

144 Contact Dermatitis: Diagnosis and Therapy

Sharon E. Jacob · Elise M. Herro · James S. Taylor

Introduction

The term contact dermatitis refers to a group of exogenous dermatoses commonly affecting both children and adults, with irritant contact dermatitis (ICD) representing the vast majority of cases (~80%) and allergic contact dermatitis (ACD) accounting for the other large proportion (~20%). The current estimate is that ACD may actually account for as much as 20% of all childhood dermatitis. Patch testing is the gold standard for diagnosing ACD and a number of studies have demonstrated that appropriate epicutaneous patch testing improves quality of life measurements by directing the avoidance of inciting allergens.

In the last decade, multiple international tertiary care centers have reported clinically relevant positive patch test (PPT) reactions being identified in a relatively significant number of pediatric patients with prevalence rates ranging from 21% to 77%. These data sets, combined, serve as a useful guide in the selection of allergens used for patch testing, especially given that many of the same allergens appear across the lists globally. For example, Beattie et al. put forth the “allergens with a positive yield,” Wöhrle et al. outlined the “allergen hit list,” and Jacob et al. described a guide for screening allergens with the highest yield for patch testing children in particular locales.

Contact allergy (rates of positive responses to contact allergens) varies by referral patterns, regional and social variations in allergen exposure, selection criteria for patch testing, and the allergens tested. It is important to recognize the diagnostic clues, signs and symptoms which alert clinicians to the presence of ACD, especially in children where “eczema” and atopic dermatitis abound.

Pathophysiology

Contact dermatitis is a general term that encompasses adverse cutaneous reactions resulting from contact of the surface of the skin or mucous membrane with an exogenous agent. The type of reactions fall into several

categories, namely ICD, ACD, as well as the less common IgE-mediated contact urticaria (CU).

Irritant Contact Dermatitis

Irritant contact dermatitis which accounts for approximately 80% of all contact dermatitis cases is a non-immunologic reaction which does not require prior sensitization or previous chemical exposure. The response is caused by contact with chemicals that directly injure the skin cells by abrasion or irritation. While damage to epidermal keratinocytes induces inflammation, it does not activate an immune cascade.

Onset of symptoms ranges from a few minutes to 48 h, appearing in any location on the skin or mucosa. The severity of the reaction is significantly affected by the concentration of the irritating substance and the duration of exposure. Patient history and clinical presentation are important clues to the diagnosis of ICD. Classically, acute ICD presents as a localized erythema, corresponding to the area of skin that was exposed to the offending agent; however, blistering and erosions may occur with strong or prolonged exposure. In addition, patients are more likely to complain of burning and pain rather than pruritus. Chronic cases can be more difficult to distinguish from ACD, especially when lichenification complicates the clinical and histopathologic picture. A common example of ICD in childhood is *liplicker dermatitis*, a perioral eruption caused by a series of events, such as irritation from cold, dry weather followed by subsequent lip licking to counteract the dryness leads to dermatitis, a secondary reaction from exposure to drying saliva.

Allergic Contact Dermatitis

It is important to differentiate sensitization (the ability to elicit a PPT, a contact allergy, in an asymptomatic person) from the clinical state of ACD, in which a sensitized person demonstrates a clinical dermatitis related to allergen exposure. Sensitization can occur very early in life. Bruckner et al.

tested a group of asymptomatic patients and found that 45% of those with PPT reactions were younger than 18 months. The top five allergens were nickel (12.9%), thimerosal (9.4%), methylchloroisothiazolinone/methylisothiazolinone (2.4%), neomycin (1.2%), cobalt (1.2%), and p-tert butylphenol (1.2%). Of note, 7 of the eleven reactions to nickel were in children less than 16.5 months of age, with exposure sources related to jewelry and clothing fasteners. Of note, sensitization does not always correlate with clinical-allergic disease, ACD. For example, in both adults and children PPT reactions to thimerosal and gold frequently have little direct clinical relevance to the contemporaneous dermatitis; the source of these PPTs may be from past exposure to vaccinations and piercings, respectively.

Allergic contact dermatitis refers to a T-cell mediated, type IV, delayed hypersensitivity reaction (clinical-allergic disease) that results when a person is sensitized to an environmental chemical. Small lipophilic chemicals (haptens) with a low molecular weight (<1,000 Da) penetrate the skin and bind with self-proteins forming hapten-protein complexes (complete antigens). Dendritic cells, antigen presenting cells (APC) of the skin, then uptake these hapten-protein complexes and express them on cell surface major histocompatibility complex (MHC) molecules. In the regional lymph nodes, the APC presents the antigen to naïve antigen-specific T-cells, which in turn differentiate into effector T-cells capable of acting on target cells presenting the antigen in the future. This induction phase of sensitization is usually asymptomatic and takes about 10–15 days.

Subsequent exposure to the antigen, which may occur transepidermally or systemically, e.g., intravenously, by inhalation, or by ingestion, leads to the second or elicitation phase. Elicitation corresponds to the clinical picture of ACD, typically characterized by pruritic, erythematous, and edematous patches and plaques in the distribution of the contactant. Further exposure to the allergen may increase the reactivity pattern to one which is more diffuse and disseminated.

Recent studies have reported relevant PPTs in children at a frequency equivalent to those of adult populations. It is vital to note that the majority of children with ACD are not patch tested and that true prevalence rates may be significantly underestimated.

Demographics and Prevalence of Allergic Contact Dermatitis in Children

In the past 5 years, there has been an influx of reports demonstrating a high prevalence of ACD in pediatric

patients. The majority come from tertiary care centers, with patients referred by dermatologists and allergists, where rates of PPT reactions are significantly higher (41–83%) than in unselected asymptomatic patients from the general population (13.5–24.5%).

In US-based referral center studies a significant number of tested patients have been Caucasian and Hispanic, with Asians and African Americans making up the minority. This data is primarily indicative of the distribution of patients in referral populations, rather than the prevalence of ACD in specific ethnic groups. These same studies have also shown that with respect to race and gender, the demographics for subjects within analyzed age groups have been similar to one another and to the population of all enrolled subjects. Differences in the prevalence rates of a given allergen among different age groups likely reflect the frequency, type, and length of exposure required to induce sensitization to specific chemicals and the age at exposure.

The true prevalence of contact allergy in both adults and children is largely unknown, because a significant number of affected patients are never patch tested.

Diagnosis

The diagnosis of ACD depends on a careful medical and environmental history, a high index of suspicion for ACD, and confirmation by diagnostic patch testing. In general, ACD presents as eczematous plaques largely localized to the site of allergen exposure. Pruritus is a main feature, in contrast to ICD, where burning is more common. Classic presentations, such as geometric shapes or linear streaks on the extremities, may be associated with plant contact dermatitis especially *Toxicodendron* spp. (e.g., poison ivy, poison oak, and poison sumac). Allergic contact dermatitis may also present in focal skin areas, such as the earlobes, periumbilical area, or eyelids.

Pediatric Patch Testing

Patch testing is the “gold standard” for the diagnosis of ACD and should be performed when there is a clinical suspicion and a suggestive history. Currently, there is no approved commercially available patch test screening kit for use in children in the USA. Moreover, in both Europe and the USA, the majority of centers comprehensively screen children with specific allergens selected for individual patients based on the history and clinical distribution

of dermatitis. Comprehensive patch testing is cumbersome and not always available; therefore, a large number of cases of ACD in children do go undetected. Patch test protocols, allergen selection, and interpretation of results are vital to proper diagnosis of ACD and techniques for patch testing children have been discussed in detail.

Special Considerations in Pediatric Patch Testing

Current consensus recommends using the same patch test chemical concentrations as in children as those used in adults. In children older than 12 years (adolescents), testing can basically be performed in the same manner as in adults. While patients as young as 2 years of age have been tested, most clinicians reserve testing for children under the age of 6 to cases in which they have a high index of suspicion, and even then, only selectively test with suspected contact allergens. In addition, the German Contact Dermatitis Research Group (DKG) proposed that patch test allergens be removed after only 24 h in younger children to reduce frequency of irritant reactions, with readings then performed at 48 and 72 h.

One of the intrinsic challenges in pediatric patch testing is the limitation imposed by the anatomically smaller back size of the young child, which translates into a smaller number of allergens applied. The emotional and psychological impact and inherent activity of children means that special attention is required to properly secure the patches. Tools such as games and videos to distract a child during application of the tests are helpful.

Interpretation and the assignment of relevance to PPT results are critical, since there may be only partial concordance between a PPT and ACD. A PPT indicates that an individual has developed sensitization to a chemical allergen, which may or may not be the cause of the patient's contemporaneous dermatitis. Patient history and allergen exposure lists are reviewed to help determine current, past, or uncertain relevance of the test results. Positive patch tests may account for all, part of, or none of the patient's dermatitis. Additionally more than one diagnosis may explain the patient's dermatitis. Allergic contact dermatitis ICD, atopic dermatitis, and CU may co-exist in the same patient. In addition to allergen avoidance, repeat open application use test (ROAT) may be employed by patients to assess improvement after the avoidance of putative allergens. In the ROAT, the patient applies the suspected substance (i.e., lotions, diaper creams, lip balms, etc.) twice daily to the upper arm or posterior auricular area on the same area for a week or more to observe if the dermatitis is reproduced.

Patient counseling is a key component of the patch test protocol, including instruction on keeping patch tests dry by avoiding bathing and sweat-provoking activities, as well as discontinuing the use of potentially interfering medications. For 2 weeks prior to testing, topical corticosteroids or calcineurin inhibitors should not be used in areas where the patches will be applied. Before testing, parents should be educated about the nature and causes of contact dermatitis, realistic expectations for outcome after testing, including allergen avoidance, and the potential for negative patch test results.

Side Effects of Patch Testing

The most common *side effects* of patch testing are the expected local pruritus, burning, and inflammation at the site of application. Pustular and blistering reactions rarely occur, and there is the potential for hypo/hyperpigmentation and persistent reactions. Exacerbations of the patient's presenting dermatitis are to be expected, and while this is usually minimal and bearable, it can serve as an important diagnostic clue in assigning clinical relevance. Information extrapolated from adult studies indicates that the risk of active sensitization to one of the tested allergens is extremely low. Serious adverse effects, such as anaphylactoid reactions to neomycin or bacitracin, are reported to be very rare. Potential benefits of patch testing clearly outweigh the procedure's potential risks and side effects. Use of commercially available patch test chemicals in generally accepted and published concentrations are associated with the fewest side effects.

Important Contact Allergens in Childhood

◆ [Table 144.1](#) lists the predominating 20 pediatric allergens found ubiquitously among patch testing reports from the USA, Canada, Europe (Germany, Italy, UK, France, Spain, Belgium), and Brazil.

Metal Allergens

Nickel is the most prevalent allergen in patch-tested patients of all ages (◆ [Fig. 144.1](#)).

Nickel contact dermatitis classically presents as an eruption on the earlobes, face, and periumbilical area, as a result of contact with items such as jewelry and clothing

■ Table 144.1

Important allergens in children (USA, Canada, Europe, Brazil)

Rank	Allergen	Description	Source	Frequent distribution
1.	Nickel	Metal	Jewelry, buckles, snaps, eyeglasses, orthodontics, studs on school chairs, musical instruments, cell phones, keys, coins	Face/eyelids, earlobes, neck, wrists
2.	Cobalt	Metal	Jewelry, buttons/snaps, ceramics, cement, vitamin B12	Earlobes, neckline, umbilical area, hands
3.	Potassium dichromate	Metal	Tanned leather, matches, cement, pigments (green felt), dental implants	Hands, generalized
4.	Gold	Metal	Jewelry and dentistry products	Eyelids, mouth/lips
5.	Neomycin sulfate	Topical antibiotic	Topical antibiotic preparations	Foot, eczema sites, wounds
6.	Bacitracin	Topical antibiotic	Topical antibiotic preparations	Foot, eczema sites, wounds
7.	Tixocortol pivalate	Corticosteroid, especially hydrocortisone	OTC and prescription creams, lotions, and ointments	Any location topical is applied
8.	Sorbitan sesquioleate	Emulsifier	Pharmaceuticals, cosmetics, ointments, creams, lotions	Iatrogenic – sites of dermatitis
9.	Propylene glycol	Solvent/moistening agent	Pharmaceuticals, foods, cosmetics, personal care products	Face, perioral, in sites of dermatitis
10.	Lanolin	Emollient	Emollients, rust-preventative waxes, soaps, lip balms	Hands, any body area with emollient use
11.	Fragrance mix 1	Mix of 8 fragrances	Perfumes, personal care products, household products, soaps, detergents, cleaners, medicaments	Eyelids/face, neck, mouth/lips
12.	<i>Myroxylon pereirae</i> (balsam of Peru)	Fragrance/flavorant – tree resin	Perfumes and cosmetics, toothpaste, lozenges, flavoring agent	Eyelids/face, neck, mouth/lips
13.	Colophony	Fragrance/adhesive – distillation product of conifers	Personal care products, adhesive bandages, pine extracts	First aid bandage application sites, eyelids/face
14.	Cocamidopropyl betaine	Detergent, surfactant	Shampoo, liquid soap, bath gel, toothpaste, contact lens solutions, make-up removers	Face, scalp, and neck
15.	p-tert butyl formaldehyde resin	Adhesive and neoprene cement allergen	Leather shoes, athletic shoes, protective sports gear, neoprene	Foot, sports gear distribution
16.	Carbamates	Rubber accelerant	Elastic waistbands, shoes, socks, gloves, swimsuits, tires	Waistline, feet, hands
17.	Thiuram	Rubber accelerant	Elastic waistbands, socks, swimsuits, shoes (soles or insoles), gloves, pesticides	Waistline, feet, hands
18.	Para-phenylene diamine	Hair dye chemical	Hair dye, “black-henna” (PPD-adulterated henna) tattoos	Hairline, ears, hands, henna tattoo sites
19.	Disperse dyes (blue 106/124)	Aniline dye	Textiles, diapers, glasses	Peri-axillary bands, diaper edge
20.	Quaternium-15	Preservative-formaldehyde releasing	Cosmetics and topical medications – non-prescription and prescription	Face and body



■ Figure 144.1
Nickel dermatitis

fasteners. With continued exposure and immune stimulation, involvement of distant sites, which are not in direct contact with the metal allergen, is seen in up to 50% of children. This is known as an *id*-reaction, where dermatitis affects sites such as the extremities and the upper trunk, is more diffuse, and may mimic follicular eczema.

Because nickel is so ubiquitous, allergic patients should purchase the commercially available dimethylglyoxime spot test kit to identify if nickel ions are released from metal objects. A few drops of 1% dimethylglyoxime-ammonia (DMG-A) are applied to a cotton tip applicator, which is rubbed against the object in question. If nickel is present at a concentration as low as 1:10,000 on a solid surface and 10 ppm in a liquid, the applicator will turn a pink color. Currently, in the U.S.A., there is an effort to encourage a limitation on the allowable release of nickel to be <0.2 mcg/cm²/week in products with prolonged skin contact, as has been instituted in Europe since 2004.

Cobalt is also a metal that is naturally found in metal ore with nickel and is often used as an alloy with nickel. It can be used to increase the overall strength of other metals. Sensitizing exposures include jewelry, clothing snaps, buttons and metal objects, as well as cosmetics, joint replacements, ceramics, paints, cement, and multi-vitamins (vitamin B12/cyanocobalamin).

Another frequently sensitizing metal is *gold*, which is found in jewelry and dentistry. PPT reactions to gold do not always correlate with the area of suspected ACD. The most clinically relevant presentations usually include eyelid involvement and stomatitis.

Chromate (potassium dichromate), a metal salt derived from chromium, is the final metal to top the

allergen chart. Tanned leather is a potential source of chromate exposure in the household and is found in couches, shoes, boots, belts, and gloves. Vegetable-tanned leather can be used as an alternative. Chromium is also used in dental implants and the metal wire used in orthodontia. Additional sources of chromium include orthopedic prostheses, suture materials (chromated catgut), vitamin supplements, green tattoo ink, some cosmetics with green tints, as well as dyes and pigments, paints, and ceramics.

Antibiotics and Medicament-Associated Allergens

Neomycin sulfate, a topical antibiotic, maintained second place on the list of most common sensitizing allergens for approximately 25 years. More recently, the prevalence has been on a decline, which may be a result of its replacement with other topical antibiotics, such as *bacitracin*. Neomycin, however, is still frequently found in many over-the-counter creams and ointments used for the treatment of superficial wounds or burns, as well as to treat skin, eye, and ear infections. Co-reactivity with other chemically unrelated substances has been noted, likely due to its use in formulations with other antibiotics, antifungals, or corticosteroids.

Corticosteroid allergy is becoming more widely recognized in children. In fact, 0.2–6% of patients have been found to display ACD to one of the five groups of *corticosteroids*. The sensitization potential of group A corticosteroids (e.g., Cortaid, Cortizone-10) is greater than that of the other structural classes [A (5.72%) > B (4.80%) > D1 (3.54%) > D2 (2.13%) > C (1.10%)], likely due to its over-the-counter usage. Tixocortol-21-pivalate is the screening substance for the group A corticosteroids (some investigators also screen with hydrocortisone 1% in alcohol), while budesonide and triamcinolone are the screening substances for class B, and hydrocortisone-17-butyrate for class D2. Cross-reactions between classes are possible, specifically between groups A and D2, as well as between certain corticosteroids in groups B and D2.

Personal Care Product and Vehicle Allergens

Another important and emerging allergen, especially in atopics, is *cocamidopropyl betaine* (CAPB), a surfactant derived from coconut oil and commonly found in foaming

cleansers, shampoos (used in “no tear” formulations), and soaps. Thus, the distribution of dermatitis often involves the head and neck region. The true sensitizers in CAPB are thought to be the manufacturing contaminants, *amidoamine*, and *3-dimethylaminopropylamine* (DMAPA). Cosmetic manufacturers are being encouraged to remove these impurities, which may reduce sensitization rates from products containing CAPB.

Contact allergy to *fragrances* has also been associated with atopic individuals. One of the chemicals used to screen for fragrance allergy is *balsam of Peru* (BOP), a substance derived from the *Myroxylon pereirae* tree. Children (and adults) are commonly sensitized to this allergen. The distribution of dermatitis has a predilection for the face, neck, and axillae. BOP and/or cross-reacting chemicals can be found in cosmetic products, such as perfumes, lotions, diaper-area care products, and toothpastes and mouthwashes, which may cause contact stomatitis or cheilitis. Moreover, BOP may be found in pharmaceutical preparations, scents, and flavorings for foods, drinks, and liquid medicaments (i.e., tomato, soda, cinnamon, chocolate, and vanilla extract); it also may be associated with hand dermatitis.

Fragrance mix 1 (FM1) is a mixture of eight chemicals (geraniol, cinnamic aldehyde, hydroxycitronellal, cinnamic alcohol, eugenol, isoeugenol, oak moss absolute, and *a-amylcinnamic alcohol*) that is also used to screen for fragrance allergy. Since the products that incorporate these eight chemicals are similar to those that include BOP, the distribution of the dermatitis may be similar. In fact, some of the fragrances included in FM1 are constituents of BOP, which explains the cross-reactivity that may be seen between these allergens.

Colophony or rosin is a resin that is derived from the distillation products of pine and spruce trees. It is commonly used as an adhesive as well as in eyebrow wax, some cosmetics, and diapers (top-layer pad). There is cross-reactivity among colophony allergic patients with fragrance and BOP, as components of both colophony and BOP occur together in nature, and may be incorporated in fragrances.

Formaldehyde and formaldehyde-releasing preservatives (FRPs) are another group of allergens which may co-sensitize with fragrances due to similar product utilization patterns. Their widely effective antibacterial and antifungal properties have led to the FRPs use as disinfectants and preservatives in a number of products, such as lotions, shampoos, body washes, and even some medications (generic corticosteroid creams and permethrin cream). Many manufacturers have replaced formaldehyde with one of the FRPs in biocides and personal hygiene

products. These FRPs include: *quaternium-15*, diazolidinyl urea (Germall II), DMDM hydantoin (Glydant), imidazolidinyl urea (Germall), 2-bromo-2-nitropropane-1,3-diol (Bronopol), and tris nitromethane (Tris Nitro). Systemic exposure to formaldehyde is possible through inhalation of cigarette smoke or ingestion of certain foods that metabolize into formic acid (i.e., aspartame-containing foods). These sources of exposure have been reported to stimulate allergic reactions, with some patients improving through dietary avoidance.

Sorbitan sesquioleate is an important emulsifier, specifically a fatty acid ester that is widely used in pharmaceuticals, such as topical corticosteroids, as well as in cosmetics, ointments, creams, and lotions; it has recently been shown to be a relevant contact allergen in the pediatric population. Its association with corticosteroids makes this allergen particularly relevant to those with atopic dermatitis, who often require topical corticosteroids for treatment.

Propylene glycol is a preservative and “wetting agent” found in a wide variety of products, including pharmaceuticals, foods (e.g., “moist” cakes), cosmetics, and personal care products, making allergen avoidance particularly challenging.

Lanolin (wool wax alcohol) is an emollient used for skin barrier protection and repair. It is made from the sebaceous excretions found on sheep’s wool. Common sources are topical ointments, moisturizers, and lip balms. Lanolin allergy is difficult to diagnose clinically with “trial and error” product substitution; patch testing is essential.

Rubber Additives

The next category of allergens that commonly affect children is rubber accelerators, to which both *thiuram* and *carbamate* belong, in addition to mercaptobenzothiazole, mercapto mix, and diakylthioureas. These chemicals are major additives in most rubber products as they promote rubber’s transformation from a liquid to a solid state. Sources of exposure include athletic shoes (insoles and soles), elastic waistbands, socks, swimwear, toys, pacifiers, cosmetic applicators, and adhesives.

Another allergen utilized with diakylthioureas in neoprene and athletic gear foam is *p-tert-butyl-formaldehyde resin*. The two are commonly referred to as the “neoprene cement” allergens. PTBFR is not a rubber accelerator, rather it is an adhesive that is used in the manufacture of shoes and cars, in addition to being an important component of neoprene and sports gear.

Dye and Textile Allergens

Para-phenylenediamine (PPDA) is a colorless aromatic amine commonly known for its use in permanent hair dye. Para-phenylenediamine acts as a primary intermediate in hair dyes, is oxidized by hydrogen peroxide, and then polymerized to a color within the hair by a coupler such as resorcinol. Although PPDA derivatives are used in screening chemicals for black rubber allergy (e.g., isopropylparaphenylenediamine and related chemicals), PPDA itself is a poor detector of sensitization for black rubber allergy and is usually patch test negative.

More recently PPDA is being used in temporary tattoos, where it is mixed with natural henna to make “black henna.” Black henna tattoos may induce sensitization with severe bullous reactions and subsequent adverse reactions with hyper- and hypopigmentation and permanent scarring. While PPDA is the most commonly used “permanent hair dye” [limit permissible for hair dyes (<6%)], it has been detected in concentrations well above 15% in henna tattoo preparations. Due to this elevated concentration, adolescents that have become sensitized through “black-henna” tattoos at younger ages are at risk for unusually severe reactions to PPDA containing hair dyes. Potential for systemic reactions is also possible among PPDA-sensitized patients when exposed to cross-reactors, such as benzocaine, hydrochlorothiazide, and sulfonamide medications. Moreover, 25% of those allergic to PPDA can also be reactive to certain dark synthetic clothing, which may contain semi-permanent dyes.

Because of their sensitizing potential, *Disperse blue dyes 106 and 124* are often used to screen for textile dermatitis in pediatric patients. These partially water-soluble dyes easily leach out of fabrics onto the skin with normal wear and repeated washing. Allergic contact dermatitis in children has been reported from sensitization to aniline dyes found in clothes, undergarments, seatbelts, diapers, and eyeglass frames. In addition to patch testing with individual dyes, a swatch of the patient’s suspect garment may also be directly applied, as many colors can make up a hue.

Another cause of textile dermatitis is *formaldehyde*, which may also cause hand and systemic contact dermatitis. In textiles, formaldehyde is used in resins to create “permanent press” or “wrinkle-resistant” clothing, and is associated with dermatitis in regions where clothing rubs against the skin, i.e., body folds. These chemicals are also used in rayon and corduroy, and patients that develop contact allergy to formaldehyde resins in textiles have also developed diffuse nummular eczema or erythroderma due to secondary sensitization from quaternium-15, a FRP.

Treating ACD

The basis of therapy for ACD is the avoidance of the causative agent(s). Once allergens are confirmed and identified through patch testing, patients are educated on allergen substitution, avoidance, and removal from their environment. With these interventions, it may be possible to “cure” dermatitis with a sustained remission.

At times, patch testing fails to identify the inciting agents, especially if multiple chemicals are involved; and in some instances, avoidance alone may not completely clear the ACD. In these cases, topical and/or systemic therapies will be necessary as an adjuvant. Cool, wet compresses are particularly useful in providing symptomatic relief to acutely inflamed skin. In cases of hand dermatitis and in “unavoidable exposures,” such as toxicodendron exposure in a woodlands hiker on a trek or in the cases of aeroallergens, physical barrier creams can be utilized. With these, the patients apply the creams before and during the exposure in an effort to avoid absorption of the allergen.

First-line therapy is topical corticosteroids, which albeit effective, may have side effects with prolonged use. A word of caution is that corticosteroids themselves may be allergens or their vehicles may contain allergenic components. Thus, careful screening during patch testing and specific prescription of appropriate formulations is necessary. For atrophy-prone areas, such as the face and intertriginous areas, topical calcineurin inhibitors (TCIs) may need to be substituted. Also of note, ACD to the active ingredients pimecrolimus, and tacrolimus themselves, and to the benzyl alcohol component of pimecrolimus has been reported.

In particular cases, where dermatitis is severe or widespread, involves the mucous membranes, or continues to progress despite the use of topical agents, systemic therapies may be necessary. Oral corticosteroids, used at 1 mg/kg/day, can be effective for acute exacerbations or episodes of ACD. When dermatitis becomes severe and chronic, “steroid sparing” agents should be considered. These include ultraviolet light therapy, cyclosporine, mycophenolate mofetil, methotrexate, and azathioprine.

In conclusion, the most important first step in treating patients with patch test confirmed ACD is avoidance education. A number of resources are available to provide patients with easy-to-read facts on where their specific allergens are found and ways to avoid them. (<http://www.contactderm.org/>, <http://www.truetest.com/>, <http://www.chemotechnique.se/>). Furthermore, many physicians educate their patients on the substitution of safe alternatives by providing them detailed shopping list instructions formulated by software such as the new

Contact Allergen Management Program (CAMP), available to members of the American Contact Dermatitis Society at www.contactderm.org and the list of Alternatives for the 2007 NACDG Standard Screening Tray developed by the American Contact Alternative Group.

References

- ACDS Contact Allergen Management Program (CAMP) <http://www.contactderm.org/files/DOCUMENTLIBRARY/How%20CAMP%20Works01032010.pdf>. Accessed 23 Jan 2011
- Alberta L, Sweeney SM, Wiss K (2005) Diaper dye dermatitis. *Pediatrics* 116:e450–e452
- Angelini G, Foti C, Rigano L, Vena GA (1995) 3-Dimethylaminopropylamine: a key substance in contact allergy to cocamidopropylbetaine? *Contact Dermat* 32:96–99
- Argonne National Laboratory EVS. Cobalt. Human Health Fact Sheet. Argonne August 2005. <http://www.ead.anl.gov/pub/doc/cobalt.pdf>. Accessed 30 Oct 2008
- Arroyo MP (2003) Black henna tattoo reaction in a person with sulfonamide and benzocaine allergy. *J Am Acad Dermatol* 48(2):301–302
- Asarch A, Scheinman PL (2008) Sorbitan Sesquioleate, a common emulsifier in topical corticosteroids, is an important contact allergen. *Dermatitis* 19(6):323–327
- Baker RR (2006) The generation of formaldehyde in cigarettes: overview and recent experiments. *Food Chem Toxicol* 44(11):1799–1822
- Bardana EJ Jr, Montanaro A (1991) Formaldehyde: an analysis of its respiratory, cutaneous, and immunologic effects. *Ann Allergy* 66(6):441–452
- Barros MA, Baptista A, Correia TM et al (1991) Patch testing in children: a study of 562 school children. *Contact Dermat* 25:156–159
- Bashir SJ, Maibach HI (2006) Contact Urticaria Syndrome. In: Chew AL, Maibach HI (eds) *Irritant dermatitis*. Springer, Berlin, pp 63–70
- Batchelor RJ, Wilkinson SM (2006) Contact allergy to disperse dyes in plastic spectacle frames. *Contact Dermat* 54:66–67
- Beattie PE, Green C, Lowe G, Lewis-Jones MS (2006) Which children should we patch test? *Clin Exp Dermatol* 32:6–11
- Belsito D, Wilson DC, Warshaw E, Fowler J, Ehrlich A, Anderson B et al (2006) A prospective randomized clinical trial of 0.1% tacrolimus ointment in a model of chronic allergic contact dermatitis. *J Am Acad Dermatol* 55:40–46
- Boffa MJ, Wilkinson SM, Beck HM (1995) Screening for corticosteroid contact hypersensitivity. *Contact Dermat* 33:149–151
- Brancaccio RR, Brown LH, Chang YT, Fogelman JP, Mafong EA, Cohen DE (2002) Identification and quantification of paraphenylenediamine in a temporary black henna tattoo. *Am J Contact Dermat* 13:15–18
- Brant WT (ed) (1896) *The metallic alloys: A practical guide for the manufacture of all kinds of alloys, amalgams, and solders, used by metal-workers: Together with their chemical and physical properties and their application in the arts and the industries*. Henry Cary Baird & Co., London
- Breithaupt A, Jacob SE (2008) Thimerosal and the relevance of patch-test reactions in children. *Dermatitis* 19(5):275–277
- Bruckner AL, Weston WL, Morelli JG (2000) Does sensitization to contact allergens begin in infancy? *Pediatrics* 105:e3
- Camarasa JMG, Aspiolea F, Alomar A (1983) Patch tests to metals in childhood. *Contact Dermat* 9(2):157–158
- Castanedo-Tardan MP, Jacob SE (2008a) Potassium dichromate. *Dermatitis* 19(4):E24–E25
- Castanedo-Tardan MP, Jacob SE (2008b) Allergic contact dermatitis to sorbitan sesquioleate in children. *Contact Dermat* 58:171–172
- Chromates. http://www.aad.org/public/publications/pamphlets/skin_allergic.html. Accessed 31 Oct 2008
- Chung WH, Chang YC, Yang LJ, Hung SI, Lin JY et al (2002) Clinicopathologic features of skin reactions to temporary tattoos and analysis of possible causes. *Arch Dermatol* 138(1):88–92
- Cohen DE, Brancaccio R (1997) What is new in clinical research in contact dermatitis. *Dermatol Clin* 15:137–148
- Cohen DE, Heidary N (2004) Treatment of irritant and allergic contact dermatitis. *Dermatol Ther* 17:334–340
- Coopman S, Degreef H, Dooms-Goossens A (1989) Identification of cross-reaction patterns in allergic contact dermatitis from topical corticosteroids. *Br J Dermatol* 121:27–34
- Davis MD, el-Azhary RA, Farmer SA (2007) Results of patch testing to a corticosteroid series: a retrospective review of 1188 patients during 6 years at Mayo Clinic. *J Am Acad Dermatol* 56:921–927
- De Groot A (1992) Allergic contact dermatitis. In: Marks R (ed) *Eczema*. Martin Dunitz, London, pp 104–125
- de Groot AC, van der Walle HB, Weyland JW (1995) Contact allergy to cocamidopropyl betaine. *Contact Dermat* 33:419–422
- de Waard-van der Spek FB, Oranje AP (2008) Patch tests in children with suspected allergic contact dermatitis: a prospective study and review of the literature. *Dermatology* 218(2):119–125
- Dotterud LK, Falk ES (1994) Metal allergy in north Norwegian schoolchildren and its relationship with ear piercing and atopy. *Contact Dermat* 31:308–313
- Duarte I, Lazzarini R, Kobata CM (2003) Contact dermatitis in adolescents. *Am J Contact Dermat* 14(4):200–204
- Elsaie ML, Olasz E, Jacob SE (2008) Cytokines and Langerhans cells in allergic contact dermatitis. *G Ital Dermatol Venereol* 143(3):195–205
- European Parliament and Council Directive 94/27/EC, Annex 28 Nickel CAS No 7440-0-20 Einacs No 2311114 and its compounds. http://eur-lex.europa.eu/smartapi/cgi/sga_doc?smartapi!celexapi!prod!CELEXnumdoc&numdoc=31994L0027&model=guichett&lg=en. Accessed 30 Oct 2008
- Fernández Vozmediano JM, Armario Hita JC (2005) Allergic contact dermatitis in children. *J Eur Acad Dermatol Venereol* 19(1):42–46
- Fisher AA (1975) Childhood allergic contact dermatitis. *Cutis* 15:635
- Fisher AA (1995) Cosmetic dermatitis in childhood. *Cutis* 55:15–16
- Foti C, Bonamonte D, Mascolo G, Corcelli A, Lobasso S, Rigano L et al (2003) The role of 3-dimethylaminopropylamine and amidoamine in contact allergy to cocamidopropylbetaine. *Contact Dermat* 48:194–198
- Foti C, Bonifazi E, Casulli C, Bonamonte D, Conserva A, Angelini G (2005) Contact allergy to topical corticosteroids in children with atopic dermatitis. *Contact Dermat* 52:162–163
- Fowler JF, Fowler LM, Hunter JE (1997) Allergy to cocamidopropyl betaine may be due to amidoamine: a patch test and product use test study. *Contact Dermat* 37:276–281
- Fowler JF Jr, Zug KM, Taylor JS, Storrs FJ, Sherertz EA, Sasseville DA et al (2004) Allergy to cocamidopropyl betaine and amidoamine in North America. *Dermatitis* 15:5–6
- Freeman S, Stephens R (1999) Cheilitis: analysis of 75 cases referred to a contact dermatitis clinic. *Am J Contact Dermat* 10:198–200
- Friedlander SF (1998) Consultation with the specialist: Contact dermatitis. *Pediatr Rev* 19(5):166–171
- Geldof BA, Roesyanto ID, vanJoost TH (1989) Clinical aspects of para-tertiary-butylphenolformaldehyde resin. *Contact Dermat* 21:312–315

- Gelpi CB, Jacob SE (2008) Instructions for educating patients on ROAT testing in conjunction with patch testing. *Dermatol Nurs* 20(2):139, 143
- Giordano-Labadie F, Rance F, Pellegrin F, Bazek J, Dutau G, Schwarze HP (1999) Frequency of contact allergy in children with atopic dermatitis: results of a prospective study of 137 cases. *Contact Dermat* 40:192–195
- Giusti F, Massone F, Bertoni L, Pellacani G, Seidenari S (2003) Contact sensitization to disperse dyes in children. *Pediatr Dermatol* 20:393–397
- Guin JD (2001) Seat-belt dermatitis from disperse blue dyes. *Contact Dermat* 44:263
- Hammonds LM, Hall VC, Yiannias JA (2009) Allergic contact dermatitis in 136 children patch tested between 2000 and 2006. *Int J Dermatol* 48(3):271–274
- Hara M, Ikezawa A (1988) Neonatal contact dermatitis. *Contact Dermat* 18:105
- Hasan T, Rantanen T, Alanko K, Harvima RJ, Jolanki R, Kalimo K et al (2005) Patch test reactions to cosmetic allergens in 1995–1997 and 2000–2002 in Finland—a multicentre study. *Contact Dermat* 53: 40–45
- Heine G, Schnuch A, Uter W, Worm M (2004) Frequency of contact allergy in German children and adolescents patch tested between 1995 and 2002: results from the Information Network of Departments of Dermatology and the German Contact Dermatitis Research Group. *Contact Dermat* 51:111–117
- Hengge UR, Ruzicka T, Schwartz RA, Cork MJ (2006) Adverse effects of topical glucocorticosteroids. *J Am Acad Dermatol* 54:1–15, quiz 6–8
- Hill AM, Belsito DV (2003) Systemic contact dermatitis of the eyelids caused by formaldehyde derived from aspartame? *Contact Dermat* 49(5):258–259
- Hogan PA, Weston WL (1993) Allergic contact dermatitis in children. *Pediatr Rev* 14(6):240–243
- Hogeling M, Pratt M (2008) Allergic contact dermatitis in children: The Ottawa hospital patch-testing clinic experience, 1996 to 2006. *Dermatitis* 19(2):86–89
- Holden CR, Gawkrödger DJ (2005) 10 years' experience of patch testing with a shoe series in 230 patients: which allergens are important? *Contact Dermat* 53:37–39
- Hunter JE, Fowler JF (1998) Safety to human skin of cocamidopropyl betaine: a mild surfactant for personal-care products. *J surfactants Detergents* 1(2):235–239
- Isaksson M, Bruze M, Lepoittevin JP, Goossens A (2001) Patch testing with serial dilutions of budesonide, its R and S diastereomers, and potentially cross-reacting substances. *Am J Contact Dermat* 12: 170–176
- Jacob SE (2007) Avoid the shriek with shrek: video-distraction assist for pediatric patch testing. *Dermatitis* 18(3):179–180
- Jacob SE, Castanedo-Tardan MP (2007) Pharmacotherapy for allergic contact dermatitis. *Expert Opin Pharmacother* 8(16):2757–2774
- Jacob SE, Stechschulte S (2008a) Formaldehyde, aspartame and migraines: a possible connection. *Dermatitis* 19(3):E10–E11
- Jacob SE, Stechschulte S (2008b) Eyelid dermatitis associated with balsam of Peru constituents: benzoic acid and benzyl alcohol. *Contact Dermat* 58(2):111–112
- Jacob SE, Steele T, Rodriguez G (2005) Focus on T.R.U.E. test allergens #21, 13, and 18: formaldehyde and formaldehyde releasing preservatives. *Skin Aging* 13(12):22–27
- Jacob SE, Burk CJ, Connelly EA (2008a) Patch testing: Another steroid-speaking agent to consider in children. *Pediatr Dermatol* 25(1):81–87
- Jacob SE, Zapolanski T, Chayavichitsilp P, Connelly EA, Eichenfield LF (2008b) p-Phenylenediamine in black henna tattoos: a practice in need of policy in children. *Arch Pediatr Adolesc Med* 162(8):790–792
- Jacob SE, Brod B, Crawford GH (2008c) Clinically relevant patch test reactions in children - a United States based study. *Pediatr Dermatol* 25(5):520–527
- Jacob SE, Yang A, Herro EM, Zhang C (2010) Contact allergens in a pediatric population: association with atopic dermatitis and comparison with other North American referral centers. *J Clin Aesthet Dermatol* 3(10):29–35
- Janeway C, Travers P, Walport M, Shlomchik M (2005) *Immunobiology: The immune system in health and disease*, 6th edn. Garland Science, New York
- Jensen CD, Paulsen E, Andersen KE (2006) Retrospective evaluation of the consequence of alleged patch test sensitization. *Contact Dermat* 55(1):30–35
- Kohl L, Blondeel A, Song M (2002) Allergic contact dermatitis from cosmetics. *Dermatology* 204(4):334–337
- Krafchik BR, Halbert A, Yamamoto K, Sasaki R (2003) Eczematous Dermatitis. In: Schachner LA, Hansen RC (eds) *Pediatric Dermatology*, 3rd edn. Mosby, Edinburgh
- Kutting B, Brehler R, Traupe H (2004) Allergic contact dermatitis in children: strategies of prevention and risk management. *Eur J Dermatol* 14:80–85
- Lacahapelle JM, Maibach HI (2003) Patch testing, prick testing – A practical guide. Springer, Berlin
- Laeijendecker R, van Joost T (1994) Oral manifestations of gold allergy. *J Am Acad Dermatol* 30(2 Pt 1):205–209
- Lewis VJ, Statham BN, Chowdhury MMU (2004) Allergic contact dermatitis in 191 consecutively patch tested children. *Contact Dermat* 51:155–156
- Leysat SD, Boone M, Blondeel A, Song M (2003) Two cases of cross-sensitivity in subjects allergic to paraphenylenediamine following ingestion of polaronil. *Dermatology* 206:379–380
- Livideanu C, Giordano-Labadie F, Paul C (2007) Cellular phone addiction and allergic contact dermatitis to nickel. *Contact Dermat* 57:130–131
- Magnusson B, Wilkinson DS (1975) Cinnamic aldehyde in toothpaste. *Contact Dermat* 1:70–76
- Mark BJ, Slavin RG (2006) Allergic contact dermatitis. *Med Clin North Am* 90(1):169–185
- Marks R (1992) Adverse side effects from the use of topical corticosteroids. In: Maibach HI, Surger C (eds) *Topical corticosteroids*. Basel, Karger, pp 170–183
- Marks JG Jr, Elsner P, DeLeo VA (2002) *Standard allergens and contact occupational dermatology*, 3rd edn. Mosby, Philadelphia, pp 65–139
- Menezes de Padua CA, Schnuch A, Lessmann H, Geier J, Pfahlberg A, Uter W (2005) Contact allergy to neomycin sulfate: results of a multifactorial analysis. *Pharmacoepidemiol Drug Saf* 14(10): 725–733
- Militello G, Jacob SE, Crawford GH (2006) Allergic contact dermatitis in children. *Curr Opin Pediatr* 18(4):385–390
- Mimesh S, Pratt M (2006) Allergic contact dermatitis from corticosteroids: reproducibility of patch testing and correlation with intradermal testing. *Dermatitis* 17:137–142
- Mortz C, Andersen KE (1999) Allergic contact dermatitis in children and adolescents. *Contact Dermat* 41:121–130
- Mortz CG, Lauritsen JM, Binslev-Jensen C et al (2002) Contact allergy and allergic contact dermatitis in adolescents: prevalence measures and associations. The Odense adolescence cohort study on atopic dermatitis and dermatitis (TOACS). *Acta Derm Venereol* 82: 352–358
- Mydlarski PR, Katz AM, Mamelak AJ et al (2003) *Contact Dermatitis*. In: Adkinson NF, Yunginger JW, Busse WW et al (eds) *Middleton's allergy principles and practice*. Mosby, Philadelphia, pp 1581–1593

- Nakamura M, Arima Y, Nobuhara S, Miyachi Y (1999) Nickel allergy in a trumpet player. *Contact Dermat* 40:219–220
- Neri I, Guareschi E, Savoia F, Patrizi A (2002) Childhood allergic contact dermatitis from henna tattoo. *Pediatr Dermatol* 19(6):503–505
- Nguyen SH, Dang TP, MacPherson C, Maibach HI (2008) Prevalence of patch test results from 1970 to 2002 in a multi-centre population in North America. *Contact Dermat* 58:101–106
- Nickel test kit. <http://www.bgiusa.com/ih/nickel.htm>. Accessed 30 Oct 2008
- Paraphenylenediamine. http://www.aad.org/public/publications/pam-phlets/skin_allergic.html. Accessed 31 Oct 2008
- Pratt M, Taraska V (2000) 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* 11:30–41
- Pratt MD, Belsito DV, DeLeo VA, Fowler JF Jr, Fransway AF, Maibach HI et al (2004) North American Contact Dermatitis Group patch-test results, 2001–2002 study period. *Dermatitis* 15:176–183
- Rajagopalan R, Anderson R (1997) Impact of patch testing on dermatology-specific quality of life in patients with allergic contact dermatitis. *Am J Contact Dermat* 8(4):215–221
- Riemann H, Schwarz T, Grabbe S (2003) Pathomechanisms of the elicitation phase of allergic contact dermatitis. *J Dtsch Dermatol Ges* 1(8):613–619
- Rietschel RL, Rosenthal LE, NACDG (1990) Standard patch test screening series used diagnostically in young and elderly patients. *Am J Contact Derm* 1(1):53–55
- Rietschel RL, Warshaw EM, Sasseville D, Fowler JF, DeLeo VA, Belsito DV, Taylor JS, Storrs FJ, Mathias CG, Maibach HI, Marks JG, Zug KA, Pratt M, North American Contact Dermatitis Group (2007) Common contact allergens associated with eyelid dermatitis: data from the North American Contact Dermatitis Group 2003–2004 study period. *Dermatitis* 18(2):78–81
- Romaguera C, Villaplana J (1998) Contact dermatitis in children: 6 years experience (1992–1997). *Contact Dermat* 39:277–280
- Roul S, Ducombs G, Taieb A (1999) Usefulness of the European standard series for patch testing in children. A 3-year single-centre study of 337 patients. *Contact Dermat* 40:232–235
- Saint-Mezard P, Rosieres A, Krasteva M, Berard F, Dubois B, Kaiserlian D, Nicolas JF (2004) Allergic contact dermatitis. *Eur J Dermatol* 14:284–295
- Saitta P, Brancaccio R (2007) Allergic contact dermatitis to pimecrolimus. *Contact Dermat* 56(1):43–44
- Salam TN, Fowler JF Jr (2001) Balsam-related systemic contact dermatitis. *J Am Acad Dermatol* 45:377–381
- Sasseville D (2004) Hypersensitivity to preservatives. *Dermatol Ther* 17(3):251–263
- Scherman A, Jacob S, Zirwas M, Warshaw E, Nedorost S, Katta R, Cook J, Castanedo-Tardan MP (2008) Contact Allergy: alternatives for the 2007 North American contact dermatitis group (NACDG) Standard Screening Tray. *Dis Mon* 54(1–2):7–156
- Seidenari S, Giusti F, Pepe P, Mantovani L (2005) Contact sensitization in 1094 children undergoing patch testing over a 7-year period. *Pediatr Dermatol* 22(1):1–5
- Shaw DW, Maibach HI, Eichenfield LF (2007) Allergic contact dermatitis from pimecrolimus in a patient with tacrolimus allergy. *J Am Acad Dermatol* 56(2):342–345
- Sidbury R, Hanifin JM (2000) Systemic therapy of atopic dermatitis. *Clin Exp Dermatol* 25:559–566
- Sosted H, Johansen JD, Andersen KE et al (2006) Severe allergic hair dye reactions in 8 children. *Contact Dermat* 54:87–91
- Spann CT, Tutrone WD, Weinberg JM, Scheinfeld N, Ross B (2003) Topical antibacterial agents for wound care: a primer. *Dermatol Surg* 29(6):620–626
- Strauss RM, Orton DI (2003) Allergic contact cheilitis in the United Kingdom: a retrospective study. *Am J Contact Dermat* 14:75–77
- Tomar J, Jain VK, Aggarwal K, Dayal S, Gupta S (2005) Contact allergies to cosmetics: testing with 52 cosmetic ingredients and personal products. *J Dermatol* 32:951–955
- Trocho C, Pardo R, Rafecas I et al (1998) Formaldehyde derived from dietary aspartame binds to tissue components in vivo. *Life Sci* 63(5):337–349
- Veien NK, Hattel T, Justesen O, Nørholm N (1985) Oral challenge with balsam of Peru. *Contact Dermat* 12(2):104–107
- Wakelin SH, Smith H, White IR, Rycroft RJ, McFadden JP (2001) A retrospective analysis of contact allergy to lanolin. *Br J Dermatol* 145:28–31
- Warshaw EM, Schram SE, Belsito DV, DeLeo VA, Fowler JF Jr, Maibach HI et al (2007) Shoe allergens: retrospective analysis of cross-sectional data from the North American contact dermatitis group, 2001–2004. *Dermatitis* 18:191–202
- Weston WL, Weston JA (1984) Allergic contact dermatitis in children. *Am J Dis Child* 138(10):932–936
- Weston WL, Weston JA, Kinoshita J et al (1986) Prevalence of positive epicutaneous tests among infants, children, and adolescence. *Pediatrics* 78:1070–1074
- Wilkinson SM (2000) Corticosteroid cross-reactions: an alternative view. *Contact Dermat* 42:59–63
- Wöhrl S, Hemmer W, Focke M et al (2001) The significance of fragrance mix, balsam of Peru, colophony and propolis as screening tools in the detection of fragrance allergy. *Br J Dermatol* 145:268–273
- Wöhrl S, Hemmer W, Focke M, Götz M, Jarisch R (2003) Patch testing in children, adults, and the elderly: influence of age and sex on sensitization patterns. *Pediatr Dermatol* 20(2):119–123
- Wöhrl S, Jandl T, Stingl G, Kinaciyan T (2007) Mobile telephone as new source for nickel dermatitis. *Contact Dermat* 56:113
- Wollina U (2007) The role of topical calcineurin inhibitors for skin diseases other than atopic dermatitis. *Am J Clin Dermatol* 8(3):157–173
- Worm M, Aberer W, Agathos M, Becker D, Brasch J, Fuchs T, Hillen U, Hoger P, Mahler V, Schnuch A, Szliska C, German Contact Dermatitis Research Group (DKG) (2007) Patch testing in children- Recommendations of the German Contact Dermatitis Research Group (DKG). *J Dtsch Dermatol Ges* 5(2):107–109
- Zug KA, McGinley-Smith D, Warshaw EM et al (2008) Contact allergy in children referred for patch testing North American Contact Dermatitis Group Data, 2001–2004. *Arch Dermatol* 144(10):1329–1336