# Botanical Briefs: Contact Dermatitis Induced by Western Poison Ivy *(Toxicodendron rydbergii)*

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PRACTICE **POINTS**

- Western poison ivy *(Toxicodendron rydbergii)* accounts for many of the cases of *Toxicodendron* contact dermatitis (TCD) in the western and northern United States. Individuals in these regions should be educated on how to identify *T rydbergii* to avoid TCD.
- Dermatologists should include TCD in the differential diagnosis when a patient presents with an erythematous pruritic rash in a linear pattern with sharp borders.
- Most patients who experience intense itching and pain from TCD benefit greatly from prompt treatment with an oral or intramuscular corticosteroid. Topical steroids rarely provide relief; oral antihistamines provide no benefit.

"Leaves of three, leave it be" serves as an apt caution for avoiding poison ivy *(Toxicodendron* species) and its dermatitis-inducing sap. *Toxicodendron* contact dermatitis (TCD) poses a notable burden to the American health care system by accounting for half a million reported cases of allergic contact dermatitis (ACD) annually. Identifying and avoiding physical contact with the western poison ivy *(Toxicodendron rydbergii)* plant prevails as the chief method of preventing TCD. This article discusses common features of *T rydbergii* as well as clinical manifestations and treatment options following exposure to this allergenic plant.

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## Clinical Importance

Western poison ivy (*Toxicodendron rydbergii*) is responsible for many of the cases of *Toxicodendron* contact dermatitis (TCD) reported in the western and northern United States. *Toxicodendron* plants cause more cases of allergic contact dermatitis (ACD) in North America than any other allergen[1]; 9 million Americans present to physician offices and 1.6 million present to emergency departments annually for ACD, emphasizing the notable medical burden of this condition.[2,3] Exposure to urushiol, a plant resin containing potent allergens, precipitates this form of ACD.

An estimated 50% to 75% of adults in the United States demonstrate clinical sensitivity and exhibit ACD following contact with *T rydbergii*.[4] Campers, hikers, firefighters, and forest workers often risk increased exposure through physical contact or aerosolized allergens in smoke. According to the Centers for Disease Control and Prevention, the incidence of visits to US emergency departments for TCD nearly doubled from 2002 to 2012,[5] which may be explained by atmospheric $CO_2$ levels that both promote increased growth of *Toxicodendron* species and augment their toxicity.[6]

## Cutaneous Manifestations

The clinical presentation of *T rydbergii* contact dermatitis is similar to other allergenic members of the *Toxicodendron* genus. Patients sensitive to urushiol typically develop a pruritic erythematous rash within 1 to 2 days of exposure (range, 5 hours to 15 days).[7] Erythematous and edematous streaks initially manifest on the extremities and often progress to bullae and oozing papulovesicles. In early disease, patients also may display black lesions on or near the rash[8] (so-called black-dot dermatitis) caused by oxidized urushiol deposited on the skin—an uncommon yet classic presentation of TCD. Generally, symptoms resolve

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without complications and with few sequalae, though hyperpigmentation or a secondary infection can develop on or near affected areas.[9,10]

## Taxonomy

The *Toxicodendron* genus belongs to the Anacardiaceae family, which includes pistachios, mangos, and cashews, and causes more cases of ACD than every other plant combined.[4] (Shelled pistachios and cashews do not possess cross-reacting allergens and should not worry consumers; mango skin does contain urushiol.)

*Toxicodendron* (formerly part of the *Rhus* genus) includes several species of poison oak, poison ivy, and poison sumac and can be found in shrubs (*T rydbergii* and *Toxicodendron diversilobum*), vines (*Toxicodendron radicans* and *Toxicodendron pubescens*), and trees (*Toxicodendron vernix*). In addition, *Toxicodendron* taxa can hybridize with other taxa in close geographic proximity to form morphologic intermediates. Some individual plants have features of multiple species.[11]

## Etymology

The common name of *T rydbergii*—western poison ivy—misleads the public; the plant contains no poison that can cause death and does not grow as ivy by wrapping around trees, as *T radicans* and English ivy (*Hedera helix*) do. Its formal genus, *Toxicodendron*, means "poison tree" in Greek and was given its generic name by the English botanist Phillip Miller in 1768,[12] which caused the renaming of *Rhus rydbergii* as *T rydbergii*. The species name honors Per Axel Rydberg, a 19th and 20th century Swedish-American botanist.

## Distribution

*Toxicodendron rydbergii* grows in California and other states in the western half of the United States as well as the states bordering Canada and Mexico. In Canada, it reigns as the most dominant form of poison ivy.[13] Hikers and campers find *T rydbergii* in a variety of areas, including roadsides, river bottoms, sandy shores, talus slopes, precipices, and floodplains.[11] This taxon grows under a variety of conditions and in distinct regions, and it thrives in both full sun or shade.

## Identifying Features

*Toxicodendron rydbergii* turns red earlier than most plants; early red summer leaves should serve as a warning sign to hikers from a distance (Figure 1). It displays trifoliate ovate leaves (ie, each leaf contains 3 leaflets) on a dwarf nonclimbing shrub (Figure 2). Although the plant shares common features with its cousin *T radicans* (eastern poison ivy), *T rydbergii* is easily distinguished by its thicker stems, absence of aerial rootlets (abundant in *T radicans*), and short (approximately 1 meter) height.[4]

Curly hairs occupy the underside of *T rydbergii* leaflets and along the midrib; leaflet margins appear lobed or rounded. Lenticels appear as small holes in the bark that turn gray in the cold and become brighter come spring.[13]

The plant bears glabrous long petioles (leaf stems) and densely grouped clusters of yellow flowers. In autumn, the globose fruit—formed in clusters between each twig and leaf petiole (known as an axillary position)—change from yellow-green to tan (Figure 3). When urushiol exudes from damaged leaflets or other plant parts, it oxidizes on exposure to air and creates hardened black deposits on the plant. Even when grown in garden pots, *T rydbergii* maintains its distinguishing features.[11]

## Dermatitis-Inducing Plant Parts

All parts of *T rydbergii* including leaves, stems, roots, and fruit contain the allergenic sap throughout the year.[14] A person must damage or bruise the plant for urushiol to be released and produce its allergenic effects; softly brushing against undamaged plants typically does not induce dermatitis.[4]

## Pathophysiology of Urushiol

Urushiol, a pale yellow, oily mixture of organic compounds conserved throughout all *Toxicodendron* species, contains highly allergenic alkyl catechols. These catechols possess hydroxyl groups at positions 1 and 2 on a benzene ring; the hydrocarbon side chain of poison ivies (typically 15–carbon atoms long) attaches at position 3.[15] The catechols and the aliphatic side chain contribute to the plant's antigenic and dermatitis-inducing properties.[16]

**FIGURE 1.** Hiker's view of red leaves on a western poison ivy shrub *(Toxicodendron rydbergii)*(photographed from a distance of 3 meters) in Spearfish Canyon, South Dakota. Photograph courtesy of Thomas W. McGovern, MD.

**FIGURE 2.** Five characteristic features for identifying western poison ivy *(Toxicodendron rydbergii)*: (1) leaves with 3 leaflets; (2) a low-growing, nonclimbing habitat; (3) early autumn colors starting in summer; (4) lack of deposits of oxidized urushiol; and (5) drupes, or fruit (arrows), where the petiole meets the branch or root (Spearfish Canyon, South Dakota). Photograph courtesy of Thomas W. McGovern, MD.

**FIGURE 3.** Mature fruit of *Toxicodendron rydbergii* in winter. "Western poison ivy" by Whitney Cranshaw is licensed under CC BY 3.0 (https://creativecommons.org/licenses/by/3.0/us/).

The high lipophilicity of urushiol allows for rapid and unforgiving absorption into the skin, notwithstanding attempts to wash it off. Upon direct contact, catechols of urushiol penetrate the epidermis and become oxidized to quinone intermediates that bind to antigen-presenting cells in the epidermis and dermis. Epidermal Langerhans cells and dermal macrophages internalize and present the antigen to $CD4^+$ T cells in nearby lymph nodes. This sequence results in production of inflammatory mediators, clonal expansion of T-effector and T-memory cells specific to the allergenic catechols, and an ensuing cytotoxic response against epidermal cells and the dermal vasculature. Keratinocytes and monocytes mediate the inflammatory response by releasing other cytokines.[4,17]

Sensitization to urushiol generally occurs at 8 to 14 years of age; therefore, infants have lower susceptibility to dermatitis upon contact with *T rydbergii*.[18] Most animals do not experience sensitization upon contact; in fact, birds and forest animals consume the urushiol-rich fruit of *T rydbergii* without harm.[3]

## Prevention and Treatment

*Toxicodendron* dermatitis typically lasts 1 to 3 weeks but can remain for as long as 6 weeks without treatment.[19] Recognition and physical avoidance of the plant provides the most promising preventive strategy. Immediate rinsing with soap and water can prevent TCD by breaking down urushiol and its allergenic components; however, this is an option for only a short time, as the skin absorbs 50% of urushiol within 10 minutes after contact.[20] Nevertheless, patients must seize the earliest opportunity to wash off the affected area and remove any residual urushiol. Patients must be cautious when removing and washing clothing to prevent further contact.

Most health care providers treat TCD with a corticosteroid to reduce inflammation and intense pruritus. A high-potency topical corticosteroid (eg, clobetasol) may prove effective in providing early therapeutic relief in mild disease.[21] A short course of a systemic steroid quickly and effectively quenches intense itching and should not be limited to what the clinician considers severe disease. Do not underestimate the patient's symptoms with this eruption.

Prednisone dosing begins at 1 mg/kg daily and is then tapered slowly over 2 weeks (no shorter a time) for an optimal treatment course of 15 days.[22] Prescribing an inadequate dosage and course of a corticosteroid leaves the patient susceptible to rebound dermatitis—and loss of trust in their provider.

Intramuscular injection of the long-acting corticosteroid triamcinolone acetonide with rapid-onset betamethasone provides rapid relief and fewer adverse effects than an oral corticosteroid.[22] Despite the long-standing use of sedating oral antihistamines by clinicians, these drugs provide no benefit for pruritus or sleep because the histamine does not cause the itching of TCD, and antihistamines disrupt normal sleep architecture.[23-25]

Patients can consider several over-the-counter products that have varying degrees of efficacy.[4,26] The few products for which prospective studies support their use include Tecnu (Tec Laboraties Inc), Zanfel (RhusTox), and the well-known soaps Dial (Henkel Corporation) and Goop (Critzas Industries, Inc).[27,28]

Aside from treating the direct effects of TCD, clinicians also must take note of any look for signs of secondary infection and occasionally should consider supplementing treatment with an antibiotic.

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