

Botanical Briefs: *Toxicodendron* Dermatitis

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PRACTICE POINTS

- *Toxicodendron* dermatitis is a pruritic vesicular eruption in areas of contact with the plant.
- Identification and avoidance are primary methods of preventing *Toxicodendron* dermatitis.

Toxicodendron dermatitis is a type IV hypersensitivity reaction resulting from exposure to urushiol found in poison ivy, poison oak, and poison sumac. The dermatitis presents as a pruritic erythematous rash with vesicles and bullae in areas that were in contact with the plant. Symptoms present after 24 to 48 hours and can be managed with a variety of treatments, depending on severity. Avoidance is the principal way to prevent *Toxicodendron* dermatitis, highlighting the importance of educating patients on identification of plants.

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Reactions to poison ivy, poison oak, and poison sumac, which affect 10 to 50 million Americans a year,¹ are classified as *Toxicodendron* dermatitis; 50% to 75% of US adults are clinically sensitive to these plants.² Furthermore, people of all ethnicities, skin types, and ages residing in most US geographical regions are at risk.³ Allergenicity is caused by urushiol, which is found in members of the Anacardiaceae family.⁴ Once absorbed, urushiol causes a type IV hypersensitivity reaction in those who are susceptible.⁵

Cutaneous Manifestations

Toxicodendron dermatitis presents with an acute eczematous eruption characterized by streaks of intensely pruritic

and erythematous papules and vesicles (Figure 1). Areas of involvement are characterized by sharp margins that follow the pattern of contact made by the plant's leaves, berries, stems, and vines.⁶ The fluid content of the vesicles is not antigenic and cannot cause subsequent transmission to oneself or others.³ A person with prior contact to the plant who becomes sensitized develops an eruption 24 to 48 hours after subsequent contact with the plant; peak severity manifests 1 to 14 days later.⁷

When left untreated, the eruption can last 3 weeks. If the plant is burned, urushiol can be aerosolized in smoke, causing respiratory tract inflammation and generalized dermatitis, which has been reported among wildland firefighters.² Long-term complications from an outbreak are limited but can include postinflammatory hyperpigmentation and secondary bacterial infection.⁸ Rare reports of



FIGURE 1. Erythematous vesicular rash with secondary crusting in a patient with *Toxicodendron* dermatitis.

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nephrotic syndrome also have appeared in the literature.⁹ *Toxicodendron dermatitis* can present distinctively as so-called black dot dermatitis.⁶

Nomenclature

Poison ivy, poison oak, and poison sumac are members of the family Anacardiaceae and genus *Toxicodendron*,⁶ derived from the Greek words *toxikos* (poison) and *dendron* (tree).¹⁰

Distribution

Toxicodendron plants characteristically are found in various regions of the United States. Poison ivy is the most common and is comprised of 2 species: *Toxicodendron rydbergii* and *Toxicodendron radicans*. *Toxicodendron rydbergii* is a nonclimbing dwarf shrub typically found in the northern and western United States. *Toxicodendron radicans* is a climbing vine found in the eastern United States. Poison oak also is comprised of 2 species—*Toxicodendron toxicarium* and *Toxicodendron diversilobum*—and is more common in the western United States. Poison sumac (also known as *Toxicodendron vernix*) is a small shrub that grows in moist swampy areas. It has a predilection for marshes of the eastern and southeastern United States.^{6,11}

Identifying Features

Educating patients on how to identify poison ivy can play a key role in avoidance, which is the most important step in preventing *Toxicodendron dermatitis*. A challenge in identification of poison ivy is the plant's variable appearance; it grows as a small shrub, low-lying vine, or vine that climbs other trees.

As the vine matures, it develops tiny, rough, "hairy" rootlets—hence the saying, "Hairy vine, no friend of mine!" Rootlets help the plant attach to trees growing near a water source. Vines can reach a diameter of 3 inches. From mature vines, solitary stems extend 1 to 2 inches with 3 characteristic leaves at the terminus (Figure 2), prompting another classic saying, "Leaves of 3, let it be!"¹²



FIGURE 2. Poison ivy consists of 3 terminal leaves.

Poison oak is characterized by 3 to 5 leaflets. Poison sumac has 7 to 13 pointed, smooth-edged leaves.⁶

Dermatitis-Inducing Plant Parts

The primary allergenic component of *Toxicodendron* plants is urushiol, a resinous sap found in stems, roots, leaves, and skins of the fruits. These components must be damaged or bruised to release the allergen; slight contact with an uninjured plant part might not lead to harm.^{2,13} Some common forms of transmission include skin contact, ingestion, inhalation of smoke from burning plants, and contact with skin through contaminated items, such as clothing, animals, and tools.¹⁴

Allergens

The catecholic ring and aliphatic chain of the urushiol molecule are allergenic.¹⁵ The degree of saturation and length of the side chains vary with different catechols. Urushiol displays cross-reactivity with poison ivy, poison oak, and poison sumac. Urushiol from these plants differs only slightly in structure; therefore, sensitization to one causes sensitization to all. There also is cross-reactivity between different members of the Anacardiaceae family, including *Anacardium occidentale* (tropical cashew nut), *Mangifera indica* (tropical mango tree), *Ginkgo biloba* (ginkgo tree), and *Semecarpus anacardium* (Indian nutting nut tree).¹²

Poison ivy, poison oak, and poison sumac cause allergic contact dermatitis as a type IV hypersensitivity reaction. First, urushiol binds and penetrates the skin, where it is oxidized to quinone intermediates and bound to haptens. Then, the intermediates bind surface proteins on antigen-presenting cells, specifically Langerhans cells in the epidermis and dermis.⁵

Presentation of nonpeptide antigens, such as urushiol, to T cells requires expression of langerin (also known as CD207) and CD1a.¹⁶ Langerin is a C-type lectin that causes formation of Birbeck granules; CD1a is a major histocompatibility complex class I molecule found in Birbeck granules.^{5,17} After Langerhans cells internalize and process the urushiol self-hapten neo-antigen, it is presented to CD4⁺ T cells.⁶ These cells then expand to form circulating activated T-effector and T-memory lymphocytes.¹⁸

The molecular link that occurs between the hapten and carrier protein determines the response. When linked by an amino nucleophile, selective induction of T-effector cells ensues, resulting in allergic contact dermatitis. When linked by a sulfhydryl bond, selective induction of suppressor cells occurs, resulting in a reduced allergic contact dermatitis response.¹⁹ In the case of activation of T-effector cells, a cell-mediated cytotoxic immune response is generated that destroys epidermal cells and dermal vasculature.² The incidence and intensity of poison ivy sensitivity decline proportionally with age and the absence of continued exposure.²⁰

Preventive Action—Patients should be counseled that if contact between plant and skin occurs, it is important to remove contaminated clothing or objects and wash them with soap to prevent additional exposure.^{14,21} Areas of the skin that made contact with the plant should be washed with water as soon as possible; after 30 minutes, urushiol has sufficiently penetrated to cause a reaction.² Forceful unidirectional washing with a damp washcloth and liquid dishwashing soap is recommended.²²

Several barrier creams are commercially available to help prevent absorption or to deactivate the urushiol antigen. These products are used widely by forestry workers and wildland firefighters.²³ One such barrier cream is bentoquatam (sold as various trade names), an organo-clay compound made of quaternium-18 bentonite that interferes with absorption of the allergen by acting as a physical blocker.²⁴

Treatment

After *Toxicodendron* dermatitis develops, several treatments are available to help manage symptoms. Calamine lotion can be used to help dry weeping lesions.^{25,26} Topical steroids can be used to help control pruritus and alleviate inflammation. High-potency topical corticosteroids such as clobetasol and mid-potency steroids such as triamcinolone can be used. Topical anesthetics (eg, benzocaine, pramoxine, benzyl alcohol) might provide symptomatic relief.^{27,28}

Oral antihistamines can allow for better sleep by providing sedation but do not target the pruritus of poison ivy dermatitis, which is not histamine mediated.^{29,30} Systemic corticosteroids usually are considered in more severe dermatitis—when 20% or more of the body surface area is involved; blistering and itching are severe; or the face, hands, or genitalia are involved.^{31,32}

Clinical Uses

Therapeutic uses for poison ivy have been explored extensively. In 1892, Dakin³³ reported that ingestion of leaves by Native Americans reduced the incidence and severity of skin lesions after contact with poison ivy. Consumption of poison ivy was further studied by Epstein and colleagues³⁴ in 1974; they concluded that ingestion of a large amount of urushiol over a period of 3 months or longer may help with hyposensitization—but *not* complete desensitization—to contact with poison ivy. However, the risk for adverse effects is thought to outweigh benefits because ingestion can cause perianal dermatitis, mucocutaneous sequelae, and systemic contact dermatitis.²

Although the use of *Toxicodendron* plants in modern-day medicine is limited, development of a vaccine (immunotherapy) against *Toxicodendron* dermatitis offers an exciting opportunity for further research.

REFERENCES

1. Pariser DM, Ceilley RI, Lefkovits AM, et al. Poison ivy, oak and sumac. *Derm Insights*. 2003;4:26-28.

2. Gladman AC. *Toxicodendron* dermatitis: poison ivy, oak, and sumac. *Wilderness Environ Med*. 2006;17:120-128. doi:10.1580/pr31-05.1
3. Fisher AA. Poison ivy/oak/sumac. part II: specific features. *Cutis*. 1996;58:22-24.
4. Cruse JM, Lewis RE. *Atlas of Immunology*. CRC Press; 2004.
5. Valladeau J, Ravel O, Dezutter-Dambuyant C, et al. Langerin, a novel C-type lectin specific to Langerhans cells, is an endocytic receptor that induces the formation of Birbeck granules. *Immunity*. 2000;12:71-81. doi:10.1016/s1074-7613(00)80160-0
6. Marks JG. Poison ivy and poison oak allergic contact dermatitis. *J Allergy Clin Immunol*. 1989;9:497-506.
7. Williams JV, Light J, Marks JG Jr. Individual variations in allergic contact dermatitis from urushiol. *Arch Dermatol*. 1999;135:1002-1003. doi:10.1001/archderm.135.8.1002
8. Brook I, Frazier EH, Yeager JK. Microbiology of infected poison ivy dermatitis. *Br J Dermatol*. 2000;142:943-946. doi:10.1046/j.1365-2133.2000.03475.x
9. Rytand DA. Fatal anuria, the nephrotic syndrome and glomerular nephritis as sequels of the dermatitis of poison oak. *Am J Med*. 1948;5:548-560. doi:10.1016/0002-9343(48)90105-3
10. Gledhill D. *The Names of Plants*. Cambridge University Press; 2008.
11. American Academy of Dermatology Association. Poison ivy, oak, and sumac: how to treat the rash. Accessed October 19, 2022. <https://www.aad.org/public/everyday-care/itchy-skin/poison-ivy/treat-rash>
12. Monroe J. *Toxicodendron* contact dermatitis: a case report and brief review. *J Clin Aesthet Dermatol*. 2020;13(9 suppl 1):S29-S34.
13. Marks JG Jr, Anderson BE, DeLeo VA. *Contact & Occupational Dermatology*. 4th ed. Jaypee Brothers Medical Publishers; 2016.
14. Fisher AA, Mitchell JC. *Toxicodendron* plants and spices. In: Rietschel RL, Fowler JF Jr, eds. *Fisher's Contact Dermatitis*. 4th ed. Williams and Wilkins; 1995:461-523.
15. Dawson CR. The chemistry of poison ivy. *Trans N Y Acad Sci*. 1956;18:427-443. doi:10.1111/j.2164-0947.1956.tb00465.x
16. Hunger RE, Sieling PA, Ochoa MT, et al. Langerhans cells utilize CD1a and langerin to efficiently present nonpeptide antigens to T cells. *J Clin Invest*. 2004;113:701-708. doi:10.1172/JCI19655
17. Hanau D, Fabre M, Schmitt DA, et al. Human epidermal Langerhans cells cointernalize by receptor-mediated endocytosis "non-classical" major histocompatibility complex class I molecules (T6 antigens) and class II molecules (HLA-DR antigens). *Proc Natl Acad Sci U S A*. 1987;84:2901-2905. doi:10.1073/pnas.84.9.2901
18. Gayer KD, Burnett JW. *Toxicodendron* dermatitis. *Cutis*. 1988;42:99-100.
19. Dunn IS, Liberato DJ, Castagnoli N, et al. Contact sensitivity to urushiol: role of covalent bond formation. *Cell Immunol*. 1982;74:220-233. doi:10.1016/0008-8749(82)90023-5
20. Kligman AM. Poison ivy (*Rhus*) dermatitis; an experimental study. *AMA Arch Derm*. 1958;77:149-180. doi:10.1001/archderm.1958.01560020001001
21. Derraik JGB. *Heracleum mantegazzianum* and *Toxicodendron succedaneum*: plants of human health significance in New Zealand and the National Pest Plant Accord. *N Z Med J*. 2007;120:U2657.
22. Neill BC, Neill JA, Brauker J, et al. Postexposure prevention of *Toxicodendron* dermatitis by early forceful unidirectional washing with liquid dishwashing soap. *J Am Acad Dermatol*. 2018;81:E25. doi:10.1016/j.jaad.2017.12.081
23. Kim Y, Flamm A, ElSohly MA, et al. Poison ivy, oak, and sumac dermatitis: what is known and what is new? *Dermatitis*. 2019;30:183-190. doi:10.1097/DER.0000000000000472
24. Marks JG Jr, Fowler JF Jr, Sheretz EF, et al. Prevention of poison ivy and poison oak allergic contact dermatitis by quaternium-18 bentonite. *J Am Acad Dermatol*. 1995;33:212-216. doi:10.1016/0190-9622(95)90237-6
25. Baer RL. Poison ivy dermatitis. *Cutis*. 1990;46:34-36.
26. Williford PM, Sheretz EF. Poison ivy dermatitis. nuances in treatment. *Arch Fam Med*. 1995;3:184.
27. Amrol D, Keitel D, Hagaman D, et al. Topical pimecrolimus in the treatment of human allergic contact dermatitis. *Ann Allergy Asthma Immunol*. 2003;91:563-566. doi:10.1016/S1081-1206(10)61535-9

28. Stephanides SL, Moore C. *Toxicodendron* poisoning treatment & management. *Medscape*. Updated June 13, 2022. Accessed October 19, 2022. <https://emedicine.medscape.com/article/817671-treatment#d11>
29. Munday J, Bloomfield R, Goldman M, et al. Chlorpheniramine is no more effective than placebo in relieving the symptoms of childhood atopic dermatitis with a nocturnal itching and scratching component. *Dermatology*. 2002;205:40-45. doi:10.1159/000063138
30. Yosipovitch G, Fleischer A. Itch associated with skin disease: advances in pathophysiology and emerging therapies. *Am J Clin Dermatol*. 2003;4:617-622. doi:10.2165/00128071-200304090-00004
31. Li LY, Cruz PD Jr. Allergic contact dermatitis: pathophysiology applied to future therapy. *Dermatol Ther*. 2004;17:219-223. doi:10.1111/j.1396-0296.2004.04023.x
32. Craig K, Meadows SE. What is the best duration of steroid therapy for contact dermatitis (*Rhus*)? *J Fam Pract*. 2006;55:166-167.
33. Dakin R. Remarks on a cutaneous affection, produced by certain poisonous vegetables. *Am J Med Sci*. 1829;4:98-100.
34. Epstein WL, Baer H, Dawson CR, et al. Poison oak hyposensitization. evaluation of purified urushiol. *Arch Dermatol*. 1974;109:356-360.