Leaflets Three, Let It Be,…

Poison Ivy and Friends
Leaflets Three, Let It Be…

• Common throughout most of the US
• Contact frequently sends people to hospitals, some by ambulance
• 50% of US has allergy
• Estimated that 50% of Worker’s Comp cases in California are due to contact
• Hypersensitive people may react to material made airborne by walking near plants
• 1 nanogram exposure can cause reaction
Hiding Places

Where It Grows:

- Rydberg’s Poison Ivy
- Western Poison Oak
- Eastern Poison Oak
- Poison Ivy
- Poison Sumac
Leaflets Three, Let It Be…

• Old folk rhyme warns
  “Leaves of three, let it be;
   berries white, run in fright”

• Members of the Poison Ivy (*Toxicodendron*) family have three distinct leaves on a single stem coming off a larger main stem
  – *Exception is poison sumac with 7-13 leaves*

• Leaves can be green in summer and red in fall, may have yellow or green flowers and white berries depending on season
The Villain’s Pictures

Poison Ivy  Poison Oak  Poison Sumac

Toxicodendron radicans  Toxicodendron toxicarium  Toxicodendron vernix
What Happens

- Plant contact gets urushiol oil on skin
  - Could get from petting dog with oil on fur
- Oil can be spread by hand
- Usually 24-48 hours to react
  - can occur in as little as eight
- Most cases have localized contact rash
  - Itching with small raised red areas & blisters
  - May develop over a week and last 3-4 weeks
What Happens

• Significant exposure can be serious
• Lungs react to smoke from burned plants
• Mowing near plants can kick up clouds of oil and contact entire skin surface
• Contact with eyes or genitals can cause severe swelling
Treatment

• Don’t get exposed (wear long pants and sleeves)
  • Kill plants with herbicides
    Oil remains even when plant is dead
• Wash skin within 2-hours with regular soap or 8-hours with special soaps (Oak-n-Ivy or TECNU)
• Scrub fingernails
• OTC Cortisone creams if reaction minor
• Prescription meds if reaction severe
  • Wash all clothes (use latex/rubber gloves)
    – Use hot water and detergent
Leaves of Three, Let Them Be: If Only It Were That Easy

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Introduction

Poison ivy (Toxicodendron radican), eastern poison oak (Toxicodendron quercifolium), western poison oak (Toxicodendron diversilobum), and poison sumac (Toxicodendron vernix) are species of the Anacardiaceae family, Rhus genus. They are indigenous to the United States and found throughout the United States, northern Mexico, and southern Canada, each favoring certain locations and climates (Guin, Gillis, & Beaman, 1981; Tanner, 2000). Contact with the plant oil, urushiol, found not only in the leaves of the plant but also in the stems and roots, results in an allergic contact dermatitis (ACD) in 50%-70% of people (Epstein, 1994). Humans of all races and skin color are susceptible and uniquely sensitive among animals to poison ivy, oak, and sumac (Rietschel & Fowler, 2001b). Approximately 50% of people are highly or moderately clinically sensitive to urushiol, and another 30%-40% will only react if they have contact with a large amount of the plant oil (Epstein). Unfortunately, direct contact with the plant is not required for transmission of the urushiol oil. The oil can remain on clothing, pets, garden tools, and sports equipment for long periods of time in dry climates (Rietschel & Fowler, 2001a), resulting in ACD.

Although other plants are known to cause dermatitis, some by irritant contact (i.e., cacti), immediate contact urticaria (i.e., stinging nettles), or phototoxicity (i.e., bavachee seeds, rue, and in some people, parsnip, parsley, and celery), the Toxicodendron plants are by far the most common cause of ACD (Juckett, 1996). Other plant members of the Anacardiaceae family may cause ACD in people with established sensitivity to urushiol; the cashew nut husk, mango peels and leaves, Japanese lacquer tree oils, and leaves and seed coverings from the ginkgo tree may all cause a similar rash to poison ivy when handled by sensitized people (Juckett, 1996).

Pathophysiology of ACD in Children and Adolescents

ACD is a cell-mediated response to exposure to an antigenic substance as compared to irritant contact dermatitis, the usual cause of the common diaper rash, which is a nonimmunologic response to a skin irritant resulting in disturbance of cell hydration (Peate, 2002). ACD requires an intact cellular immune system and an antigen with low molecular weight that can penetrate the epidermis (Weston & Bruckner, 2000). Most contact allergens are weak requiring repeated exposures to establish an immune response, but poison ivy, oak, or sumac are strong antigens requiring only two exposures to establish sensitization in susceptible people (Weston & Bruckner, 2000).

The first exposure is called the sensitization phase. The antigen has contact with the skin penetrating the epidermal layer and attaches to Langerhans’ cells. The Langerhans’ cells migrate to regional lymph nodes where they have contact with the T-lymphocytes. The T-lymphocytes develop recognition of the antigen, experience cytokine-induced proliferation and clonal expansion within the lymph nodes, then are released into the bloodstream and circulated throughout the body, including the skin (Weston & Bruckner, 2000).

The second phase, elicitation, occurs when a sensitized individual is again in contact with the antigen. The antigen-bearing Langerhans’ cells interact with the antigen-specific T-lymphocytes resulting in a proliferation of
the antigen-specific T-lymphocytes and the release of inflammatory mediators. An allergic response usually occurs within 24-48 hours of exposure and will last for 2-4 weeks if unmitigated by therapy (Weston & Bruckner, 2000).

Although exposure to many potential allergens occurs early in infancy, sensitization develops in only about 20%-25% of children before 5 years of age (Bruckner, Weston, & Morelli, 2000; Fisher, 1994; Hogan & Weston, 1993, 1997). It was previously believed that allergic contact dermatitis was rare in children due to infrequent exposure to allergens and a less mature immune system. Sensitization to plants of the Rhus genus has been experimentally induced in infants showing that sensitization to this strong antigen is possible during infancy (Epstein, 1961). Lack of clinical experience with this type of ACD in infancy is probably determined more by infrequent exposure of infants to poison ivy, oak, or sumac than to their inability to develop an allergic response. In general, it is believed that contact allergy and ACD is rare in infants and young children and becomes more frequent with increasing age, reaching adult sensitization patterns after the first decade of life (Mortz & Andersen, 1999).

Sensitivity to urushiol, as with most allergens, runs in families with parental sensitization being a strong predictor of potential sensitization in children. The exact sensitization patterns have been found in identical twin children (Thestrup-Pederson, 1997).

**Clinical Manifestations of ACD to Poison Ivy, Oak, and Sumac**

ACD presents with an erythematous, papulovesicular rash at the site of contact that is intensely pruritic (Rietschel & Fowler, 2001b; Tanner, 2000; Weston & Bruckner, 2000). The cell-mediated response usually occurs within 24-48 hours. Erythematous papular lesions that are intensely itchy appear first (see Figure 1). Within 2-3 days vesicular lesions develop and quickly erupt releasing plasma that forms a crust (see Figure 2). Vesicles are often numerous and small, but bullae can occur in severe reactions and "weeping" lesions are not uncommon (Tanner, 2000) (see Figure 3). The rash may grow in size and development of new vesicles can occur during the first 2 weeks due to the allergenic response of the host without additional contact with urushiol leading to the common belief that the serum from vesicles is allergenic. The serum released from the vesicles is not antigenic and does not spread the allergic contact reaction.

![Figure 1. Erythematous Papular Lesions of Poison Ivy. Printed with permission from Poison Ivy, Oak, and Sumac Information Center, www.poisonivy.us](image-url)
Lesions vary greatly in intensity due to the extent of exposure, the thickness of the skin, the sensitivity of the individual, and the allergenicity of the urushiol (Gartner, Wassed, Rodrigues, et al., 1993; Tanner, 2000). Lesions can occur on skin locations without direct contact to urushiol due to the nonspecific effect of stimulated,
antigen-specific circulating T-lymphocytes, occasionally resulting in numerous widespread edematous red papules (Weston & Bruckner, 2000). Frequently, allergic reactions occur on skin surfaces not directly exposed to plants, such as the inner thighs, abdomen, or genitals due to contact with urushiol on hands, clothing, pets, or garden, sports, or camping equipment. Secondary contact is often the reason for the development of new lesions when there is no further known contact with the plant (i.e., lesions appearing a week after a camping trip as a result of contact with urushiol on camping gear).

Pruritus is intense in all phases of the lesions and is a hallmark characteristic of poison ivy, oak, and sumac allergic dermatitis. The severe itching can lead to secondary skin damage and infection but the primary lesions usually dry and clear without hyperpigmentation in 3 weeks.

The distribution pattern of skin lesions often provides clues as to the cause of the lesions. Contact with poison ivy, oak, or sumac often results in papular vesicular lesions in an asymmetrical linear pattern on exposed body parts (i.e., arms, legs, and face). A history of activity and possible exposure in wooded or non-cultivated areas prior to eruption of the skin lesions is a strong clue as is a positive history of previous reaction to these plants. A negative history of reaction to the Rhus genus does not rule out current reaction. Smoke from fires burning Toxicodendron may have particulate matter containing urushiol oil causing wide spread skin contact and reaction or systemic absorption (Tanner, 2000). Urushiol can also become airborne when plants are cut with weed-whackers or lawn mowers. Ingested plants do not cause mucous lesions but can result in gastrointestinal symptoms including perianal itching (Rietschel & Fowler, 2001a).

### Differential Diagnosis

ACD can be so inflamed with large areas of redness and edema that it may mimic cellulites, but there will not be a history of fever or leukocytosis on complete blood count. The vesicular lesions and crusting may be similar in appearance to herpes simplex and impetigo, but neither of these have the intense itching associated with ACD. Other forms of dermatitis, such as atopic dermatitis, have symmetrical patterns of distribution, and this is usually not found with poison ivy, oak, and sumac. Interestingly, studies have generally not found a higher incidence of ACD sensitivity in children with atopic dermatitis (McAlvany & Sheretz, 1994).

### Complications

Lesions from poison ivy, oak, or sumac usually resolve within 3-4 weeks. Hyperpigmentation and or lichenification of skin lesions may occur in darkly pigmented individuals but is usually not found in lighter skinned people (Rietschel & Fowler, 2001b). The incidence of complications is low, but secondary skin infections can occur. Brook, Frazier and Yeager (2000) found a wide variety of anaerobic and aerobic bacteria as causative agents in secondary infections. The dermatitis may be accompanied by regional lymphadenitis during the acute phase (Weston, 1997). Nephropathy has been reported and is thought to be secondary to complications of a streptococcal skin infection (Fisher, 1996b). Erythema multiforme associated with poison ivy has also been reported (Cohen & Cohen, 1998).

### Treatment

#### Prevention

Prevention of contact with the Toxicodendron group of plants is the primary means of treating a child with a known sensitivity to these plants. Unfortunately, this is often difficult. The old adage “leaves of three, let them be” is only partially true. Poison ivy leaves usually come in threes but not always, are usually notched but can have smooth edges, can be 3-15 cm in length, and grow on hairy-stemmed vines or as low shrubs (see Figures 4a & b). The leaves turn bright colors in the autumn (see Figures 5a & b) and the stems have clusters of whitish-green berries. Poison ivy is most often found east of the Rocky Mountains. Poison oak is found west of the Rocky Mountains. Its leaves are 3-7 cm in length with lobulated notched edges in groups of 3, 5, or 7 leaves on small bush-like plants or vines (see Figure 6). It produces whitish flowers from late summer through fall and the leaves also turn bright colors in autumn. Poison sumac prefers boggy areas, especially in the south. The leaves are approximately 10 cm long, oval, and in clusters of 7-13 leaves angled upward on one stem of a tall shrub or small tree (see Figure 7) (Guin, Gillis, & Beaman, 1981; Tanner, 2000). All forms of Toxicodendrons produce small
clusters of greenish white berries, also allergenic, along their stems in the autumn. Children cannot be expected to identify all the various forms of the Toxicodendron group but may be taught the most common forms in their immediate environment.

**Figure 4a.** Poison Ivy Leaves
Figure 4b. Poison Ivy Vine Growing on Tree
Figure 5. Poison Ivy Leaves in Autumn

Figure 6. Poison Oak. Printed from Agriculture and Agri-Food Canada - www.agr.gc.ca
Removal of plants including the root systems, in areas frequented by children, is the best way to reduce the incidence of contact and allergic dermatitis. Vines should be severed at the root source and the roots removed if possible. Dried roots and leaves are still allergenic. Herbicides (amitrole, silvex, ammonium sulfamate, or 2,4-D acetic acid) can be used for control of large areas of poison ivy, oak, or sumac but are not species-specific and have the potential of killing surrounding plants or coming in contact with children or pets (Rietschel & Fowler, 2001a). Children and pets should not be in the area of application while the herbicide is still wet, and the herbicide should not be applied if there is any wind. The greatest danger of poisoning, however, occurs because of careless handling of gloves, shoes, clothing, or equipment used to apply the herbicide or because of unsafe storage or disposal of these chemicals (Rietschel & Fowler, 2001a).

When contact with poison ivy, oak, or sumac is anticipated, protective clothing (i.e., long sleeve shirts, long pants, socks and shoes) should be worn to cover arms and legs. The catechols in urushiol are soluble in rubber, therefore rubber gloves and rubber boots are not protective (Rietschel & Fowler, 2001a). Heavy-duty vinyl gloves are recommended when actively trying to remove these plants. Hands and skin that comes in contact with exterior surfaces of clothing with possible urushiol oil on it should be washed immediately with soap and water. If done immediately ACD can be prevented, but if even 10 minutes elapse with the oil on the skin only 50% will be removed (Fisher, 1996a). The oil residue on clothes, pets, garden and sports equipment, or toys can be removed with liberal soap and water washing.

Since skin area of contact often cannot be washed immediately with soap and water, attempts have been made to identify solvents or compounds that could be used hours after contact to leach the oil from the skin and prevent an allergic reaction. A study done by Stibich, Yagan, Sharma, Herndon, and Montgomery (2000) compared the effectiveness of three agents to prevent or reduce allergic reactivity in volunteers exposed to urushiol for 2-8 hours: Tecnu®, an over-the-counter product stated to "prevent/reduce poison ivy dermatitis up to eight hours post-exposure;" Goop®, an oil-removal product used to remove automobile grease; and Dial® Ultra, a commercial dishwashing soap. The study found 70%, 61.8%, and 56.4% effectiveness respectively in preventing erythema, vesiculation, and induration in positive responders to the allergen. Due to the small number of responders in the study (only 9 out of 20 volunteers) the differences in product effectiveness was not significant,
but all did decrease the occurrence of allergic dermatitis. There was a significant cost difference between agents with Tecnu costing $1.25 per ounce, while Goop and Dial cost only $0.07 cents per ounce (Stibich et al., 2000). None of these compounds prevented ACD in all individuals; 30% of the study subjects developed a reaction in spite of washing the exposed skin areas.

Barrier preparations have been tried with varying success as a preventative measure when there is anticipated contact with poison ivy, oak, or sumac (i.e., hiking, camping, or working in areas with known Toxicodendron infestation) (Grevelink, Murrell, & Olsen, 1992). Marks and colleagues (1995) found that 144 test subjects with positive reactions to urushiol, if pretreated with a 5% quaternium-18 bentonite lotion, an organoclay substance commonly used in cosmetics, had absent or significantly reduced reactions (p < 0.0001). The mechanism of action is unknown, but 5% quaternium-18 bentonite lotion (Enviromed Pharmaceuticals, trade name Ivy Block®) leaves a visible film on the skin and may interfere with absorption of urushiol by physically blocking skin contact. Ivy Block is the only FDA-approved preventive treatment for poison ivy, oak, and sumac ACD (www.enviroderm.com, 6-28-03).

There is no U.S. Food and Drug Administration approved and reliable oral regimen for desensitization to urushiol. Attempts have been made in the past to develop tolerance through oral ingestion of dilutions of poison ivy and poison oak extract, but these were unsuccessful in developing long-term tolerance and resulted in many side effects such as pruritus ani, hives, blisters, or dermatitis (Epstein, 1994; Tanner, 2000).

Topical Treatment for Lesions

Treatment for lesions caused by contact with poison ivy, oak, or sumac varies by the extent and severity of the lesions and the need for comfort or symptom relief. Treatment does not alter the duration of symptoms, however (Tanner, 2000). Topical lotions such as Calamine®, Ivy Rest®, Ivy Sooth®, can reduce itching and dry oozing lesions. The active ingredients in most topical lotions for ACD contain menthol, camphor, or phenol to sooth the skin and low potency hydrocortisone (Tanner, 2000). Topical antihistamines, benzocaine, and neomycin sulfate are no longer recommended because of possibility of sensitization to these agents (Williford & Sheretz, 1994). Tepid baths, oatmeal baths, cool compresses with astringents such as Burrow's® or Domeboro® can decrease pruritus. Aloe, calendula, Jewell Weed, and other emollients are frequently used herbal preparations that can be soothing to the skin, but no clinical trials are reported in the medical literature (Gardiner, Coles, & Kemper, 2001). Blisters can be opened to allow release of serum and enable topical preparations to have contact with the underlying skin. Caretakers should be reassured the fluid draining from the blister will not spread the lesions to other parts of the child's body or to other individuals. Occlusive dressings should not be used to cover lesions but a light gauze dressing can be used when lesions are weeping.

Topical glucocorticoids are frequently used, but low dose preparations are of little use in controlling symptoms and do not shorten the course of the condition. If less than 10% of the skin is involved, topical, moderate-to-high potency glucocorticoids once or twice a day are recommended except for use on the face or genitals, which should only be treated with low dose glucocorticoids (Lee & Arriola, 1999; Raimer, 2001; Tanner, 2000; Weston & Bruckner, 2000; Williford & Sheretz, 1994) (see Table 1). If high dose topical steroids are being used, abrupt discontinuation may result in rebound inflammation (Williford & Sheretz, 1994).

Systemic Treatment

For severe symptoms of allergic dermatitis caused by poison ivy, oak, or sumac, systemic medication may be necessary. Pruritus can be severe and many children cannot heed the directions to not scratch. Antihistamines given orally, especially at night, can be helpful in both mild and severe cases. Diphenhydramine (Benedryl®), chlorpheniramine (Chlor-Trimeton®), or hydroxyzine hydrochloride (Atarax®, Vistaril®) dosed by weight three to four times a day will help relieve itching (Brodell & Williams, 1999). Dosing this frequently can make the child drowsy and unable to function well in school, and the dosage may need to be decreased and topical soothing agents used liberally during the day. Dressing the child in cotton clothing that covers the lesions and keeping hands clean and fingernails short may help reduce secondary trauma to the skin.

If greater than 10% of the skin is affected, then systemic glucocorticoids are often used in conjunction with
topical glucocorticoids, but individual assessment of the child for possible risks due to systemic steroid use must be made (Weston & Bruckner, 2000). The usual recommended treatment is prednisone 1 mg/kg/day as a single morning dose for 7-10 days followed by dose tapering for an additional 7-10 days (Bruckner & Weston, 2001). It is necessary to taper the prednisone over a 1-2 week period to avoid repeated flares of the lesions.

Occasionally, ACD will become secondarily infected requiring systemic antibiotics. The application of topical steroids or oral steroids may mask the signs of inflammation associated with secondary infection so lesions will need to be examined closely for indication of infection (i.e., pus verses translucent serum discharge, pain or tenderness, increasing inflammation and redness, increasing warmth, or fever). Research indicates multiple organisms can cause secondary infections with poison ivy, oak, and sumac so a broad-spectrum antibiotic that covers aerobic and anaerobic bacteria, such as amoxicillin/clavulanate, second generation cephalosporins, or azithromycin is recommended (Brook, 2002; Brook et al., 2000).

Conclusion

Poison ivy, poison oak, and poison sumac are common plants in many regions of the United States and Canada. Pediatric nurses will frequently care for children with allergic reactions to these plants. Caretakers and affected children need to understand the allergic nature of the lesions, their probable life-time sensitivity, how to manage the symptoms associated with the allergy, the expected duration and characteristics of the lesions, and how to effectively use over-the-counter preparation or prescribed preparations for symptom relief (see Table 2). Once the symptoms have been treated, the child and family should be given information on plant identification, common mechanisms for contact with the allergenic urushiol oil, and methods of preventing or reducing that contact. Parents and child caretakers should also be told that even though a child has not shown reactivity to these plants in the past, avoidance is recommended because sensitization may have occurred. Parents, caretakers, and camp or recreational leaders should be encouraged to keep children away from these plants whenever possible and to make a concerted effort to remove the plants from areas used by children. Internet Web sites can be an excellent source of information on plant identification, treatment of rash, and eradication of identified plants (www.poisonivy.us/, www.fda.gov/fdac/features/796_ivy.html, http://res2.agr.gc.ca/ecorc/poison/). Although ACD as a result of contact with poison ivy, oak, or sumac is rarely a serious affliction requiring hospitalization, it is a common and unpleasant condition often requiring medical intervention. Unfortunately, it is not as simple as "leaves of three, let them be."

The Primary Care Approaches section focuses on physical and developmental assessment and other topics specific to children and their families. If you are interested in author guidelines and/or assistance, contact Patricia L. Jackson Allen at Pat.Jackson@nursing.ucsf.edu

Tables

Table 1. Treatment of Poison Ivy, Oak, and Sumac Dermatitis

**Topical Treatment after Contact**

- Wash skin with soap and water as soon as contact identified, preferably within 10 minutes of contact.

- If greater than 10 minutes, but not more than 8 hours has elapsed since contact, wash skin with Tecnu®.

**Topical Treatment of Lesions**

- Keep lesions clean by washing with mild soap and water daily. Mild debridement of crusted lesions with soap and water is beneficial.

- Open vesicles so serum can drain. May lightly wrap weeping lesions with gauze but do not use prolonged occlusive dressings.
May use over-the-counter preparation such as Calamine lotion or Ivy Rest to reduce itching and dry lesions. Do not use topical preparations with antihistamines, benzocaine, or with neomycin sulfate due to the potential for development of sensitization.

Tepid baths with colloidal oatmeal (Aveeno) may reduce itching. Hot water baths or showers increase pruritus.

Tap water or Burrow's or Domeboro solutions applied with dressings to blistered or oozing lesions for 20 minutes twice a day helps dry weepy lesions.

Moderate glucocorticoid (eg., betametasone dipropionate 0.05% lotion [Diprosone®], hydrocortisone valerate 0.2% cream [Westcort®], triamcinolone acetonide 0.1% cream or lotion [Kenalog®]) can be used twice daily for mild to moderate dermatitis or for larger affected areas. Temporary application of an occlusive dressing over medication will increase its effectiveness but also systemic absorption. Do not use moderate or high potency glucocorticoid preparations on the face or genitals.

High potency glucocorticoid (eg., betamethasone dipropionate 0.05% cream [Diprosone®], monetasone furoate 0.1% ointment [Elocon®], tiamcinolone acetonide 0.5% cream [Aristocort®]) can be used once or twice daily on areas covering less than 10% of the skin surface. Occlusive dressings should not be used with high potency glucocorticoids.

**Systemic Treatment**

**Antihistamines** are effective for controlling pruritus. They may be taken 3-4 times per day unless sedation interferes with activities of daily living or school.

- Diphenhydramine (Benedryl®): Children >9.1 kg 12.5-25 mg q 4-6 hr, < 9.1 kg 6.25-12.5 mg q 4-6 hr.
- Chlorpheniramine (Chlor-Trimeton®): Children 6-12 yr 2 mg 3-4 times daily, not to exceed 12 mg/day.
- Hydroxyzine (Atarax®, Vistaril®): Children 6-12 yr 12.5-25 mg q 6 hr 3-4 times daily, children > 6 yr 12.5 mg q 6 hr 3-4 times daily.

**Systemic glucocorticoids** are used for severe allergic dermatitis unless there are contraindications.

- Prednisone: 1-2 mg/kg once per day for 7-10 days, then taper for 7-10 days.

**Antibiotics** for secondary bacterial infection. Need broad spectrum antibiotic to cover multiple potential organisms.

- Amoxicillin/clavulanate (Augmentin®): Children > 40 kg 500 mg bid, infants and children > 3 mo 12.5 mg/kg bid
- Cefuroxime (Ceftin®): Children > 12 yr 125-250 mg bid, children 3 mo-12 yr 15 mg/ kg bid.
- Azithromycin (Zithromax®): Children 2-15 yr 10 mg/kg once a day (not to exceed 500 mg/dose) on day 1, then 5 mg/kg once a day (not to exceed 250 mg/dose) for 4 more days.

Table 2. Facts about Poison Ivy, Poison Oak, and Poison Sumac

- All parts of the plant, including the root system, contain urushiol and are potentially allergenic. Plants are allergenic all year round, not just when they have leaves. Even dead plants can cause lesions.

- The serum released from the vesicular lesions of poison ivy, oak, or sumac does not spread the rash and is not contagious to others. Scratching the lesions will not spread the rash but may cause secondary skin damage or infection.

- The majority of children are sensitive to urushiol, the allergic oil of the plant, with sensitivity increasing during the first decade of life. Prior lack of allergic response to urushiol does not guarantee future lack of response.

- Soap and water wash will not prevent lesions if the urushiol has been on the skin for greater than 10 minutes.

- Urushiol can remain on gardening, sporting, or camping equipment for weeks causing allergic contact dermatitis unless washed off with soap and water.

- Treatment will not reduce the duration of symptoms but can reduce the severity.

- Urushiol oil can become airborne when plants are burned, or cut with a mower or weed-whacker. Poison ivy, oak, and sumac should not be burned, and protection should be taken when doing yard work in areas with these plants.

References


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