Rashes come in many different forms, and causes and triggers vary widely. While medical expertise is required to diagnose the root cause of a rash and determine a treatment plan, there are some common signs that can help narrow down where a rash came from.

The word “rash” is generic and only refers to a symptom. Most rashes are characterized by itching, inflammation and redness, or other discoloration of the skin.

**Hives (Urticaria)**
Hives, or urticaria, is an outbreak of raised bumpy red welts on the skin. The causes of hives can vary, and are often difficult to determine. Autoimmune dysfunction often lies at the root of the problem, but food intolerances can also trigger hives, as well as stress and excess body heat. Certain medications, insect bites and allergens have been linked to some cases of hives. Some patients experience a one-time episode of hives, where others may experience chronic hives.

**Shingles (Herpes Zoster)**
Shingles is an extremely painful rash that can appear anywhere on the body. Shingles is caused by the varicella-zoster virus, the same virus that causes the chicken pox. In patients who have contracted the chicken pox, the virus enters a dormant state. Shingles results from a reactivation of the virus. Shingles is more common in adults over age of 60, particularly those with weak immune systems. The rash typically leaves behind blisters that form scar tissue. Some patients who suffer from shingles continue to experience pain or itching long after the rash is gone, due to permanent nerve damage in the affected area.

**Christmas Tree Rash (Pityriasis Rosea)**
Pityriasis rosea usually appears on the chest, abdomen and back. It is not uncommon for the rash to appear in a small area at first, then grow larger over the course of several weeks. This rash gets its nickname from the shape of the scars, which resemble “Christmas trees.” Pityriasis rosea most often lasts six to eight weeks, but can last much longer.

**Rosacea**
Rosacea is a chronic condition characterized by redness of the skin, particularly on the face. Rosacea “flares up” in connection with aggravating factors, including certain foods, alcoholic beverages, strenuous exercise and extreme temperatures (hot or cold).

**Poison Ivy/Poison Oak/Poison Sumac**
These plants, well known for their three-leaf structure, contain an oily substance called urushiol, which causes a rash when it comes into direct contact with the skin. Treatment of these rashes typically requires application of a topical steroid over a two-to-three-week period, until the rash subsides.

**Psoriasis**
Psoriasis stemming from an autoimmune disorder comes in many different varieties affecting different areas of the body. Psoriasis consists of red, scaly patches on the skin. During a psoriasis flare, “scales” form as dead skin cells accumulate. These scaly areas often take on a gray or silvery appearance.

**Eczema (Atopic Dermatitis)**
Eczema refers to many different conditions resulting in generally similar symptoms: patches of red and inflamed skin. Eczema is often linked to environmental allergens and chemical irritants. Some patients with eczema have found that avoiding harsh soaps can bring a degree of relief, and it is...
sometimes necessary to test for allergic reactions, especially in children. Treatments for eczema include topical creams and steroids, and may sometimes require the use of antibiotics if the area becomes infected.

In general, it is advisable to consult a physician about any rash that persists for more than a few days. A dermatologist can usually identify the specific type of rash through physical examination, although additional testing is sometimes required. As any rash can potentially indicate a serious condition, early diagnosis is always best.

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**Poliovirus Triggers Innate Immune Response in Glioblastoma Therapy**

An investigational therapy using modified poliovirus to attack cancer tumors appears to unleash the body’s own capacity to fight malignancies by activating an inflammation process that counter’s the ability of cancer cells to evade the immune system.

Describing this process in a paper published Sept. 20 in the journal *Science Translational Medicine*, Duke Cancer Institute researchers provide the first published insight into the workings of a therapy that has shown promise in early clinical trials in patients with recurrent glioblastoma, a lethal form of brain cancer. The modified poliovirus received a breakthrough therapy designation from the United States Food and Drug Administration last year, expediting research, according to a Duke press advisory.

“We have had a general understanding of how the modified poliovirus works, but not the mechanistic details at this level,” said co-senior author Matthias Gromeier, M.D., a professor in the Duke Department of Neurosurgery who developed the therapy. “This is hugely important to us. Knowing the steps that occur to generate an immune response will enable us to rationally decide whether and what other therapies make sense in combination with poliovirus to improve patient survival.”

Gromeier, with expertise in cancer biology, collaborated with fellow Duke researcher and co-senior author Smita Nair, Ph.D., an immunologist and professor in the Department of Surgery. The research team elucidated how the poliovirus works not only to attack cancer cells directly, but also to trigger a longer-lasting immune response that appears to inhibit regrowth of the tumor.

Using human melanoma and breast cancer cell lines, and then validating the findings in mouse models, the researchers found that the modified poliovirus therapy starts by attaching to malignant cells, which have an abundance of CD155 protein. The CD155 protein is otherwise known as the poliovirus receptor. The modified virus then begins to attack the tumor cells, directly killing many, but not all. This releases tumor antigens.

The second phase of assault is more complicated. By killing the cancer cells, the modified poliovirus triggers an alarm within the immune system, alerting the body’s defenses to go on the attack.

This appears to occur when the modified poliovirus infects dendritic cells and macrophages. Dendritic cells then present the tumor to T cells to launch an immune response. Once the immune system is activated against the poliovirus-infected tumor, the cancer cells can no longer hide and they remain vulnerable to ongoing immune attack.

“Not only is poliovirus killing tumor cells, it is also infecting the antigen-presenting cells, which allows them to function in such a way that they can now raise a T-cell response that can recognize and infiltrate a tumor,” Nair said. “This is an encouraging finding, because it means the poliovirus stimulates an innate inflammatory response.”

Nair and Gromeier said further studies will focus on the additional immune activity following exposure to the modified virus.

In addition to Gromeier and Nair, study authors include Michael C. Brown, Eda K. Holt, David Boczkowski, Elena Dobrikova, Mubeen Mosaheb, Vidya Chandramohan and Darell D. Bigner.

The study received support the Public Health Services (CA197264, CA124756 and CA190991), the Department of Defense, (W81XWH-16-1-0354); the Lefkofsky Family Foundation, Hope & Gavin Wolfe, and the BLAST Glioblastoma Foundation.

Nair and Gromeier, along with Brown, Chandramohan and Bigner, own intellectual property related to this research, which has been licensed to a Istari Oncology Inc. Gromeier and Bigner are cofounders and equity holders in the company.

Dr. Matthias Gromeier holds a sample of the modified poliovirus he developed that attacks glioblastoma brain tumor cells.