Rochelle Parpana

Poison Ivy: Biochemical Mechanism Suggests Methods for Relief

Leaves of three; let it be, goes the timeless adage warning children and adults to keep away from the three-leaf clustered poison ivy. Poison ivy, or *Toxicodendron radicans*, is notorious for the itchy rash it induces on the skin known as urushiol-induced contact dermatitis. The culprit of this ailment, the oily allergen, urushiol, is found in the sap of poison ivy and also in that of poison oak and poison sumac. When in contact with skin, urushiol can stimulate a painful response from our immune system.

Urushiol is a mixture of several closely related organic compounds. Each compound is comprised of a catechol with an attached alkyl R group as shown in Figure 1. The length of the alkyl R group varies according to the botanical species to which it belongs, but is generally 15-17 carbons long. Catechols in poison ivy typically have R groups consisting of 15 carbons. R may contain all single carbon-carbon bonds (i.e. saturated), or one or more double carbon-carbon bonds (i.e. unsaturated). Severity of response to urushiol correlates with the number of double bonds found in the R group. While response to saturated urushiol is very rare, 85-90% of people react to unsaturated urushiol when it has at least two double bonds. The longer the catechol's alkyl R chain, the more effective urushiol is at inducing an allergic reaction in people exposed to it.

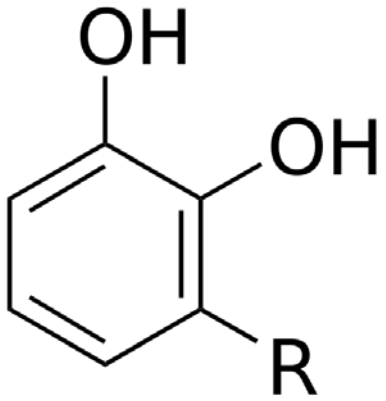

Figure 1: Typical urushiol compound with alkyl chain R group

Urushiol is generally found in the space between plant cells beneath the outermost layer of poison ivy stems and leaves, also know as the cuticle. As long as the poison ivy plant remains intact and unbroken, we are generally out of harm's way. When, however, the plant is damaged (e.g. leaves or stems broken), the oily allergen seeps out of the plant and, if in contact with bare skin, will be absorbed. Urushiol molecules bind to comparatively large integral membrane proteins (Figure 2) on the affected skin cells. These integral membrane proteins are permanently bound to our skin cell membranes and, once bound by urushiol, change their physical conformation, to which our immune systems then respond.

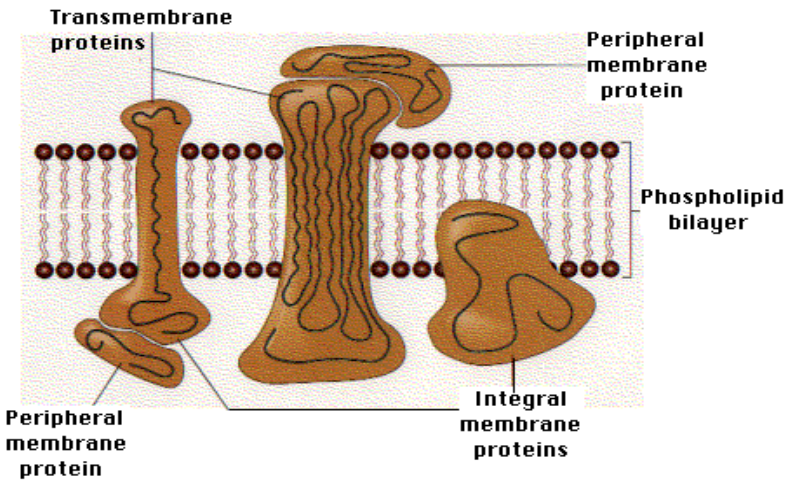

Figure 2: Integral membrane proteins, unlike peripheral membrane proteins, are permanently fixed to the cell membrane

White blood cells, or leukocytes, are a fundamental part of our immune system. Unlike the other cells in our body, leukocytes act as independent organisms, free-floating among the body's tissues and checking cells for abnormalities as they pass by. B cells are a specific class of leukocytes; when a B cell encounters a foreign invader, referred to as an antigen, the stimulated B cell matures into a plasma cell that produces antibodies specific to the activating antigen (Figure 3).

Urushiol changes the conformation of integral membrane proteins. B cells incorrectly interpret the modification as a sign of a threat and produce large amounts of antibodies specific to the invader.

During the sensitization phase, these specifically-engineered antibodies, also known as immunoglobulins (IgE for allergic reactions), bind to other cells in our body known as mast cells and basophils. Mast cells are known for housing powerful chemical mediators, while basophils contain an anticoagulant that keeps blood from clotting. Both cell types produce histamine, that, when released in large quantities into the bloodstream, can cause the body to respond violently. The appropriate amount of sensitizing exposure for mast cells and basophils to become fully saturated with IgE varies from person to person; some people are born extremely sensitive to urushiol, others not at all.

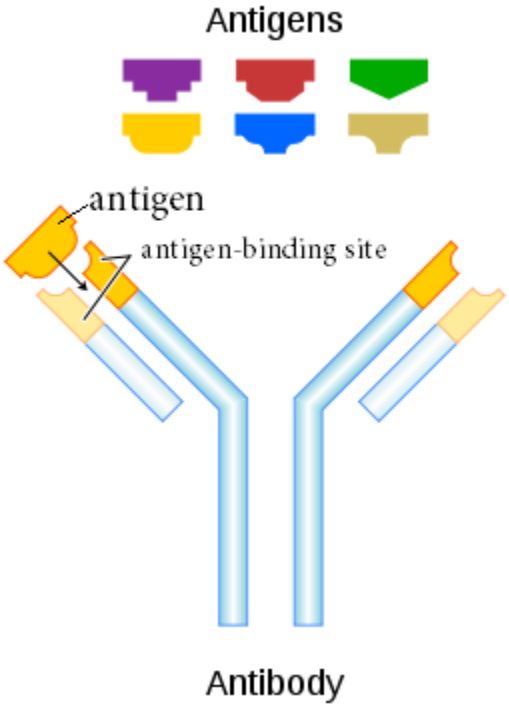

Figure 3: B cells form antibodies that correspond specifically to each antigen

When an allergy-prone individual comes into contact with poison ivy after the sensitization period has passed, the immune system is ready to unleash a full attack. IgE in the IgE-saturated mast cells and basophils bind to proteins on the surface of the recognized antigens (modified integral membrane proteins) while still attached to the mast cells or basophils. This binding triggers the response of the complement complex, a

family of about 20 proteins circulating in the blood. Once an IgE antibody has bound to the antigen, the first complement protein binds elsewhere to the antigen, prompting the next complement protein to bind, and so forth.

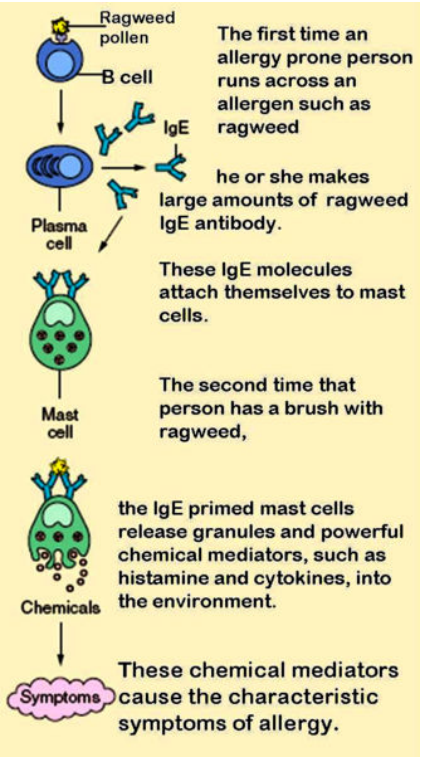

Figure 4: Role of mast cell in typical allergic reaction, e.g. as initiated by ragweed pollen

After the complement complex has been created, the offending cell and its attached substituents—including the bound mast cells and basophils—are destroyed, releasing histamine and other allergy mediators into the bloodstream (Figure 4). Histamine triggers an increase in blood flow to the site of inflammation by increasing the permeability of capillaries to white blood cells, allowing them to engage and eliminate the pathogens. Increase in permeability, however, leads to leakage of plasma proteins and fluid in the tissue, which ultimately producing swelling, flushing and rash. Histamine is also responsible for the contraction of smooth muscle, which can inhibit airways in the lungs, and increased mucus production. Symptoms of a poison ivy rash normally last for 2-4 weeks.

Urushiol is not contagious, as it is neither a virus nor a pathogen. As there are no vaccines to counteract urushiol symptoms, the best treatments isolate the urushiol and its symptoms once they have developed. The oily allergen holds onto almost anything it contacts; towels, sweatshirts, blankets, etc. are common areas of exposure between our skin and urushiol. It is especially important to wash the exposed area thoroughly with

soap and hot water as soon as possible. Urushiol oils are insoluble in water (i.e. hydrophobic) and, when exposed to water and soap, will crowd together to form micelles (Figure 5) that are easily washed off.

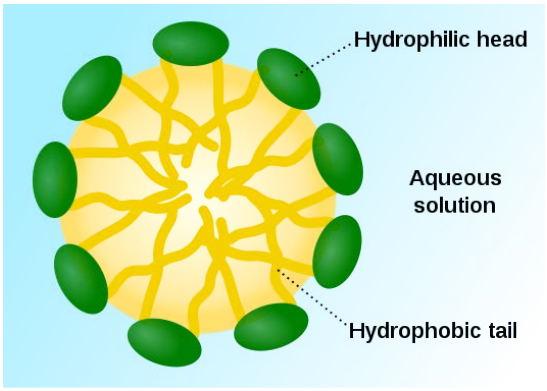

Figure 5: Typical micelle

Over-the-counter body lotions may be used to soothe itching and dry up blisters. It is important, however, that the lotion not contain benzocaine, zirconium or topical antihistamine, as these substances can also induce allergic reactions on the already irritated skin. 1%-hydrocortisone cream may also be applied topically or, in more severe cases, injected. Cortisol, another name for hydrocortisone, is produced naturally within our bodies. T cells are another type of leukocyte with several different roles; one includes helping B cells mature into plasma cells. By preventing the proliferation of T cells, cortisol can weaken the immune system. Side effects may include a temporary increase in blood glucose levels, and dryness and itchiness at the site of application.

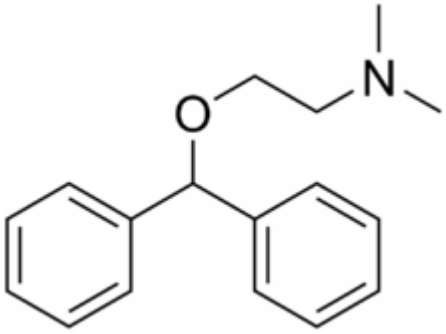

Figure 6: diphenhydramine

Oral antihistamines may also be taken to alleviate the dermatitis symptoms. Diphenhydramine (Figure 6), commercially known as Benadryl, is a commonly used non-prescription antihistamine. Benadryl, known as an H_1-receptor antagonist, competes with histamine for the receptor sites on nerves, vascular smooth muscle,

glandular cells, endothelium and mast cells. While by itself it induces no biological response from the H1-receptor, Benadryl is able to block or dampen the effects of active histamine-bound H_1-receptor complexes. Side effects include dizziness, drowsiness, headache, constipation and dry mouth.

Corticosteroids such as prednisone are another treatment that can be taken either orally or via injection. Although prednisone (Figure 7) is not naturally produced by our bodies like hydrocortisone, both prednisone and hydrocortisone function in similar ways. By suppressing the proliferation of T cells, corticosteroids inhibit our immune system. Anti-inflammatory responses are up-regulated, while pro-inflammatory responses are repressed. The side effects of prednisone can be severe, including (but not limited) to vision problems, depression, change in personality and irregular heartbeat. In children prednisone may slow growth and development, and may increase chances of developing osteoporosis. This medication should only be taken in severe cases.

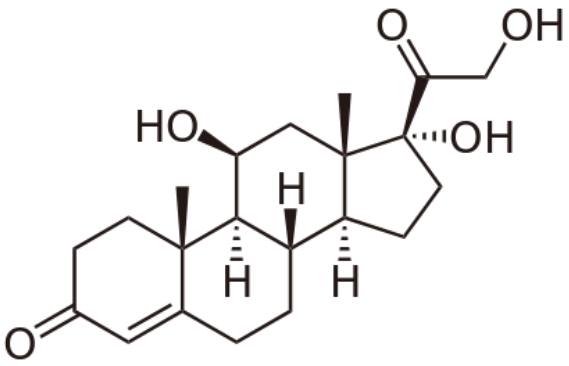

Figure 7: Prednisone

Although poison ivy has ailed humans indiscriminately for centuries, growing biomedical and pharmaceutical industries may one day develop a way to administer immunity prior to urushiol exposure. Until then, our best defense remains the same; avoid it when you can: *Leaves of three; let it be.*

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