even rarer (67). Polidocanol has been indicated a cause in a few cases, including children (68).

### Juvenile plantar dermatosis and foot eruptions

Foot eruptions in children are usually self-limiting, but occasionally symptoms may persist and be resistant to treatment. Allergic contact dermatitis must be considered in such cases.

Teixeira reported on a 5-yr-old female patient, with family and personal history of atopic dermatitis and plantar juvenile dermatitis, presenting as bilateral, symmetrical diffuse erythema, vesicles, bullae and ulcerations on the dorsum of the feet and toes as well as on the plantar surfaces, accompanied by severe itching. Results of patch tests were positive to potassium dichromate, cobalt chloride, colophony, balsam of Peru and PTBF resin (69). The main discussion is whether the patients affected by juvenile plantar dermatosis would be or not be more prone to develop ACD. In fact, many cases show a mixed clinical picture of atopy associated with ACD. Many patients referred to the clinic for further investigation because of a suspected ACD to shoe materials are ultimately diagnosed with juvenile plantar dermatosis. The high prevalence of this atopic manifestation may lead to a misdiagnosis in some cases. Many atopic children develop erythema and desquamation plantar surfaces of both feet during spring or in relation to sports. Some patients also demonstrate subtle vesicular aspects, and in very exceptional cases, this endogenous eczema may also affect the dorsi of the feet. Darling et al. (70) evaluated the relevance of all patch tests performed in children (<18 yr) with dermatoses of the soles between 1997 and 2009. Forty-one children were identified, including 27 children with inflammatory dermatitis affecting the sole and 14 with juvenile plantar dermatosis (JPD). Forty-eight percent of children with inflammatory dermatitis of the sole and 29% of children with JPD had at least one relevant reaction. Of the children with relevant reactions, 76% had a personal or family history of atopy. Rubber additives and potassium dichromate were the most frequent allergens identified.

Contact dermatitis to shoes has not been studied extensively in children, accounting for its probably underestimated incidence and prevalence (71). The most common sensitizers present in shoes resulting in ACD are potassium dichromate, PPD, and PTBF resin. Recently, cases related to dimethyl-fumarate were described mostly in adults but some also in children (109). If contact dermatitis to shoes is diagnosed, avoiding the causative shoes or identified allergens is often sufficient to alleviate the symptoms.

### Shin guards

In spite of the worldwide popularity of soccer among the youth and the common requirement of the use of protective shin guards, reports of allergy to shin guards are sparse. This is surprising in light of the fact that this equipment is often made of materials known to cause allergies and that friction and moisture from the use of shin guards during sport activities would seem to predispose soccer players to the development of an allergic response (72). Weston has published a retrospective analysis of eight children aged 9–16 yr evaluated for a persistent or recurrent dermatitis that appeared under soccer shin guards. To examine the possibility of contact allergy, all were patch tested with a series of 51 related allergens and three or four additional tests to pieces of the shin guard components. The tests were read at 48- and 120 h and all remained negative. Irritant contact dermatitis (ICD), not ACD, was ultimate diagnosis in these subjects, sweating, and friction postulated as the main contributors to the irritancy (73). A recent study reported contradictory results, as some of the patients with



**Figure 1** A boy with persistent, sharply demarcated dermatitis that appeared on the lower legs under soccer shin guards.

suspected ICD also showed allergic sensitization (72). De Waard-van der Spek et al., reported positive allergic patch test reactions in five children, all boys aged 9–10 yr, to contact material from shinbone protectors. All suffered from shin eczema (Fig. 1), three of them had atopic dermatitis (23). In shin guards, rubbers components and thiourea derivatives are the most common sensitizers.

### Tattoos

Henna is a greenish brown vegetable dye that rarely causes allergic contact dermatitis. In black henna tattoos, *p*-phenylenediamine (PPD) is added to increase the intensity of the color. PPD is also used as a permanent hair-coloring agent, and as an accelerator for rubber vulcanization. This allergenic chemical may cause severe hypersensitivity reactions. Temporary tattoos painted with PPD-contaminated henna may have permanent consequences. Extreme patch test reactions to PPD are not uncommon. Exposure to 'black henna' tattoos and to hair dyes, are the main cause of strong patch test reactions to PPD in children aged 14 yr and younger (74). The fashion of having temporary henna tattoos in children should be discouraged because of the potentially serious consequences of sensitization to PPD for the future, including severe ACD from hair dyes and cross-reactive chemical compounds such as azo dyes, sulfonamides, *p*-aminobenzoic acid sunscreens, and local anesthetics such as benzocaine or procaine (23, 75–79).

### Textiles

The prevalence of textile dermatitis in children has been poorly investigated in spite of the enormous variety of clothing on the market and frequent use of synthetic fibers and dyes in children's clothing. In most countries, disperse dyes have not been included in the standard patch test series. Contact allergy to disperse dyes in textiles is documented in prevalence studies mostly in adult population (80). It was demonstrated that frequency of textile-dye allergy is increasing (81). However, only few authors studied contact sensitization to textile dyes in children and found the prevalence to be 3.1–4.6% (4, 82–91). In the study by Manzini et al. (4) in 21 children sensitized to disperse dyes, the thighs were most frequently involved and could have resulted from a frequent use of synthetic material in children's trousers. Giusti et al. (82), found that from 51 disperse dye-allergic children, the feet, the axillae, and the groin were most commonly involved in a subgroup of the patients without AD, whereas in children with AD, the face and the flexural areas of the limbs appeared to be affected most.

Formaldehyde and its resins are found in clothing labeled as 'wrinkle-resistant' and can also cause ACD (92). The highest concentration of formaldehyde is found in rayon, corduroy, silk and blended cottons, the lowest in 100% polyester (93). Apart from the most widely known dyes and fixing resins, the glues, rubber, and metal accessories may also lead to sensitization (94). Infants' and children's clothing often have metal components such as snaps and buckles. Fisher described a 7-month-old infant with ACD to nickel-plated snaps in his sleepwear (95).

## Diapers

Diaper dermatitis is an acute inflammatory toxic eczematous disorder in the diaper area. More than 50% of the infants have one or more episodes of irritant diaper dermatitis. Irritant diaper dermatitis generally involves the buttocks, the genitals, the lower abdomen, and the upper thighs, but the skin folds are usually spared (94). Clinical manifestations can range from asymptomatic erythema to painful scaling papules and superficial erosions.

The prevalence of diaper ACD is unknown, but seems to be extremely low (96–98). A subset of allergic diaper dermatitis, called 'Lucky Luke' dermatitis develops because of the sensitization to rubber components in diapers. This variant is localized on the outer buttocks and the hips, which reminds of a cowboy's gun belt holsters. Children with 'Lucky Luke' dermatitis, tested positive to rubber components of the diapers, the rubber chemical mercaptobenzothiazole, the glue PTBF resin, and cyclohexyl thiophthalimide, a vulcanization retarder (97, 100–102).

Another observed manifestation was the development of miliaria-like rash under the place of the stickers. The modern disposable ultrathin diapers do have a very low allergenic potential (personal communications 2011, APO).

Allergic contact dermatitis should be considered in the differential diagnosis of diaper dermatitis. Patch testing may be helpful in identifying the cause. The use of dye-free diapers helps to achieve the improvement in infants with diaper dye dermatitis. Improved product design and features may explain the decline in observed diaper dermatitis among infants. Children with frail, sensitive skin or with skin diseases may benefit from using high-quality products with superabsorbent polymers and water vapor-permeable back sheets, to minimize the risk of complications (103).

## Toys

Toys are another potentially important source of hapten exposure in children, especially from toy-cosmetic products, such as perfumes, lipstick, and eye shadow. Fragrances are the main culprit, and levels of exposure from toys can well exceed industrial guidelines (104). Although the presence of nickel, chromium, and cobalt in cosmetics is prohibited by European Law, toy make-up such as lipstick, lip gloss, and especially powdery eye shadow has been occasionally found to contain nickel, chromium, and cobalt at sensitization levels, exceeding the recommended 5 ppm limit (104). Nickel is also found in more conventional toys such as costume jewelry at levels detectable with the dimethylglyoxime test which marks the sensitizing level. Early nickel exposure from toys may not only be responsible for inducing nickel allergy in children, but also can sustain existing nickel dermatitis (106, 107).

Very recently in a follow-up study in 1206 young adults from a cohort of 1501 unselected 8th grade schoolchildren established 15 yr ago was found that nickel was the most common contact allergen (11.8%) followed by cobalt (2.3%), colophony (2.0%), thimerosal (1.4%), and p-phenylenediamine (1.1%).

Next to many persistent nickel reactions, a significant number of new nickel sensitizations were found. Nickel was still the most common contact allergen and new sensitizations occur despite the EU nickel regulation (108).

## Conclusion and advices

Sensitization to contact allergens can develop already at an early age. Recent literature reported sensitization rates of 26.6–95.6% in selected groups of children with suspected ACD, which is higher than the previously reported prevalence. The associated relevance was 51.7–100%. Neither sex nor the presence or absence of atopic dermatitis appeared to influence the risk of ACD in children. The rate of positive patch test results reflects different regional exposure patterns, but also the local selection criteria and referral rules for patch testing, and finally the compositions of local patch test series. Therefore, negative patch test results do not fully exclude allergic contact dermatitis. False-negative reactions may, for instance, be due to a missing the causative allergen, which may be identified by further detailed history taking. Nickel is still the most common contact allergen and new sensitizations occur despite the EU nickel regulation.

The epidermal barrier is crucial for the development of sensitization and elicitation of ACD. Recently, the key role of the protein filaggrin (FLG) in maintaining an effective skin barrier against the external environment was reported.

Factors that may influence the onset of early sensitization in children are atopic dermatitis, skin barrier defects and intense or repetitive contact with allergens.

The role of contact allergy in AD patients is frequently underestimated. Many of the additives in emollients contain potential contact sensitizers. Systematic patch testing is necessary in children with moderate-to-severe atopic dermatitis whose condition is refractory to treatment or whose history is suggestive of allergic contact dermatitis. Furthermore, given the high exposure to the same haptens in later life, prevention through exposure avoidance from an early age to the most frequent contact sensitizers, especially fragrances in patients with atopic dermatitis, is very important. Ingredient labeling on cosmetics is very important to help identifying possible allergens in products.

Contact dermatitis to shoe materials has not been extensively studied in children, accounting for its probably underestimated incidence and prevalence. Identifying the presence of causative sensitizers in footwear remains a challenge both to clinicians and researchers. Rubber additives and potassium dichromate are frequent sensitizers responsible for footwear dermatitis. It is important to patch test children with dermatoses affecting the feet, including the soles. A history of atopy or a diagnosis of juvenile plantar dermatosis should not deter this investigation.

Reports of allergy to shin guards are sparse. Irritant contact dermatitis is in most cases mentioned as the cause of the sharply demarcated dermatitis on the lower legs. However, some of the patients also showed true contact sensitization. In the shin guards, rubbers and thioureas are the most common allergens.

The fashion of temporary henna tattoos in children needs to be discouraged because of the future risk of serious consequences of sensitization to PPD. Children's clothes are usually bright in color, and disperse dyes present in children's clothes are causes of textile dermatitis. Diaper allergic contact dermatitis seems to be extremely rare, but may be considered in the differential diagnosis of persisting diaper dermatitis.

Toys are another important source of hapten exposure in children, especially from toy-cosmetic products such as perfumes, lipstick, and eye shadow.

It is very important to remember that ACD in children is not rare and should always be considered when children with

recalcitrant eczema are encountered. Contact sensitization can be found also in very young children. Children should be patch tested with a selection of allergens having the highest proportion of positive, relevant patch test reactions in the given group. The exposure patterns differ between age groups and adolescents may be exposed to occupational allergens.

More studies in unselected, general populations of children are still necessary to obtain more information on the real prevalence and the incidence of contact allergy and ACD and to follow the trends of contact allergy and different contact sensitizations in children.

## References

1. Simonsen AB, Deleuran M, Johansen JD, Sommerlund M. Contact allergy and allergic contact dermatitis in children – a review of current data. *Contact Dermat* 2011; **65**: 254–65.
2. Mortz CG, Andersen KE. Allergic contact dermatitis in children and adolescents. *Contact Dermat* 1999; **41**: 121–30.
3. Romaguera C, Alomar A, Camarasa JM, et al. Contact dermatitis in children. *Contact Dermat* 1985; **12**: 283–4.
4. Manzini BM, Ferdani G, Simonetti V, Donini M, Seidenari S. Contact sensitization in children. *Contact Dermat* 1998; **15**: 12–7.
5. Rudzki E, Rebandel P. Contact dermatitis in children. *Contact Dermat* 1996; **34**: 66–7.
6. Hammonds LM, Hall VC, Yiannias JA. Allergic contact dermatitis in 136 children patch tested between 2000 and 2006. *Int J Dermatol* 2009; **48**: 271–4.
7. Belloni Fortina A, Romano I, Peserico A, Eichenfield LF. Contact sensitization in very young children. *J Am Acad Dermatol* 2011; **65**: 772–9.
8. Mortz CG, Lauritsen JM, Bindslev-Jensen C, Andersen KE. Nickel sensitization in adolescents and association with ear piercing, use of dental braces and hand eczema. The Odense Adolescence Cohort Study on Atopic Diseases and Dermatitis (TOACS). *Acta Derm Venereol* 2002; **82**: 359–64.
9. Barros MA, Baptista A, Correia TM, Azevedo F. Patch testing in children: a study of 562 schoolchildren. *Contact Dermat* 1991; **25**: 156–9.
10. Dotterud LK, Falk ES. Contact allergy in relation to hand eczema and atopic diseases in north Norwegian schoolchildren. *Acta Paediatr* 1995; **84**: 402–6.
11. Weston WL, Weston JA, Kinoshita J, et al. Prevalence of positive epicutaneous tests among infants, children and adolescents. *Pediatrics* 1986; **78**: 1070–4.
12. Goon AT, Goh CL. Patch testing of Singapore children and adolescents: our experience over 18 years. *Pediatr Dermatol* 2006; **23**: 117–20.
13. Seidenari S, Giusti F, Pepe P, Mantovani L. Contact sensitization in 1094 children undergoing patch testing over a 7-year period. *Pediatr Dermatol* 2005; **22**: 1–5.
14. Roul S, Ducombs G, Taieb A. Usefulness of the European standard series for patch testing in children. A 3-year single-centre study of 337 patients. *Contact Dermat* 1999; **40**: 232–5.
15. Dotterud LK, Falk ES. Metal allergy in north Norwegian schoolchildren and its relationship with ear piercing and atopy. *Contact Dermat* 1994; **31**: 308–13.
16. Milingou M, Tagka A, Armenaka M, Kimpouri K, Kouimintzis D, Katsarou A. Patch tests in children: a review of 13 years of experience in comparison with previous data. *Pediatr Dermatol* 2010; **27**: 255–9.
17. Sandilands A, O'Regan GM, Liao H, et al. Prevalent and rare mutations in the gene encoding filaggrin cause ichthyosis vulgaris and predispose individuals to atopic dermatitis. *J Invest Dermatol* 2006; **126**: 1770–5.
18. Novak N, Baurecht H, Schafer T, et al. Loss-of-function mutations in the filaggrin gene and allergic contact sensitization to nickel. *J Invest Dermatol* 2008; **128**: 1430–5.
19. Reich K, Westphal G, König IR, et al. Association of allergic contact dermatitis with a promoter polymorphism in the IL16 gene. *J Allergy Clin Immunol* 2003; **112**: 1191–4.
20. Nacak M, Erbgci Z, Aynacioglu AS. Human arylamine N-acetyl-transferase 2 polymorphism and susceptibility to allergic contact dermatitis. *Int J Dermatol* 2006; **45**: 323–6.
21. Oranje AP, Bruynzeel DP, Stenveld HJ, Dieges PH. Immediate- and delayed-type contact hypersensitivity in children older than 5 years with atopic dermatitis: a pilot study comparing different tests. *Pediatr Dermatol* 1994; **11**: 209–15.
22. Clayton TH, Wilkinson SM, Rawcliffe C, Pollock B, Clark SM. Allergic contact dermatitis in children: should pattern of dermatitis determine referral? A retrospective study of 500 children tested between 1995 and 2004 in one U.K. centre. *Br J Dermatol* 2006; **154**: 114–7.
23. de Waard-van der Spek FB, Oranje AP. Patch tests in children with suspected allergic contact dermatitis: a prospective study and review of the literature. *Dermatology* 2009; **218**: 119–25.
24. Spiewak R. Contact dermatitis in atopic individuals. *Curr Opin Allergy Clin Immunol* 2012; **12**: 491–7.
25. Vender RB. The utility of patch testing children with atopic dermatitis. *Skin Ther Lett* 2002; **7**: 4–6.
26. Zug KA, McGinley-Smith D, Warshaw EM, et al. Contact allergy in children referred for patch testing: north American Contact Dermatitis Group data 2001–2004. *Arch Dermatol* 2008; **144**: 1329–36.
27. Duarte I, Lazzarini R, Kobata CM. Contact dermatitis in adolescents. *Am J Contact Dermat* 2003; **14**: 200–2.
28. Rystedt I. Atopic background in patients with occupational hand eczema. *Contact Dermat* 1985; **12**: 247–54.
29. Lammintausta K, Kalimo K, Fagerlund VL. Patch test reaction in atopic dermatitis. *Contact Dermat* 1992; **26**: 234–40.
30. Klas PA, Corey G, Storrs FJ, Chan SC, Hanifin JM. Allergic and irritant patch test reactions and atopic disease. *Contact Dermat* 1996; **34**: 121–4.
31. Heine G, Schnuch A, Uter W, Worm M, Information Network of Departments of Dermatology (IVDK); German Contact Dermatitis Research Group (DKG). Type-IV sensitization profile of individuals with atopic eczema: results from the Information Network of Departments of Dermatology (IVDK) and the German Contact Dermatitis Research Group (DKG). *Allergy* 2006; **61**: 611–6.
32. Seidenari S, Giusti F. Skin sensitivity, interindividual factors: atopy. In: Van Der Valk PMG, Maibach H, eds. *The Irritant*

- Contact Dermatitis Syndrome*. Boca Raton, FL: CRC Press, 2006: 266.
33. Herro EM, Matiz C, Sullivan K, Hamann C, Jacob SE. Frequency of contact allergens in pediatric patients with atopic dermatitis. *J Clin Aesthet Dermatol* 2011; **4**: 39–41.
  34. Mailhol C, Lauwers-Cances V, Rancé F, Paul C, Giordano-Labadie F. Prevalence and risk factors for allergic contact dermatitis to topical treatment in atopic dermatitis: a study in 641 children. *Allergy* 2009; **64**: 801–6.
  35. Uter W, De Pádua CM, Pfahlberg A, Nink K, Schnuch A, Lessmann H. Contact allergy to topical corticosteroids—results from the IVDK and epidemiological risk assessment. *J Dtsch Dermatol Ges* 2009; **7**: 34–41.
  36. Kränke B, Derhaschnig J, Komericki P, Aberer W. Bufexamac is a frequent contact sensitizer. *Contact Dermat* 1996; **34**: 63–4.
  37. Uter W, Schnuch A. EMA revokes marketing authorization for bufexamac. *Contact Dermat* 2011; **64**: 235–6.
  38. Paulsen E, Otkjae A, Andersen KE. Sesquiterpene lactone dermatitis in the young: is atopy a risk factor? *Contact Dermat* 2008; **59**: 1–6.
  39. Darsow U, Wollenberg A, Simon D, et al. EFADEADV eczema task force 2009 position paper on diagnosis and treatment of atopic dermatitis. *JEADV* 2010; **24**: 317–28.
  40. Grimalt R, Mengeaud V, Cambazard F, Study Investigators' Group. The steroid-sparing effect of an emollient therapy in infants with atopic dermatitis: a randomized controlled study. *Dermatology* 2007; **214**: 61–7.
  41. Lodén M, Andersson AC, Lindberg M. Improvement in skin barrier function in patients with atopic dermatitis after treatment with a moisturizing cream (Canoderm). *Br J Dermatol* 1999; **140**: 264–7.
  42. Oranje AP, De Waard-van der Spek FB, Ordonez C, De Raeve L, Spierings M, Van der Wouden JC. Emollients for eczema. *Cochrane Rev* 2012: [submitted for publication].
  43. Funk JO, Maibach HI. Propylene glycol dermatitis: re-evaluation of an old problem. *Contact Dermat* 1994; **31**: 236–41.
  44. Wolf G, Höger PH. Hypoallergenic and non-toxic emollient therapies for children. *J Dtsch Dermatol Ges* 2009; **7**: 50–60.
  45. Lack G, Fox D, Northstone K, Golding J. Factors associated with the development of peanut allergy in childhood. *N Engl J Med* 2003; **348**: 977–85.
  46. Boussault P, Léauté-Labrèze C, Saubusse E, et al. Oat sensitization in children with atopic dermatitis: prevalence, risks and associated factors. *Allergy* 2007; **62**: 1251–6.
  47. Uter W, Balzer C, Geier J, Frosch PJ, Schnuch A. Patch testing with patient's own cosmetics and toiletries: results of the IVDK, 1998–2002. *Contact Dermat* 2005; **53**: 226–33.
  48. Jensen CD, Johansen JD, Menné T, Andersen KE. Methyl dibromoglutaronitrile in rinse-off products causes allergic contact dermatitis: an experimental study. *Br J Dermatol* 2004; **150**: 90–5.
  49. Jensen CD, Johansen JD, Menne T, Andersen KE. Methyl dibromo glutaronitrile contact allergy: effect of single versus repeated daily exposure. *Contact Dermat* 2005; **52**: 88–92.
  50. de Groot AC, White IR, Flyvholm MA, Lensen G, Coenraads PJ. Formaldehyde-releasers in cosmetics: relationship to formaldehyde contact allergy. *Contact Dermat* 2010; **62**: 2–17.
  51. Fonacier LS, Aquino MR. The role of contact allergy in atopic dermatitis. *Immunol Allergy Clin North Am* 2010; **30**: 337–50.
  52. Küttling B, Brehler R, Traupe H. Allergic contact dermatitis in children – strategies of prevention and risk management. *Eur J Dermatol* 2004; **14**: 80–5.
  53. Hausen BM. Evaluation of the main contact allergens in propolis (1995 to 2005). *Dermatitis* 2005; **16**: 127–9.
  54. Bonitsis NG, Tatsioni A, Bassioulas K, Ioannidis JP. Allergens responsible for allergic contact dermatitis among children: a systematic review and meta-analysis. *Contact Dermat* 2011; **64**: 245–57.
  55. Pietowska J, Czarnobilska E, Spiewak R. The most frequent contact sensitizers and atopic diseases among consecutive patients of a Polish patch test clinic. *Allergy* 2008; **63** (Suppl 88): 320.
  56. Czarnobilska E, Obtulowicz K, Dyga W, Spiewak R. The most important contact sensitizers in Polish children and adolescents with atopy and chronic recurrent eczema as detected with the extended European Baseline Series. *Pediatr Allergy Immunol* 2011; **22**: 252–6.
  57. Hausen BM. Evaluation of the main contact allergens in oxidized tea tree oil. *Dermatitis* 2004; **15**: 213–4.
  58. Jacob SE, Chimento S, Castanedo-Tardan MP. Allergic contact dermatitis to propolis and carnauba wax from lip balm and chewable vitamins in a child. *Contact Dermat* 2008; **58**: 242–3.
  59. Jansen T, Melnik BC, Schadendorf D. Steroid-induced periorificial dermatitis in children—clinical features and response to azelaic acid. *Pediatr Dermatol* 2010; **27**: 137–42.
  60. Kumar P, Parashette KR, Noronha P. Perioral dermatitis in a child associated with an inhalation steroid. *Dermatol Online J* 2010; **16**: 13.
  61. Laude TA, Salvemini JN. Perioral dermatitis in children. *Semin Cutan Med Surg* 1999; **18**: 206–9.
  62. Abeck D, Geisenfelder B, Brandt O. Physical sunscreens with high sun protection factor may cause perioral dermatitis in children. *J Dtsch Dermatol Ges* 2009; **7**: 701–3.
  63. Guarneri F, Marini H. Perioral dermatitis after dental filling in a 12-year-old girl: involvement of cholinergic system in skin neuroinflammation? *Scientific World J* 2008; **8**: 157–63.
  64. Reilly KE, McCarthy LH. Toothpaste allergy with intractable perioral rash in a 10-year old boy. *J Am Board Fam Pract* 2000; **13**: 73–5.
  65. Muñoz FJ, Bellido J, Moyano JC, Alvarez M, Fonseca JL. Perioral contact urticaria from sodium benzoate in a toothpaste. *Contact Dermat* 1996; **35**: 51.
  66. Satyawan I, Oranje AP, Van Joost T. Perioral dermatitis in a child due to rosin in chewing gum. *Contact Dermat* 1990; **22**: 182–3.
  67. Kränke B, Trummer M, Brabek E, Komericki P, Turek TD, Aberer W. Etiologic and causative factors in perianal dermatitis: results of a prospective study in 126 patients. *Wien Klin Wochenschr* 2006; **118**: 90–4.
  68. Frosch PJ, Schulze-Dirks A. Contact allergy caused by polidocanol. *Hautarzt* 1989; **40**: 146–9.
  69. Teixeira M, Machado S, Teixeira A, et al. Severe contact allergy to footwear in a young child. *Contact Dermat* 2005; **52**: 159–60.
  70. Darling MI, Horn HM, McCormack SKA, Schofield O. Sole dermatitis in children: patch testing revisited. *Pediatr Dermatol* 2012; **29**: 254–7.
  71. Roul S, Ducombs G, Leaute-Labreze C, Labbe L, Taieb A. Footwear contact dermatitis in children. *Contact Dermat* 1996; **35**: 334–6.
  72. Powell D, Ahmed S. Soccer shin guard reactions: allergic and irritant reactions. *Dermatitis* 2010; **21**: 162–6.
  73. Weston WL, Morelli JG. Dermatitis under soccer shin guards: allergy or contact irritant reaction? *Pediatr Dermatol* 2006; **23**: 19–20.
  74. Spornraft-Ragaller P, Schnuch A, Uter W. Extreme patch test reactivity to p-phenylenediamine but not to other allergens in children. *J Dtsch Dermatol Ges* 2012; **10**: 258–64.

75. Neri I, Guareschi E, Savoia F, Patrizi A. Childhood allergic contact dermatitis from henna tattoo. *Pediatr Dermatol* 2002; **19**: 503–5.
76. Marcoux D, Couture-Trudel P-M, Riboulet-Delmas G, Sasseville D. Sensitization to para-phenylenediamine from a street side temporary tattoo. *Pediatr Dermatol* 2002; **19**: 498–502.
77. Sosted H, Kohansen JD, Andersen KE, et al. Severe allergic hair dye reactions in 8 children. *Contact Dermat* 2006; **54**: 87–91.
78. Jacob SE, Zapolanski T, Chayavichitsilp P, Connelly EA, Eichenfield LF. P-Phenylenediamine in black henna tattoos: a practice in need of policy in children. *Arch Pediatr Adolesc Med* 2008; **162**: 790–2.
79. Almeida PJ, Borrego L. Temporary henna tattoos with long-term consequences. *Med J Aust* 2009; **191**: 11–2.
80. Ryberg K, Isaksson M, Gruvberger B, Hindsén M, Zimerson E, Bruze M. Contact allergy to textile dyes in southern Sweden. *Contact Dermat* 2006; **54**: 313–32.
81. Hatch KL, Maibach HI. Textile dye allergic contact dermatitis prevalence. *Contact Dermat* 2000; **42**: 187–95.
82. Giusti F, Massone F, Bertoni L, Pellacani G, Seidenari S. Contact sensitization to disperse dyes in children. *Pediatr Dermatol* 2003; **20**: 393–7.
83. Dooms-Goossens A. Textile dye dermatitis. *Contact Dermat* 1992; **27**: 321–3.
84. Pratt M, Taraska V. Disperse blue dyes 106 and 124 are common causes of textile dermatitis and should serve as screening allergens for this condition. *Am J Contact Dermat* 2000; **11**: 30–41.
85. Pecquet C, Assier-Bonnet H, Artigou C, Verne-Fourment L, Saiäg P. Atypical presentation of textile dye sensitization. *Contact Dermat* 1999; **40**: 51.
86. Lazarov A. Textile dermatitis in patients with contact sensitization in Israel: a 4-year prospective study. *J Eur Acad Dermatol Venereol* 2004; **18**: 531–7.
87. Baldari U, Alessandrini F, Raccagni AA. Diffuse erythema multiforme-like contact dermatitis caused by disperse blue 124 in a 2-year-old child. *J Eur Acad Dermatol Venereol* 1999; **12**: 180–1.
88. Giusti F, Mantovani L, Martella A, Seidenari S. Hand dermatitis as an unsuspected presentation of textile dye contact sensitivity. *Contact Dermat* 2002; **47**: 91–5.
89. Lee PW, Elsaie ML, Jacob SE. Allergic contact dermatitis in children: common allergens and treatment: a review. *Curr Opin Pediatr* 2009; **21**: 491–8.
90. Seidenari S, Mantovani L, Manzini BM, Pignatti M. Cross-sensitizations between azo dyes and para-amino compound. A study of 236 azo-dye-sensitive subjects. *Contact Dermat* 1997; **36**: 91–6.
91. Kiec-Swierczynska M, Krecisz B, Swierczynska-Machura D. Allergy to p-phenylenediamine from a black transferable picture tattoo: hypopigmentation and sensitization to clothing dyes in a little girl. *Contact Dermat* 2008; **58**: 174–5.
92. Fowler JF Jr, Skinner SM, Belsito DV. Allergic contact dermatitis from formaldehyde resins in permanent press clothing: an underdiagnosed cause of generalized dermatitis. *J Am Acad Dermatol* 1992; **27** (6 Pt 1): 962–8.
93. Rao S, Shenoy SD, Davis S, Nayak S. Detection of formaldehyde in textiles by chromotropic acid method. *Indian J Dermatol Venereol Leprol* 2004; **70**: 342–4.
94. Pigatto P, Martelli A, Marsili C, Fiocchi A. Contact dermatitis in children. *Ital J Pediatr* 2010; **13**: 36.
95. Fisher AA. Allergic contact dermatitis in early infancy. *Cutis* 1994; **54**: 300–2.
96. Smith WJ, Jacob SE. The role of allergic contact dermatitis in diaper dermatitis. *Pediatr Dermatol* 2009; **26**: 369–70.
97. Roul S, Ducombs G, Leaute-Labreze C, Taieb A. 'Lucky Luke' contact dermatitis due to rubber components of diapers. *Contact Dermat* 1998; **38**: 363–4.
98. Karlberg A-T, Magnusson K. Rosin components identified in diapers. *Contact Dermat* 1996; **34**: 176–80.
99. Alberta L, Sweeney SM, Wiss K. Diaper dye dermatitis. *Pediatrics* 2005; **116**: e450–2.
100. Landro AD, Greco V, Valsecchi R. 'Lucky Luke' contact dermatitis from diapers with negative patch tests. *Contact Dermat* 2002; **46**: 48–9.
101. Belhadjali H, Giordano-Labadie F, Rance F, Bazex J. 'Lucky Luke' contact dermatitis from diapers a new allergen? *Contact Dermat* 2001; **44**: 248.
102. Jacob SE, Herro EM, Guide S, Cunningham B, Connelly EA. Allergic contact dermatitis to pampers™ drymax. *Pediatr Dermatol* 2012; **29**: 672–4.
103. Runeman B. Skin interaction with absorbent hygiene products. *Clin Dermatol* 2008; **26**: 45–51.
104. Rastogi SC, Johansen JD, Menné T, et al. Contents of fragrance allergens in children's cosmetics and cosmetic-toys. *Contact Dermat* 1999; **41**: 84–8.
105. Corazza M, Baldo F, Pagnoni A, Miscioscia R, Virgilli A. Measurement of nickel, cobalt and chromium in toy make-up by absorption spectroscopy. *Acta Derm Venereol* 2009; **89**: 130–3.
106. Hsu JW, Jacob SE. Children's toys as potential sources of nickel exposure. *Dermatitis* 2009; **20**: 349–50.
107. Fischer LA, Menné T, Johansen JD. Experimental nickel elicitation thresholds—a review focusing on occluded nickel exposure. *Contact Dermat* 2005; **52**: 57–64.
108. Mortz CG, Bindley-Jensen C, Andersen KE. Prevalence, incidence rate and persistence of contact allergy and allergic contact dermatitis in the TOACS cohort: A 15 years follow-up. *Br J Dermatol* 2012; doi: 10.1111/bjd.12065. [Epub ahead of print]
109. Toledo F, Silvestre JF, Cuesta L, Borrego L, Pérez M. Correlation between lesion site and concentration of dimethyl fumarate in different parts of shoes in patients with contact dermatitis caused by dimethyl fumarate in footwear. *Contact Dermat* 2011; **65**: 92–5.