Surface Eruptions: A Primer on Skin Rashes and Botanical Medicine Approaches to Their Treatment

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Rash is a very broad medical term defined as a "widespread eruption of skin lesions." They can vary widely in appearance, with causes ranging from insect bites to HIV and reactions to medications, they can affect one small part of the body or cover large areas. Rashes can be dry, moist, bumpy, smooth, cracked, or blistered. They can involve pain, itching, and color changes. There is a vast spectrum for how skin rashes manifest in patients with more and less pigmentation and this is an arena of medicine fraught with controversy (most skin rashes are presented in photos of less pigmented people in the medical literature, but most skin manifestations of STI's are presented with photos of people of with more pigmentation--a significant effector of bias when that distortion is presented to medical students). Some rashes will clear up on their own, some respond to herbal remedies, and others might be a sign of something more serious that needs to be addressed differently. We will discuss many of the reasons why skin rashes occur and the underlying mechanism for their presentation, including skin infections from bacterial, fungal, viral, or parasitic infections, allergies, and medications. We discuss the internal and topical botanical medicine interventions and their rationale, we also discuss concepts of suppression vs. healing.

Dermatology is arguably the most allopathic (*allo*-'other' or 'opposite', *pathy*-suffering) of the medical specialties. I recall one of my dermatology professors in Naturopathic medical school summing this science up in the following way. “In regard to rashes, if it’s wet, make it dry; if it’s dry, make it wet. If the condition persists, begin administration of steroids.” In natural medicine, we recognize that a skin rash is usually superficial manifestation of a potentially greater underlying disorder. Suppression of this manifestation will often result in a worsening of some deeper pathology. A classic example that most of us have seen in clinical practice is the steroidal suppression of eczema in young children resulting in a later manifestation of asthma. Skin disorders can also be some of the most frustrating conditions for any clinician to be met with, not to mention for which the patient contends. The chronicity and slow resolution of these conditions make it sometimes difficult for the patient to maintain faith in non-suppressive processes. Those patients experienced with the quick fix of conventional medical interventions, will often become discouraged and/or non-compliant and not follow the process of elimination that we prescribe, especially when a red, burning, itchy rash is becoming temporarily worse. This can reflect negatively on the practitioner and is acutely true when a patient with a rash has been treated with botanical medicines for several weeks with minimal results, goes to a dermatologist and two days into a prednisone taper, the rash is completely resolved. There are precious few resources available to the practitioner of alternative medicine for means of dealing with these seldom life threatening but always quality-of-life threatening conditions. I refer the reader to the references section of this paper for those resources that have been invaluable for me in my practice. I am hopeful that this discussion will itself become such a resource,

amenable to updates and additions as we become more aware of and deepen our understanding of skin conditions.

We will discuss briefly the major features of the pathology, a description the lesions and causes for them. Then we will discuss potential strategies for resolution of the condition. What is presented here is not meant to be used in protocol fashion, rather as a strategic base for the practitioner to develop their own individualized strategies to best meet their patient's needs. We could spend days on the subject of dermatology; this discussion is certainly not intended to be all-inclusive, so we start with common manifestations of skin rashes and by creating some categories of skin diseases. There are those conditions that are related to microbial influences, these include bacterial related diseases like acne, impetigo and cellulitis, fungal infections, and viral diseases such as warts, measles, mumps and rubella. We will also include conditions that are due to a dysregulation of immune response; these include hypersensitivities like urticaria and eczema. These categories of conditions are by no means mutually exclusive to one another. As we shall see there are many instances where one manifestation evolves into another, these categories provide us with a frame of reference.

Naturopathic medicine frequently emphasizes that "the microbe is nothing, the milieu is everything", addressing the terrain within the body and in the skin makes far more sense than killing the opportunists who take advantage of that terrain. Recognizing that the human body is a largely water-soluble terrain with special mechanisms in place for dealing with fat-soluble substances is an important place to begin. The skin is one of the major organs of elimination for the whole body with the pores of the skin being the actual route of elimination. What the body is eliminating becomes food for whatever microorganisms are living in those pores and other surface irregularities. Hormones are an example of a fat-soluble material that, when in excess, either from endogenous production, or from exogenous intake, can be excreted through the skin and provide a more favorable environment for the growth of many microbes. The ability of the liver to process fatty material (phase 1 and phase 2 activity) determines how much will end up being excreted through the skin. The relative degree of oxidation of those fatty substances while being processed through the body also has a bearing on how irritating they will be when deposited within the pores of the skin.

For any microbially induced or mediated skin condition, diet becomes the first point of order when trying to change the terrain of the body. Generally speaking, a diet low in processed foods, high in fiber, with special consideration of the quality of the fats that one is consuming provides a decent dietary foundation. Avoiding simple carbohydrates (some dermatologists refer to acne as skin diabetes) provides less sugar substrate for the overgrowth of skin bacteria and fungi. Consider food sensitivities, the use of an elimination diet and the patient's digestive capacity (enzymes vs. HCl). For some conditions, like acne and some cases of eczema, food sources of hormonal modulators, lignans from flax and root vegetables, beta-sitosterol, and isoflavones from soy are helpful. Increasing consumption of anti-inflammatory and anti-microbial food items like garlic, turmeric, cumin, essential fatty acids and cruciferous vegetables. It is important to emphasize monounsaturated fats like olive oil, nuts, and avocado, and to

avoid saturated fats (too solid), hydrogenated fats (too mutated) and polyunsaturated fats (too reactive).

Bacterial overgrowth tends to present with inflammation, redness and irritation leading to pain. Acne vulgaris is a good example of a process that can be described as a chronic inflammatory dermatosis affecting the hair follicle. It is extremely prevalent in middle to late teenage years, affecting both males and females, with males typically afflicted with a more severe manifestation of the disease. Acne is seen in all races but seems to be milder in those of Asian ancestry. The pathogenesis of acne is considered to be due to the bacterial lipases of *Cutibacterium acnes* (formerly *Propionibacterium acnes)* which break down the sebaceous oils, liberating highly irritating fatty acids inside the hair follicle, inducing the initial inflammatory phase. Its manifestation varies tremendously between individuals and may be induced or exacerbated by drugs (corticosteroids, testosterone, gonadotropins, contraceptives, iodides, and bromides), occupational contacts (cutting oils, chlorinated hydrocarbons, and coal tars), and occlusive conditions such as heavy clothing, tropical climates, and the application of comedogenic topical agents (make-up, lotions, ointments, etc.) The pathogenesis of acne vulgaris involves the interaction of several host factors, including the stimulation of sebaceous glands by circulating androgens, dysbiosis of the pilosebaceous follicle microbiome, and cellular immune responses. In addition, other factors such as genetics and diet may also influence the development and progression of the disease[1]. The micro-comedo (the pimple) serves as the primary lesion and is the precursor for all clinical manifestations of acne vulgaris. It is characterized by a small, hyperkeratotic plug primarily composed of corneocytes and located in the lower region of the follicular infundibulum. Micro-comedones gradually evolve and develop into other acne lesions, which include closed comedones (whiteheads), open comedones (blackheads), and inflammatory papules, pustules, and nodules. The progression of micro-comedones into other types of acne lesions has been theorized to involve the following 4 primary pathogenic events:

- Increased sebum production (seborrhea)
- Follicular hyper-keratinization
- Overgrowth of *Cutibacterium acnes*-Anaerobic diphtheroid that naturally exists as part of the skin flora.
- Inflammation

The gradual accumulation of keratinous material and sebum converts a micro-comedo into a closed comedo (pimple to a whitehead). The follicular orifice can gradually expand, resulting in the formation of an open comedo (blackhead) where oxidized lipids and melanin within the comedo contribute to its characteristic dark black color. *C acnes* contributes to the development of inflammatory pustules and papules. Eventually, follicles rupture with the release of bacteria, keratin, and proinflammatory lipids into the surrounding dermis, exacerbating the inflammation and creating nodules within the skin.

Most patients with acne vulgaris typically have normal androgen levels in their body. However, in certain conditions such as congenital adrenal hyperplasia, polycystic

ovarian syndrome, and adrenal or ovarian tumors, excessive androgen production is produced in the body, ultimately leading to a proportionate severity of acne. The role of androgens in the development of acne involves several modifiable mechanisms[2]:

- The adrenal glands and the gonads secrete androgens.
- Sebaceous glands can also synthesize androgens through the conversion of DHEA to testosterone via the action of enzymes whose effects can be modified by diet and botanical medicines.
- 5-alpha reductase in the sebaceous gland converts testosterone to 5-alpha-dihydrotestosterone (DHT)--which is up to 5 times stronger in its androgenic potential.
- Sebaceous glands and the outer root sheath keratinocytes of the follicular epithelium have androgen receptors that bind DHT and testosterone.
- Androgens stimulate the growth of sebaceous glands and increase their secretory function, leading to seborrhea and acne formation.
- Seborrhea provides a growth medium for *C acnes* from which they hydrolyze triglycerides in sebum, as a nutrient source, into free fatty acids and glycerol.

The development of acne involves several factors associated with *C. acnes* and the immune response as listed below[3,4]:

- The acne-associated strains of *C. acnes* have been found to possess a heightened capacity to stimulate the pro-inflammatory cascade, specifically involving TH17 cells which stimulate secretion of cytokines such as interferon (IFN)-gamma and interleukin (IL)-17, which are promotors of inflammation. In contrast, the strains associated with healthy skin have been shown to stimulate TH17 cells to produce the anti-inflammatory cytokine IL-10.
- Antibiotic resistance in acne has been linked to acne-associated strains of *C acnes,* which form biofilms within the follicles.
- *C. acnes* stimulate the innate immune response to produce IL-1 by activating the nod-like receptor P3 (NLRP3) inflammasome in human sebocytes and monocytes.
- *C. acnes* activates toll-like receptor-2 on perifollicular macrophages, which triggers the release of more pro-inflammatory cytokines such as IL-8 and IL-12. These cytokines attract neutrophils, whose lysosomal enzymes contribute to follicular rupture.

Many of the botanical products that have been found to be beneficial for the treatment of acne vulgaris contain plant sterols at high doses. Botanicals such as saw palmetto, soy products, pygeum bark, stinging nettle root, and pumpkin seed extract are rich in plant sterols and beta-sitosterol is the most commonly occurring, it can often be found in supplement form in your local health food store. The mechanism of its action is believed to work through inhibition of cholesterol catabolism and modulating hormonal influences that trigger inflammation[5]. Sterols block the absorption of cholesterol from the intestinal tract and are the active ingredient in cholesterol-lowering margarine products such as Take Control (beta-sitosterol) and Benecol (sitostanol). Sterols may also have

benefit because they cannot be converted to testosterone in the body, beta-sitosterol specifically inhibits 5-alpha reductase activity and interferes with androgen binding at the site of the receptor. The recommended dose of beta-sitosterol is 20–30 mg three times a day.

There are three well known herbal agents that play a specific role in decreasing 5 alpha-reductase activity. These agents are *Serenoa repens, Pygeum africanum*, and *Urtica dioica; Pygeum* and *Urtica* have also been shown to inhibit the aromatization of testosterone to estrogen. Beta-sitosterol is a constituent of both *Serenoa* and *Pygeum*. Overharvesting and lack of sustainable agriculture has reduced populations of Pygeum, and as the medically useful component is the bark of the tree, a single harvest can be unrecoverable. *Urtica dioica,* stinging nettle, is a widely available plant that has been intensively researched for its effects on hair growth, mostly related to the 5 alpha-reductase qualities in its dried root with some studies also showing a stimulation of dermal papilla cells. Urtica root also contains beta sitosterol which stimulates angiogenesis by increasing vascular endothelial growth factor (VEGF) synthesis and supports turnover of compromised skin cells[5]. Usual dosage of these agents looks like 100 mg/day of Pygeum, and 500-1500 mg/day of Urtica root.

The saw palmetto plant (*Serenoa repens*) is a small palm native to the southeastern United States and grows wild in Florida’s natural areas. It is a slow-growing, clumping, multi-trunked palm that typically grows 5 to 10 feet tall and spreads 4 to 10 feet wide. The plant produces small berries that are an important food source for wildlife species and are commonly harvested for medicinal purposes in late summer to early fall, usually from August to October. Harvesters usually look for berries that have turned from green to blue-black in color. Harvesting involves hand-picking the berries, which grow in clusters on the plant. The fruit of saw-palmetto has been eaten by humans for centuries, it was a staple food item for many of the early Native Americans of Florida, although the taste is definitely not for everyone. The medicinal value of the fruit has been described in scientific literature since the 1800s where it is prescribed in tinctures of the fruits and crushed seeds and used for relief of prostate gland swelling, reduction of acne severity and promoting hair growth. Saw palmetto berry's active ingredients include fatty acids, plant sterols, flavonoids, and high molecular weight polysaccharides (sugars). The perceived medical benefit from eating the berries includes inhibition of 5α-reductase I and II, reducing DHT conversion from testosterone, and inhibition of DHT binding to the cytosolic androgen receptors, as well as other anti-inflammatory effects, with 320 mg/day being a decent daily oral dosage.

Although acne may be unavoidable, it can be effectively controlled through several measures. Regularly washing one's face with a pH-balancing cleanser can help manage acne symptoms. Avoiding high glycemic index foods, dairy-based products may help manage acne symptoms in some individuals. Enough cannot be said about the far reaching role that psycho-social stress can play in exacerbating acne--especially in adolescents. Benzoyl peroxide, Salicylic acid, oral and topical antibiotics and oral contraceptives are all used fairly commonly in conventional treatment of acne, and these measures can be very logical for some presentations.

Benzoyl peroxide possesses both comedolytic and antimicrobial properties. It is available in various formulations and concentrations ranging from 2.5% to 10%. Typically, it is applied once daily, focusing on 1 or 2 small areas during the initial 3 days to test for any potential hypersensitivity reactions. Benzoyl peroxide should not be applied simultaneously with tretinoin due to its oxidizing effect on tretinoin. To avoid any reaction, benzoyl peroxide should be applied in the morning, whereas tretinoin should be applied in the evening. It is important to note that skin irritation may occur with benzoyl peroxide use, particularly at high concentrations. It may also have a bleaching effect on clothing and hair. Salicylic acid is a desquamating keratolytic (it promotes the sloughing of old, dead and dying skin cells), it is also comedolytic and antimicrobial and available over the counter at concentrations of 1-5%.

Vitamin A derivatives such as tretinoin, tazarotene, adapalene, and trifarotene, are often included in the initial management for patients with acne. Vitamin A effectively targets both comedones and inflammatory papules and pustules. For patients with predominantly comedonal acne, topical retinoids can be effective all by themselves and are also recommended for maintenance therapy after successful treatment. Patients with papulopustular acne can benefit from adding a topical antimicrobial agent, which can be used in combination with benzoyl peroxide to minimize the risk of antibiotic-resistant bacteria emerging. Topical retinoids are vitamin A derivatives that act by binding to retinoic acid receptors (RARs) and retinoid X receptors (RXRs) within the keratinocytes. Tretinoin, tazarotene, and adapalene are topical retinoids that primarily act upon the RAR-beta and RAR-gamma receptors. Tretinoin also targets the RAR-alpha receptor. Upon binding, the retinoid-receptor complexes are transported into the cell's nucleus, activating the retinoid hormone response element, stimulating the transcription of several regulatory genes. The skin response to this process includes the normalization of follicular keratinization and the loosening of the cohesiveness of the keratinocytes, thereby decreasing the formation of micro-comedones[6]. The topical retinoid should be applied once daily, preferably at night, due to the reported photo-lability of vitamin A derivatives. Before applying a retinoid, ensure that the skin is dry, apply a thin layer to the whole affected area rather than spot-treating individual lesions, 1/2 gram or a pea-sized amount of the medication should be sufficient to cover the entire face. The frequency of application should be adjusted to minimize the risk of skin irritation, most people can tolerate every other night or every third night in the beginning and, as tolerance improves, the frequency of application can be increased. Some common adverse effects of topical retinoids are dryness, irritation, flaking of the skin, and sensitivity to sunlight due to skin thinning. However, these adverse effects can often be managed using moisturizers and sunscreens. It is important to note that retinoids, topical and oral, are not recommended for use during pregnancy, or for any female of childbearing age, without full knowledge and consent of the potential harm.

Our clinic makes a topical hydrogel formula for topical treatment of acute acne exacerbations that looks like, per oz:

1% Allantoin (300 mg)
3% Pentoxifylline (900 mg)

0.025% Vitamin A (tretinoin, hydroxypinacolone, retinyl palmitate)
(7.5 mg)(usually 8,333 ius/ml)
10 drops Lemon Essential oil
in an Aloe based hydrogel,
Apply to skin lesions and irritated skin 1-2 times per day.

Impetigo contagiosa is strongly associated with *group A hemolytic Strep* in children under 5 and usually manifests as a thick, sticky, honey-colored crust over ulcers. Bullous impetigo is caused by *Staph. aureus* and presents with thin, varnish-like crusts. It resembles ringworm in that it heals centrally while spreading peripherally. Effective treatment requires dramatically changing the terrain at the site and reducing the bacterial load. An antibacterial soap (my personal favorite is Dr. Bronner's Lavender Castille soap) to wash the area three times a day, followed by an antibacterial rinse, a strong decoction of *Hydrastis, Mahonia or Berberis* usually works well. Unpasteurized honey can be placed directly on the lesion with a cover; the sugar in the honey has a desiccating effect on the bacteria and presents some anti-microbial action of its own. These lesions usually resolve within 5-7 days without complication. When there is complication, one can have scarring, especially on the face that will usually resolve after several months[7].

Cellulitis is a common bacterial skin infection, with over 14 million cases occurring in the United States annually. It accounts for approximately 3.7 billion dollars in ambulatory care costs and 650000 hospitalizations annually. It is usually attributed to the same bacteria as impetigo, that is, *Group A beta-hemolytic Strep, Staph aureus* and other gram negative bacteria[8]. The streptococci produce streptokinase, DNAse, and hyaluronidase each of which digests a component of the cellular architecture of the body that would normally keep the infection contained to that area. This condition usually requires some break in the skin, scratching or other trauma, in order for the bacteria to be able to descend deep enough into the tissue to cause cellulitis. Increased susceptibility to this condition is recognized in alcoholics, patients with nephrotic edema, stasis dermatitis, lymphedema and malnutrition. Erysipelas is a type of cellulitis also related to *Streptococcal* bacteria. It was previously most commonly seen in infants, young children and the elderly and often after upper respiratory infection. Now, we also see it quite often in young adults with piercings of the head and face. The manifestation of cellulitis begins with redness, swelling, heat and tenderness, occasionally there will be blistering or an infiltrated surface that looks like the skin of an orange. It is an acute bacterial infection causing inflammation of the deep dermis and surrounding subcutaneous tissue. The infection is without an abscess or purulent discharge. Treatment should be more aggressive than that for impetigo; *Veratrum viride* tincture as an external treatment is suggested by Ellingwood in his *American Materia Medica, Therapeutics and Pharmacognosy*[9]. I have had opportunity to use it with good effect as the moistening agent in a warming compress applied directly to the infected area. Internally, a tincture of Echinacea (4 parts), Mahonia (3 parts), Phytolacca (2 parts), and Baptisia (1 part) can be quite effective in resolving this situation, especially when used in tandem with alternating hot/cold application to the area. Sunlight is essential and drawing poultices made from ground flax, bentonite clay or slippery elm can also be helpful. It is imperative that the

patient work to resolve those issues that caused them to have an increased susceptibility to this condition. Patients who are immunocompromised, colonized with methicillin-resistant *Staphylococcus aureus*, bitten by animals, or have comorbidities such as diabetes mellitus may become infected with other bacteria. If the clinician correctly identifies and promptly treats cellulitis, it typically resolves with appropriate antimicrobial approaches, delayed treatment allows more microbial opportunists time to add to the colony, antimicrobial resistance is a horizontally shared trait in bacteria, that is, the genes for resistance are spread across different species, within the same generation, the magic handshake. The more traffic you get through a wound, the more likely you are to develop antimicrobial resistance. The other option for inheritance is vertical, you got that trait from your parents or grandparents and you will likely pass it on to your grandchildren.

Fungal infections range from superficial infections like the *Tineas* (ringworm, athlete's foot, jock itch) to systemic infections like *Candida* or *Aspergillus*, dermatophytes are the most common pathogenic filamentous fungi, with an infection rate of as high as 20%-25% worldwide. They generally infect the nails, skin, and hair and commonly manifest as tinea capitis, onychomycosis, tinea corporis, and tinea pedis. Dermatophytes may also invade the dermal tissue and even deep organs, particularly in immunocompromised patients with congenital or acquired immunodeficiency, and these infections can progress to life-threatening conditions. There are three basic types of dermatophytes depending on host preferences and ecological niches: anthropophilic dermatophytes are primarily transmitted from person to person and usually result in chronic infections with moderate clinical symptoms; zoophilic dermatophytes prefer selective animal hosts but can normally infect other species, including humans, often causing inflammatory skin infections; and geophilic dermatophytes survive on keratinized waste present in the soil and are rarely pathogenic but can produce more severe inflammation than anthropophilic species. *Trichophyton rubrum* is among the most frequently detected species globally and is responsible for 50%-90% of dermatophytoses. There are several genera of fungi that cause *Tinea* infections; they are all more and less susceptible to the measures that we will discuss here. These fungi all create a keratolytic enzyme, causing scaling of the skin, crumbling nails and breaking hair. They create filae, hair-like projections like a mycelium in the skin that cause vesicles and redness; many people develop an allergic response, which causes an additional output of histamine in the area and incredible, voluptuous itching. Diagnosis can be made by scraping the infected area, applying potassium hydroxide and visualizing the spores and hyphae under a microscope[10]. This condition is one of the reasons why the modern health care practitioner really needs to have microscope in their office. Treatment varies based on the location involved; this is a situation where radical shifts in the biological terrain are essential. Shifting the pH of the climate around the feet by putting baking soda in one's shoes is a simple and effective measure for athlete's foot. Fungal infections of the nail beds are particularly long-lived infections, partly because the nails grow so slowly, but also because there is often not a great deal of blood flow to the ends of the body. I have found that a solution of 5% thyme oil in ethanol is very effective for

nail fungus of the hands and feet, it requires a twice a day application for up to two months, sometimes adding 2% DMSO makes it work a little quicker. For skin manifestations I find that a beeswax and olive oil salve with oregano and thyme oils added at the rate of 5 mls of each per two ounces of salve works fairly well. I have seen Tea tree oil work intermittently well for most skin fungi. Many times a fungal infection of the scalp will manifest as dandruff and responds pretty well to Aveda's Rosemary Essence shampoo into which is added Rosemary essential oil to make a 1% combination (5 ml in 500 ml), I have also had good results with Paul Mitchell's Tea Tree Oil Shampoo and Conditioner. An astringent rinse administered after shampooing helps control itching at least for a few hours, I recommend a diluted solution of 10% Glyco-thymuline (Alka-thyme and Alkalol work at least as well) mouthwash in water (these are 100 plus year old products that are essential oils in various alkaline solutions-promoted as mouthwashes and nasal rinses). Alternating hot and cold application in the shower can be helpful to encourage blood flow into and out of the scalp area. Internally, an alterative combination with diaphoretic properties can make a significant difference in the duration of the infection. A combination of Achillea (15 ml), Phytolacca (15 ml), Tilia (15 ml), Stillingia (10 ml), and Zanthoxylum (5 ml) dosed at 60 drops twice a day for a month to six weeks is pretty helpful.

Viral diseases of the skin include warts and most of the rash diseases of childhood. Warts are fairly straightforward and seem to have a strong association with the psyche. Sometimes the practitioner can "purchase" the warts of their younger patients, giving them a dollar for each wart usually makes the wart disappear in 1-2 weeks. Psychotherapy, homeopathy and constitutional botanical prescribing: including a lengthy interview is usually successful in adult patients. Topical interventions like 25% Podophyllum in alcohol, Thuja oil, salicylic acid, liquid nitrogen and surgical removal can be construed as superficial and suppressive, some level of internal treatment with agents that enhance host anti-viral defenses may be more ideal, consider botanical 'anti-viral' agents like *Lomatium, Hypericum, Glycyrrhiza*, vitamin A and *Melissa*. This concept of an herbal antiviral can get complicated-these are not necessarily items that kill viruses in a petri-dish--nor in the far more complicated living organism. The plant and its constituents stimulate a wide range of host defenses that include some responses to viral organisms that might lead to their eradication, or their assimilation into the micro-ecology of the body, as the case may be.

An in-depth discussion of the rash diseases of childhood would have to include a discussion of vaccinations, as much as I personally would love to forge into such a controversial realm, it goes beyond the scope of this discussion. Just know that, vaccines are like any other product, some of what is in the marketplace is well-made and accomplishes its stated objective, and other products are not as good...discernment doesn't make one an "anti-vaxxer" any more than lack of discernment makes one a "good citizen". There are governmental regulatory processes in place to ensure a consistent and safe supply of product deemed essential to the public's health and well-being. Your confidence in those processes may be proportionate to your confidence in your political leaders and the choices that they make on your behalf. It may be worth your time

exploring that industry before we engage in rhetorical shouting matches, because there is a lot of ignorance being pushed forward by both sides of the discussion.

A viral rash (also known as an exanthem*)* is an eruptive skin rash that is related to a viral infection, they most commonly occur in childhood and include chickenpox (varicella), fifth disease, measles (rubeola), roseola, and rubella (German measles). Over the past 7 decades, improved public health measures have dramatically decreased the number of cases of these conditions and it is easy to forget that each of the viral rashes have a distinct pattern of emergence, which is an aid in diagnosis[11].

Chickenpox, caused by the *Varicella-zoster (VZV)* virus, is a highly infectious disease, usually associated with childhood with around 4,000,000 cases diagnosed every year. By the time they reach adulthood, more than 95 percent of Americans have had chickenpox. Transmission occurs from person-to-person by direct contact or through the air. Chickenpox most commonly occurs in children between the ages of 5 and 9. In the US, in areas with a large number of children in daycare settings, chicken pox in children between the ages of 1 and 4 is more common[12].

Since 1995, a chickenpox vaccine has been available for children 12 months of age and older. The Center for Disease Control & Prevention's Advisory Committee on Immunization Practices, the American Academy of Pediatrics, and the American Academy of Family Physicians recommend that all children be vaccinated with the chickenpox vaccine between 12 and 18 months of age with a booster vaccination between 11 and 12 years of age. The UK reviewed the cost effectiveness of the vaccine and initially decided that it was not worth the expense. Their stated rationale is that if chickenpox in children disappears as a result of a vaccine program, adults would no longer have their immunity boosted by exposure to their chickenpox-suffering children and grandchildren and would be more likely to get shingles, which would cost more than complications from chicken pox[13]. It was not deemed cost-effective for the NHS to immunize children against chickenpox until November of 2023, when the vaccine was finally approved in the UK. Another situation where just because we can, doesn't mean we should...and, of course, the economic argument wins, always interesting when that happens in healthcare.

Symptoms are usually mild among children, but may be life threatening to infants, adults, and people with impaired immune systems:

- Fatigue and irritability one to two days before the rash begins
- Itchy rash on the trunk, face, under the armpits, on the upper arms and legs, and inside the mouth
- Fever/Malaise
- Decreased appetite
- Muscle and/or joint pain
- Cough or runny nose

Chickenpox has an incubation period of 10 to 21 days after exposure, it is contagious for one to two days before the appearance of the rash and until the blisters have dried and become scabs, which usually happens within 4 to 5 days of the onset of

the rash. Children should stay home and away from other children until all of the blisters have scabbed over. Family members who have never had chickenpox have a 90 percent chance of becoming infected when another family member in the household is infected.

First Disease-Measles

The first detailed description describing measles was published in 910 by Rhazes (860–932), who was chief physician at the hospital in Baghdad. According to his book *al-Judari wa al-Hasbah* (Treatise on Smallpox and Measles), measles is a disease caused by too much bilious blood. He states that even an experienced physician may have difficulty distinguishing smallpox from measles, both diseases are eruptive fevers but, smallpox is much more severe and leaves indelible skin scars on survivors, while the measles rash does not cause ulceration and disappears by peeling. Rhazes' treatise was translated into Latin by Gerard of Cremona in the 13th century when measles was referred to by the Latin word *morbilli* ("little disease", derived from *morbus*). In the 11th-12th centuries, several measles epidemics were reported in Europe and it is generally accepted that the disease was widespread in Europe and in South and East Asia, India and China all through the Middle Ages. In England and Scotland, measles epidemics were reported in the 17th century in 1670 and 1674, both of which were documented by the Englishman Thomas Sydenham (1624–1690), in a chapter entitled '*On the Measles'*, in his *Complete Works* published in 1693. He coined the term *measles* from the medieval English *mesles* and the Latin *misella* (diminutive of *misery*). His work differentiated measles from scarlet fever and smallpox. The disease persisted in an endemic state without interruption in Great Britain and Europe throughout the 18th century and even increased during the 19th and 20th centuries. In 1896, the American physician Henry Koplik (1858–1927) described a pathognomonic sign of measles, "Koplik's spots" which are bluish-white spots in a patchy pattern on the buccal mucosa, which present a few days before the skin rash manifests[14].

The general course of the infection begins after a 14 day incubation period, the disease begins with a 2–4 day invasion phase, and a progressive fever that can reach 40 °C, with general malaise and headaches. Patients often suffer with rhinitis, a runny nose, cough, and conjunctivitis that causes watery eyes. Köplik's sign is detectable in 70% of cases. The disease may be accompanied by diarrhea, abdominal pain and vomiting. More rarely, convulsions or a meningeal syndrome can be observed. A maculopapular rash appears, with little pruritus, formed by large irregular and confluent plaques. The rash begins on the face and neck and spreads in a single outbreak over 3–4 days to the entire body, including the palms of the hands and soles of the feet. While the fever subsides in 3–4 days, the rash sheds before disappearing. Patients are contagious 2–3 days before the invasion phase and remain contagious for 10 days[15].

Second Disease-Scarlet Fever

Scarlet fever is not caused by a virus but by the bacteria *Streptococcus pyogenes* (Group A *Streptococcus*) which causes a syndrome characterized by a blanching, erythematous, maculopapular rash often described as "sandpaper-like," a "strawberry

tongue," and exudative pharyngitis. The causative organism is a gram-positive bacterium adapted to humans that grows in pairs and chains and is responsible for a range of infections, including superficial, deep, and invasive conditions such as cellulitis, pharyngitis, erysipelas, and necrotizing fasciitis. Group A Strep produce streptococcal pyrogenic exotoxins (SPEs), which act as superantigens and are exceptionally potent activators of T cells, they are the primary cause of the erythematous rash associated with scarlet fever[16].

Scarlet fever epidemics were common in the 19th century and while the prevalence of scarlet fever declined in the 20th century, a resurgence of Group A Strep infections occurred in the 1980s, and now, more virulent epidemic strains have emerged, leading to an increase in scarlet fever cases. Notable complications can arise from Group A Strep infections, including rheumatic heart disease and poststreptococcal glomerulonephritis. Prompt treatment of acute infections is essential to prevent these complications.

Third Disease-Rubella-German measles, Three-day measles-

Rubella virus is an enveloped, positive-stranded RNA virus classified as a Rubivirus in the Matonaviridae family, its average incubation period is 17 days, with a range of 12 to 23 days. People infected with rubella are most contagious when the rash is erupting, but they can be contagious from 7 days before to 7 days after the rash appears. Rubella is transmitted primarily through direct or droplet contact from nasopharyngeal secretions, humans are the only natural hosts, and in temperate climates, infections usually occur during late winter and early spring. Rubella is characterized by a mild, maculopapular rash along with lymphadenopathy, and a slight fever, 1/4-1/2 of infections are asymptomatic[17].

The skin rash:

Usually starts on the face
Becomes generalized within 24 hours
Lasts an average of 3 days
Occurs in 50% to 80% of people infected with rubella

The Lymphadenopathy:

May precede rash
Often involves posterior auricular or suboccipital lymph nodes
Can be generalized
Lasts between 5 and 8 days

It is considered a relatively mild condition that can lead to complications and death and include thrombocytopenic purpura and encephalitis.

Arthralgia or arthritis may occur in up to 70% of adult women with rubella.

When rubella infection occurs during pregnancy, especially during the first trimester, serious consequences can result. These include:

Miscarriages/Fetal deaths/stillbirths

Severe birth defects known as Congenital Rubella Syndrome (CRS), a condition that affects a developing baby in the womb when the mother is infected with the

rubella virus. CRS can affect almost everything in the developing baby's body and cause complications such as:

Deafness
Cataracts
Heart defects
Intellectual disabilities
Liver and spleen damage
Low birth weight
Skin rash at birth
Glaucoma
Brain damage
Thyroid and other hormone problems
Inflammation of the lungs

Although Rubella has been eradicated from the US as of 2004, international exposure still manifested in 15 babies born last year with CRS.

Fourth disease--Duke's Disease

This controversial disease, also not a virus, was described by Clement Dukes in 1900 and was what we now refer to as staphylococcal scalded skin syndrome (SSSS or Ritter's disease). This disease is caused by epidermolytic (exfoliative) toxin-producing strains of *Staphylococcus aureus*. The term was dropped from medical textbooks in the 1960's. SSSS is usually see in infants and begins with an abrupt appearance of perioral erythema that is well-demarcate and tender to the touch, a rash covers most of the body in around 2 days. Applying slight pressure with side-to-side movement of a finger to the skin lesions results in displacement of the epidermis from the dermis (positive Nikolsky's sign). In most cases the lesions become fluid filled bullae or cutaneous blisters. The fluid in the bullae and blisters is clear and does not contain bacteria or white blood cells. The bullae and blisters will break and will then desquamate. The lesions do not always fill with fluid and in this case some refer to the disease as staphylococcal scarlet fever. Desquamation of lesions also occurs with staphylococcal scarlet fever. Within 7-10 days of lesion appearance the skin heals without any scarring. Secondary bacterial infections of the lesions can result in scarring[18].

Fifth Disease-*Erythema infectiosum*

Fifth disease is a common viral exanthem caused by *parvovirus B19,* a non-enveloped, single-stranded DNA virus, that is commonly seen in children between five and fifteen years old, in the spring and summer months. It is one of the six most common viral rashes in children and can also affect adults. Route of transmission is primarily via droplets from respiratory secretions, although it can also be spread through blood. Pruritus, low-grade fever, malaise, and sore throat precede the rash in approximately 10% of cases, lymphadenopathy is not generally present, and adults may complain of joint pain. Facial erythema ("slapped cheek") that consists of red

papules on the cheeks that rapidly coalesce in hours, forming red, slightly edematous, warm, plaques that are symmetric on both cheeks but do not cover the nasolabial fold and the circumoral region. The "slapped cheek" appearance fades in 4 days. Lace or net patterned rash is a unique characteristic eruption that begins on the extremities approximately 2 days after the onset of facial erythema and extends to the trunk and buttocks, fading in 6 to 14 days. At times, the rash (exanthem) begins with a blanket of redness and does not become characteristic until irregular clearing begins to take place. The eruptions may fade and then reappear in previously affected sites on the face and body during the next 2 to 3 weeks (recurrent phase). Extreme temperature changes, emotional upsets, and excess sunlight may stimulate recurrences. The rash fades without scaling or pigmentation and there may be a slight lymphocytosis or eosinophilia on complete blood count[19].

Sixth Disease-*Roseola infantum*

Roseola infantum is a common disease of childhood caused by a primary infection with human herpesvirus 6 (HHV-6) and human herpesvirus 7 (HHV-7). This disease, also known as exanthema subitum, presents in children between the ages of 6-12 months with 90% of cases occurring in children younger than two years. Caused by the B variant of HHV-6, patients with the virus classically present with an acute onset of a high-grade fever,104 F, for three to five days. The child will experience a rapid reduction (breaking) of the fever with an accompanying nonpruritic, pink papular rash that begins on the trunk. It is found all over the world and is considered to be the cause of 10% to 45% of febrile illness in infants. Due to the high fever and the ability of the virus to cross the blood-brain barrier, 15% of children will also experience an acute febrile seizure. HHV-6 will likely remain latent in immunocompetent patients but can be a major cause of morbidity and mortality in patients who are immunosuppressed. Primary symptoms include malaise, conjunctivitis, orbital edema, inflammation of the tympanic membranes, lymphadenopathy, irritability, anorexia, a bulging fontanelle, diarrhea, cough and other upper respiratory tract symptoms. Uvulo-palatoglossal spots also referred to as Nagayama spots, are erythematous papules found on the soft palate and uvula that are seen in two-thirds of patients[20]. The fever typically breaks after three to five days and small, rose-pink to red, maculopapular rash develops with numerous 2-5mm lesions. A pale halo can occur around the macules and papules in some cases. The rash usually begins on the trunk and can spread to the neck, extremities, and face, it is typically nonpruritic, blanching and persists from one to two days.

It is important for any health care practitioner to be able to differentiate between these fairly common illnesses. For the most part, they are illnesses that are benign when allowed to run their course naturally. Effective management of fever is probably the most critical for parents. It is useful to remember that your immune system works best at a body temperature of 102-103 degrees. Herbal teas are wonderful in this instance; yarrow, ginger and eupatorium can be sweetened with honey or stevia for kids. Their diaphoretic

action helps dissipate excess heat from the body. EmergenC packets by the Alacer Company (there are a dozen equivalent brands) make pretty good “Popsicles” rich with minerals, which prevent dehydration. Most febrile seizures are more related to dehydration and electrolyte imbalances rather than simple increases in body temperature. Hydrotherapy measures are usually the most effective for children of all ages. A wet sheet pack can turn the tide of even the most frightening febrile conditions and involves preparing a damp sheet, spread onto a dry warm blanket, preferably wool, and placed on a bed. Place the child in a warm to hot bath (fevers less than 101.5, use a hotter bath, fevers above 101.5, use a more tepid bath) for at least ten minutes. The patient can be drinking herbal tea, you can place oils and salts in the bath, or they can just play in the water, when you take them out of the bath wrap them up snugly in the wet sheet and the blanket, being sure to tuck the blanket in firmly around the neck and feet. Most children will go to sleep like this, leave them at least 15 minutes, the longer the better, if you can keep them there for several hours, the result will be more dramatic. Measles, Covid and RSV involve some immunosuppression that increases risk for secondary infections.

Some potentially relevant part of discussing infectious processes is a discussion on the basic functions of the immune system, that part of the immune system that is specific and learned. The cellular component of this part of the immune response is the lymphocyte; immunoglobulins make up the soluble component (floating around in your bloodstream and congregating in tissues). Lymphocytes are divided into three subsets, those derived from the thymus gland (T cells), those derived from the bone marrow (B cells) and natural killer cells (NK cells). T cells are responsible for cell-mediated immunity while the B cells are responsible for humoral immunity (antibodies, cytokines). NK cells are associated with the major histocompatibility complex system of the body; they determine what is self and what is non-self and help to eradicate neoplastic and foreign tissue. All of the lymphocytes can be found in the lymphatic tissues of the body, this includes the spleen, the intestinal mucosa, and the bone marrow as well as the peripheral lymphatic vessels and nodes. The lymphocytes have a preformed structure and an anticipatory strategy for the immune system. There are at least a million different types of preformed B cells and equally as many preformed T cells. Each different type of preformed lymphocyte has a particular affinity for different families of invading organisms.

When a specific antigen is presented to these elements of the specific immune response they begin to form antibodies to that substance. These antibodies are simply immunoglobulins with a specific affinity for that antigen. Immunoglobulins make up about 20% of plasma proteins and they are separated into 5 classes; IgM is the first antibody formed after initial exposure to an antigen, IgG is the most prevalent antibody in the blood, and is produced after IgM levels decline and serves to maintain the immunity that IgM began. IgA is an antibody that is secreted to the surface of the body through mucus membranes (saliva, tears, respiratory, intestinal, genitourinary tracts and colostrum) where it provides an early antibacterial and antiviral defense. IgD is important in the development and growth of B cells and serves to further alert the system about what it should be attacking and what should be left alone. IgE, like IgA, is found in the

mucosal secretions of the body. It is found in very low concentrations in the blood and higher concentrations in the tissues. IgE levels are elevated in patients with atopic conditions like eczema and other allergies. All types of antibodies bind vigorously to their antigen and contribute to their destruction directly or indirectly through initiation of the complement cascade.

Immunoglobulins have a fundamental structure shaped like a “Y” consisting of a constant portion, the stem of the Y and halfway up the arms, and a variable portion at the ends of the arms. The constant portion of the antibody always and forever recognizes certain classes of antigens, whereas the variable portion accommodates the diversity of substances within the class of antigens. It is the variable portion that binds to the antigenic substance because it is allowed to vary its structure in ways to that make a more exact fit to the antigen. In the case of IgE, the variable portion is closely related to mast cells and basophils. When an antigen is bound to IgE, it causes the membrane of the mast cell or basophil to rupture, releasing histamine, leukotrienes, eosinophil chemotactic substances, protease, heparin and platelet activating factors. These agents work together to cause a dilation of the local blood vessels, attraction of eosinophils and neutrophils, increased permeability of the capillaries and loss of fluid into the tissues, damage to the local tissues, and contraction of the local smooth muscle cells. These are the cellular elements that create the symptoms of allergy. Urticaria and eczema occur when this process is triggered in the skin and is unable to be shut down again because the cascade of events carries with it too much momentum.

Urticaria or hives refers to a solid edema of the skin related to a hypersensitivity that may be confined to one small area of the body or could include the entire body. Urticaria is classified as anaphylaxis if several organ systems are involved, especially the structures around the throat. In acute urticaria, a precipitating agent can usually be uncovered by interviewing the patient: a new medication, a different food, an infection (bacterial, viral, fungal, or parasitic), or a change in environment. Chronic stress seems to play a substantial role in the etiology of most of these cases. Essentially, some antigen is presented; IgE is created to deal with that antigen and mast cells and basophils are destabilized and torn up as the antigen antibody complex comes across the membrane. This will account for an acute, first episode of urticaria. Chronic urticaria becomes a type of learned response on the part of the body, that is, the system survived the previous onslaught(s) by utilizing this strategy, and so it continues to employ it. Chronic urticaria loses its dependence on IgE and can be triggered by a multitude of agents. Treatment involves initially elimination of the offending agent, if known, and stabilizing mast cells so that they are less likely to blow off histamine, over time a tolerance can develop. Conventional medical treatment employs antihistamines and steroids, these are agents that suppress the bodies desire to rid itself of a potential invader and usually the body will resume its process when those agents are gone[21]. Botanically derived flavonoids can play a major role in decreasing the fragility of mast cells and basophils, by making the membranes of these structures more stable, requiring a greater amount of stimulation to cause them to destabilize and release their histamine soup. We have enjoyed a measure of success with a powder made from equal parts Amalaki fruit (*Emblica officinalis*), Gambir

(*Uncaria gambir*), turmeric (*Curcuma longa*), and Licorice (*Glycyrrhiza glabra*) dosed at 1 tsp. 3-4 times a day with patients suffering from chronic urticaria with fairly good results. The trick with a lot of these patients is keeping them from becoming sensitive to the agents that we are employing in order to help them resolve the condition. This process requires great flexibility on the part of the practitioner and is not always amenable to pre-packaged products. I encourage these patients to increase their consumption of colorful fruits and berries, especially darker berries and yellow/orange fruits and vegetables. Fish oil can be immensely helpful, I recommend at least a gram, three times a day. An elimination/rotation diet is critical for these patients and a diet/exposure diary can be most enlightening for uncovering offending agents in the environment. I have found that in some desperate cases, the tricyclic antidepressant Doxepin, will help them sleep, and provides stability to the mast cells and often can change the course of chronic urticaria within a few days. I use a very small dose, 5 mg before bed--where a usual adult dose might be 25-50 mg.

A skin rash is dermatitis, literally "inflammation of the skin" which can manifest from contact with an irritant or allergen, contact dermatitis, or from some other sensitivity happening in the system that is manifesting with skin symptoms, termed atopic dermatitis.

Contact dermatitis tends to be an itchy and inflamed skin disease, generally caused by an inflammatory response to contact with some ingredient or object that a person is either allergic to or that is irritating to the skin. Contact dermatitis can occur anywhere on the body that comes in contact with allergens or irritants, including hands, feet, scalp, face, arms, legs, chest, abdomen and genitals. In the case of irritant dermatitis, the goal of therapy is to remove the irritant and soothe the skin. Irritants may damage normal skin or worsen an existing dermatitis. Weak irritants may include soaps, detergents, acetone and even water, strong irritants include acids, alkalis, phenol and other chemical exposures, it can take weeks or months of exposure to a weak irritant before symptoms manifest, it could take a single very brief exposure to a strong irritant to initiate symptoms. Washing the irritant away and the use of clay or charcoal drawing poultices can be very effective for these types of dermatitis. Treatment involves removing the irritant and triaging the wound, in the case of straightforward contact dermatitis--consider a battery acid burn, or some soap left to dry behind your ear. I recommend Dr. Bronner’s aloe castille baby soap for washing and a comfrey goo or flaxseed meal poultice placed on the area to soothe it. These types of dermatitis will generally resolve within 5-7 days. Irritant contact dermatitis is more closely related to direct chemical injury to the epidermis which happens when skin cells are damaged by exposure to irritating substances, like soaps, hair dye, and chemical solvents. The immune system is activated, whether the ingredient causes an allergy or not is somewhat irrelevant and is related to the quality of the skin barrier. You can even develop contact dermatitis from things you’ve been in contact with for years or your entire life, repeated exposure of compromised membranes to irritants, tends to produce allergy mediated responses to those irritants so that later, minor exposure creates much more severe symptomatology.

Seborrheic dermatitis is an example of a contact dermatitis that has some allergic potential in that it is the oily secretions that often exacerbate the condition, the rash usually develops on the scalp, armpits or pubic hair. On darker skin tones, the rash often looks pink, slightly purple, or lighter than the surrounding skin. People with lighter skin tones tend to have a red, raised rash. Dandruff may be considered a milder form of seborrheic dermatitis. The two conditions are similar in that they both cause white-to-yellowish scales and flakes, which may be dry itchy or greasy and itchy. Sometimes, both dandruff and seborrheic dermatitis can be treated the same way. Dandruff confines itself to the scalp where seborrheic dermatitis can appear anywhere on the body, it is also more inflammatory than dandruff, with more swelling and discoloration. When a baby has seborrheic dermatitis, it usually appears on the scalp and is called cradle cap. Babies can also develop seborrheic dermatitis in other areas like the folds of their skin, chest, back, or diaper area. No matter where seborrheic dermatitis appears on a baby's body, it's usually still called cradle cap.

Allergic contact dermatitis specifically arises from an allergic immune reaction that can be immediate or it can be delayed by a day or two. When allergic contact dermatitis manifests, the skin becomes hyper-responsive to the presence of the irritant/allergen, so while we still get potential irritant damage, we also get local release of pro-inflammatory mediators, degranulation of mast cells releasing histamines and locally active proteolytic enzymes which create additional tissue destruction. Topically applied agents should be well strategized as the reactive membrane tends to acquire new things to become allergically sensitive to as they are slathered on the compromised field. Treatment of allergic contact dermatitis requires uncovering the offender, avoiding such and soothing the skin as mentioned above. Baking soda and Epsom salt baths can be very effective for the allergic component as they have a strong alkalinizing effect that neutralizes residual histamines in the skin, increase in peripheral circulation helps eliminate remaining allergens.

Allergens and irritants that cause contact dermatitis are constantly changing with new agents being pronounced every year that cause contact dermatitis, especially in cosmetics and personal care products. For the last several decades, both the North American Contact Dermatitis Group and the American Contact Dermatitis Society have named an "allergen of the year" to help both healthcare providers and the general public understand what products contain the featured ingredients and how to avoid them. Here are some of the most common allergens that lead to contact dermatitis:

Metals, such as nickel, gold and cobalt
Medicines like neomycin and glucocorticoids
Fragrance mixes such as myroxylon pereirae (balsam of Peru), commercial fragrance mixes
Rubber and latex products, such as gloves and balloons
Preservatives like formaldehyde and parabens

Personal care products containing the chemical methylchloroisothiazolinone or methylisothiazolinone
Borax, which can be used to make a “slime” toy for children
Lanolin from sheep, which can be found in lotions and clothing
Plants like henna or poison ivy
Hair dyes

A systematic review of scientific literature on toys that cause contact dermatitis identified several common offenders, including electronics (video game controllers), toy cars, costume jewelry, bicycles, slime and children’s clay. The most common ingredient causing contact dermatitis in children is nickel which can be an allergen for both children and adults and is often found in toys and electronic devices like cell phones, iPads and computers.

Eczema is derived from the Greek word meaning “to boil over” an apt description of the presentation of most cases of eczema. Eczematous dermatitis has many different forms, it can be related to contact with foreign material (this can be allergic or irritant), it can be atopic, seborrheic, exfoliative, nummular or dishydrotic. This is by no means a comprehensive list but shows that there are numerous manifestations of this complex condition. Atopic dermatitis is the most common manifestation of eczema, and, for many, it becomes a chronic, recurrent skin condition. It is the skin manifestation of allergic tendency and is closely related to asthma and hay fever. There are many different predisposing factors for an individual to develop atopic dermatitis, not the least of which is heredity. There is a documented abnormality of cyclic nucleotide function in these patients where cAMP levels are reduced due to higher levels of cAMP phosphodiesterase activity and lack of prostaglandin precursors. This results in excessive histamine release and diminished immune function. Elevated serum IgE levels and decreased IgA levels are also present in these patients[22].

Atopic dermatitis is the most common form of eczema and affects people of all ages, it causes dry, itchy and inflamed skin. It's common in young children but can occur at any age. Atopic dermatitis is long lasting (chronic) and tends to flare at times, often related to stress. It can be irritating but it's not contagious. Moisturizing regularly and following other skin care habits can relieve itching and prevent new outbreaks. The vast majority of atopic eczema patients that I see have a chronic condition which originated in their bowel and is manifesting on the skin as a type IV hypersensitivity reaction. Typically, the lining of the bowel has been compromised, either from infection, chronic stress, use of NSAIDS, or abuse of irritant laxatives. Over time the patient develops small patches of eczema which eventually get larger, prompting the patient to seek some sort of treatment. Once the mucus membrane of the bowel becomes compromised, it is only a matter of time before the permeability of bowel becomes altered due to inflammation. Larger particles of relatively undigested food are allowed to pass across this membrane; they become perceived as foreign invaders/allergens and become covered in IgE antibodies on their way through. The presence of the IgE antibodies causes a wide scale disruption of mast cell membranes, allowing the release of histamine soup and more

tissue damage ensues. Some percentage of the circulating antibody-antigen complexes is able to reach the systemic circulation and will cause destabilization of mast cells and histamine release wherever they might end up, the surfaces of the body are the usual sites, skin, lungs and sinuses.

Treatment begins with reconditioning the bowel lining, soothing and protecting it while avoiding known allergens. A rotation/elimination diet can be very effective with increased intake of high flavonoid containing foods, yellow and orange fruits and vegetables and dark berries. Licorice tincture, powder or pills can be helpful in minimizing inflammation in the bowel itself. The amino acid glutamine is the principal fuel for small intestine enterocytes and epithelial tissue. It is the most abundant amino acid in the bloodstream and is considered a "conditionally essential" amino acid, as there are times where demand cannot be met by mobilization from other tissue stores. The lungs and skeletal muscle are the major producers of circulating glutamine, and the intestinal tract is the primary user, accounting for as much as 40% of total glutamine uptake by the entire body. Glutamine supplementation has been shown to increase the height of intestinal villi and mucosal thickness, as well as increasing secretory IgA secretion. I usually start a patient on 5 grams 3-4 times a day and work up to 15 grams twice a day. A digestive enzyme taken with meals can be helpful in reducing the size of the food that is being passed across the intestinal membrane and will often reduce symptoms accordingly. The addition of soluble fiber like flaxseed meal or oat bran is also very soothing to the intestinal membrane and has a normalizing effect on the bacteria that live there. Quercetin chalcone and Hesperidin methyl chalcone as water soluble flavonoids are helpful to improve the integrity of the mast cell membranes. Diamine oxidase, usually derived from legumes like the sugar snap pea (*Pisum Sativum)* and lentils (*Lens culinaris*), and also made in our kidneys, liver and small intestine, is one of the major enzymes involved in the metabolism of histamine...it is often very helpful in reducing excessive levels histamine in the body--it is dose in units and 200,000 units 3-4 times a day is usually a decent starting point. Cromolyn sodium, ultimately derived from the Ayurvedic herb *Ammi visnaga*, is a topical mast cell stabilizer that has an effect for about six hours and is useful to prevent release of histamine when some offender enters the system. We purchase this material as a bulk powder and reconstitute it ourselves to 2% solution and dosed at 5 ml or 100 mg three times a day, prior to meals.

The use of comfrey goo topically on the eczematous areas can be very helpful. Put chopped comfrey root and a small amount of water in a blender, blend until well mixed and apply the goo to the area. Cover with flannel or bandage material and leave on overnight, by morning the rash is generally quite a lot better. Alterative, lymph-moving tinctures are excellent forms of intervention and can often be curative. Blue flag *(Iris versicolor*) 10 drops three times a day can be used as a lymphatic alterative to help restore proper lymphocytic response to food allergens. The idea is to increase IgM and IgG antibodies in the serum over IgE antibodies.

Occasionally a patient will present who has either had long-term steroid therapy or has such an adrenal insufficiency that steroid intervention might seem necessary (very fatigued, very inflamed patients form whom no intervention seems to be making a

difference). This topic is a hotbed of philosophical controversy. I find that the pharmaceutical steroidal preparations are very heavy handed and do not allow reasonable recovery of adrenal function for the patients that they are used on, this is even true when these patients are tapered off of these agents. My favorite alternative is a 'Licort' cortisol tincture, I produce this in my office by adding 30 grams of micronized cortisol acetate to 1000 ml of a 1:1 fluid extract of *Glycyrrhiza glabra*, this produces a tincture that contains 1 mg of cortisol per drop. According to Dr. Jeffries, the author of the text, "Safe Uses of Cortisol", the average daily equivalent of cortisol output for most people is 25-30 mg. When a patient is given more than this quantity it can be too suppressive and disruptive of normal physiology. This is not to say that some people aren't able to produce quantities of cortisol that are far above this level, just that from a supplementation basis, we may not want to risk suppression or disruption in our interventions. I have found that replacing an existing steroid regimen with an equivalent dosage of licort makes it far easier to wean the patient down later, with very little to no ill effects after discontinuation. For those patients who seem to need some kind of cortisolic augmentation, I will add the cortisol tincture to their existing botanical preparation and dose it 5 drops 4 times a day. I find that it mixes quite well with any preparation that also contains licorice, for all of the reasons that we discuss when we talk about licorice.

There is evidence that atopic and contact dermatitis can coexist and that patients with the atopic form may even be more sensitive to allergens and irritants that cause local contact dermatitis. The current thought among researchers is that active atopic disease — with inflamed skin and a compromised skin barrier — can result in an increased possibility that allergens and irritants will trigger even more inflammation. When both are present, treatment for either can be less effective because the patient may continuously be exposed to factors that cause rashes associated with contact dermatitis. The number one goal for preventing and treating contact dermatitis symptoms is to avoid contact with the culprit agent (or irritant).

I really appreciate the simple efficacy of hydrogels for dermatitis. A hydrogel is any swellable network of polymers that exhibit 3-dimensional, hydrophilic, insoluble structures composed of crosslinked polymeric chains that attract aqueous solutions without disintegration[23]. Hydrogels are the slippery slimy components widely present in plants like *Aloe*, *Ulmus, Comfrey* and *Astragalus* and can be synthesized from natural or synthetic polymers. Functioning as a physical barrier, hydrogels provide protection for the wound bed against external contamination while creating a moist environment that facilitates wound repair. Most hydrogels are immunologically inert, and their high water content provides topical cooling and soothing effects, making them particularly suitable for burn wound dressings. *Aloe vera* is probably the most universally applied hydrogels for contact dermatitis--it seems to make intuitive sense to put a cooling soothing gel on a red and inflamed section of skin. We make an aloe/hyaluronic acid hydrogel for different manifestations of dermatitis that consists of 10 mg/ml hyaluronic acid in distilled water mixed into a gel and then mixed 50/50 in an aloe gel, for chronic conditions, I find that the addition of ceramides (3%) and glycyrrhizinate (also a gel former) at 1 mg/ml are often very helpful in a topical application. *Pilocarpus jaborandi*, 4 drops 3 times a day,

acts as a diaphoretic, helping to promote expulsion of metabolic debris through the skin and increasing IgA secretion to the surfaces of the body; yarrow, eupatorium and ginger can be used similarly. *Coleus forskohlii* can also be employed as an agent that helps promote cAMP over cGMP in the body and can be very helpful in resolving acute eczema attacks. Liver function is extremely important in promoting cAMP production, the classic liver tonifying herbs like *Arctium, Taraxacum, Trifolium* and *Melilotus* can be very helpful in this regard.

Psoriasis is a chronic, proliferative and inflammatory condition of the skin that is characterized by pinkish-red plaques covered with silvery scales, particularly over the extensor surfaces of the body, the scalp, and the lumbosacral area, also affecting the joints (psoriatic arthritis). There are several subtypes, with the "plaque type" being the most common (85-90% of cases) with pinkish-red plaques with white silvery scales presenting on the trunk, extremities, and scalp[24]. Removal of the psoriatic scales creates pinpoint bleeding called the 'Auspitz sign' and can be used to confirm the diagnosis. The pathophysiology of psoriasis involves infiltration of the skin by activated T cells which stimulate the proliferation of keratinocytes. This dysregulation in keratinocyte turnover allows layers of skin to build on one another resulting in the formation of the thick plaques, inflammation produces epidermal hyperplasia (increased production of skin cells) which fail to secrete adequate amounts of lipids, resulting in the flaky and scaly skin that is typical of psoriasis. Conventional topical therapy involves the use of emollients and moisturizers to improve barrier function and retain the hydration of the stratum corneum. Topical agents that have found clinical use include coal tar, dithranol, corticosteroids, vitamin A and D analogs. Because there is an autoimmune element to the manifestation of psoriasis, immuno-suppressants are often used for short-term management and include methotrexate, cyclosporine and fumarates. Biologicals are manufactured proteins (cytokine inhibitors, monoclonal antibodies) that interrupt the immune process in psoriasis and include infliximab, adalimumab, etanercept, and interleukin antagonists. The language leaves clues, anything that ends in the suffix -mab, is a monoclonal antibody. Before starting any biological agent, the patient should be worked up for tuberculosis and hepatitis. There is a serious risk of infections in these patients and all precautions should be taken that the patient is not severely immunocompromised.

Psoralen is the parent compound in a family of naturally occurring organic compounds known as the linear furanocoumarins, structurally related to coumarin by the addition of a fused furan ring, and is considered as a derivative of umbelliferone. Psoralen occurs naturally in *Ficus carica* (fig) (the most abundant source of psoralens), they are also found in smaller quantities in *Ammi visnaga*, *Pastinaca sativa* (parsnip), *Petroselinum crispum* (parsley), *Levisticum officinale* (lovage), *Foeniculum vulgare* (fennel seeds), *Daucus carota* (carrot), *Psoralea corylifolia* (babchi), *Apium graveolens* (celery), and essential oil of *Citrus bergamia* (bergamot oil). Psoralen is a mutagen, it intercalates into DNA (replaces hydrogens in the nucleoside bases) and on exposure to ultraviolet radiation (UVA) can form interstrand cross-links (ICL) with thymine, which inducing apoptosis. Psoralen plus exposure to UVA is called PUVA therapy, and is used

to treat hyperproliferative skin disorders like psoriasis and skin cancer, and PUVA treatment itself leads to a higher risk of skin cancer. Phototherapy includes PUVA therapy as well as NBUVB (Narrowband UVB light) with a range of 311 nanometers to 313 nanometers. NBUVB is equally effective without the side effects of psoralen like gastrointestinal upset, cataract formation, and carcinogenic effects. It can safely be given to children, pregnant and lactating females, and even older patients. Patients with psoriasis should avoid all skin trauma for fear of inducing a Koebner reaction, new psoriasis plaques at sites of injury. In addition, psoriatic patients should avoid the use of beta-blockers, chloroquine, or NSAIDs and alcohol because of the risk of developing fatty liver.

A number of studies have explored the efficacy of dietary fish oils as a safe therapy for various skin conditions due to their anti-inflammatory, anti-chemotactic and moisturizing properties. Inhibition of inflammation helps to improve the barrier function of the skin, induces stratum corneum maturation and differentiation, inhibits proinflammatory eicosanoids, improves lamellar body formation, contributes to cytokine suppression, and lipoxygenase inhibition[25]. 1000 mgs of a good quality omega 3 fatty acid three times a day is a good starting place.

The vitamin D system is historically well known for its role intracellular calcium and bone metabolism, more recent evidence over the past 20 years has shown a wide variety of biological activities for vitamin D, including cell differentiation, control over cellular development, immunomodulation, and regulation of many other hormonal systems. Deficiency has become more and more prevalent, or we are looking more frequently at those levels and maybe they were always on the low side for some locations. Dietary sources are classically animal products like cod liver oil, salmon, tuna, beef, eggs, and chicken breast, as well as milk, yogurt, butter and cheese (especially cheddar), and some mushrooms contain vitamin D2. The epidermal skin cells are unique in that they synthesize vitamin D and possess the enzymatic mechanism to convert it into its active form, calcitriol (1,25(OH). Vitamin D has an effect on both B and T cells, as well as the adaptive immune response by altering B-cell activity, limiting differentiation and proliferation and inducing apoptosis, it also causes a modulation of immunoglobulin production, including autoantibodies. Vitamin D affects T-cell activity by inhibiting T helper (Th) cell proliferation, as well as supporting a transition from a pro-inflammatory into a more tolerant immunological condition through several mechanisms, including inhibition of the cytokine production required for Th17 and Th1 differentiation, encouraging T cells to release IL-10, which is an anti-inflammatory Th2 cytokine, it also decreases the intensity of class II molecules of the main histocompatibility complex that affect dendritic cells. These impacts on keratinocyte development and proliferation are central to vitamin D's function in the treatment of psoriasis. Dosage is 800-2400 ius/day of cholecalciferol and subject to testing blood levels.

Vitamin E is a powerful lipid-soluble antioxidant found in all cell membranes that exists in eight forms in nature, generally as mixes of tocopherols and tocotrienols, the tocopherols are the most physiologically active, the tocotrienols play a significant supportive role, it is important that we utilize a mix of tocopherols and tocotrienols as D-

alpha-tocopherol is inherently unstable by itself, and, D,L-alpha tocopherol shouldn't be eaten at all. Patients with psoriasis tend to a statistically significant decrease in vitamin E plasma levels when compared to their peers, and those with the most severe presentation had the lowest vitamin E levels, which also correlated with the highest alcohol consumption. Psoriasis, alopecia areata, and vitiligo patients generally have lower tissue and blood vitamin E concentrations than healthy controls. Our clinic finds tha a combination of vitamin E (mixes of tocopherols and tocotrienols--AC Grace Co and Life Extension make decent products), selenomethionine, and coenzyme Q10 is very helpful for people with psoriasis and psoriatic arthritis.

Aloe barbadensis is one of our best and oldest first-aid interventions for superficial wounds and burns, it belongs to the *Liliaceae* family, and has a long history of use worldwide in traditional medicine. References to the medicinal use of the Aloe plant date back 4000 years, with the first inscriptions mentioning the plant found on Sumerian clay tablets from 2100 BC. The Egyptian Ebers Papyrus of 1552 BC also mentions therapeutic uses of the plant, including internal use as a laxative. According to Greek legend, in 333 BC, Aristotle advised Alexander the Great to capture the island of Socotra in the Indian Ocean for its famous Aloe vera plantations, which would be of great help in treating his wounded soldiers. Aloe leaf gel began to be industrialized and commercialized in the 1950s, and the global Aloe market size is projected to grow from $2.65 billion in 2023 to $4.55 billion by 2030. Aloe vera is a plant that is often cultivated in people’s homes throughout the world as a natural compound intended for widespread use by both adults and children and recognized in clinical practice as a dependable tool for wound healing. It is universally associated with the treatment of skin injuries such as cuts, burns, frostbite, radiation, and electrical injuries and a large part of the reason why Aloe gel is able to be helpful is due to its physical properties as a hydrogel. Acemannan and Aloe-emodin are antibacterial active ingredients that have been shown to reduce the severity of redness and scaling in patients with psoriasis. Aloe vera has shown immunomodulatory, antioxidant, anti-inflammatory, anti-fungal, and anti-tumor properties, it provides hydration to the skin increases collagen activity. Several studies have shown aloe extracts to be as effective as topical steroids in patients with psoriasis, with virtually no side effects.

Mahonia aquifolium, Oregon grape, is an evergreen shrub in the Berberidaceae family, it is indigenous to the United States and has a long history of being employed to treat a variety of inflammatory skin conditions. Berberine, as an extracted alkaloid, has been demonstrated to have anti-inflammatory effects in the body which include the downregulation of lipoxygenase enzymes, protection from lipid peroxidation, inhibition of T cells infiltration into psoriatic lesions, a decrease in cyclooxygenase activity, a reduction in IL-8 and prostaglandin E2. Berberine modulates rapid and uncontrolled cell reproduction by intercalating (replacing hydrogen) into DNA, preventing replication of DNA and cell proliferation. Other alkaloids present in Mahonia also inhibit lipoxygenase, such as oxyberberine, jatrorrhizine, corytuberine, and columbamine and contribute to anti-inflammatory action)[26]. A 10% *Mahonia aquifolium* hydroethanolic extract has been shown to suppress IL-8, IL-1, T cells, TNF-alpha, and TNF-beta, each of which plays a

significant role in the progression of psoriasis. We use it internally as well as topically (it does tend to make things (everything) yellow), most of our other berberine plants are interchangeable, depending on your region. Here in the southwest, we are more likely to use Algerita root (*Mahonia trifoliata)*, Kofa's barbery (*Berberis harrisonia),* or red barberry (*Berberis haemotocarpa)*.

Psoriasis is a complex metabolic condition, our approach with patients is to encourage a dramatic reduction in their simple sugar intake--in fact, taking total carbohydrate intake down to 50 grams or less can seem practically miraculous in some patients. Focusing on anti-inflammatory fats and avoiding hydrogenated oils and fried foods also makes a huge difference for many people. Ensuring adequate sleep and regular nutrition, tending towards a less is more approach and pulling families of foods out of the diet for a month to 6 weeks at a time can be a helpful calibration for how certain foods may be influencing our inflammatory processes. I have personally seen PUVA therapy work for people, while loading them with agents to hopefully prevent skin cancer later on. Topical salves (beeswax and macadamia nut oil) with 5-6% salicylic acid can be very helpful for the reduction of the superficial scaling, a 10% saffron hydrogel (10 mg/ml hyaluronic acid in saffron water decoction) with an added 3% ceramides (Ceramide x-blend) is often very very helpful once the scaling has been removed, we have them apply it 2-3 times a day.

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