Allergic Contact Dermatitis versus Irritant Contact Dermatitis

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Dr. Skotnicki has been practicing in the Toronto area since 1997. She is currently on staff at both Woman’s College Hospital and St Michael’s Hospital as a consultant Dermatologist.

She is a member of the Occupational Disease Specialty Program at St Michael’s Hospital where she is responsible for seeing complex Allergic and Occupational Skin Disease. She is one of a handful of dermatologists in Canada with a subspecialty interest in Allergic Contact Dermatitis and Patch or Allergy Skin Testing. She is an also a consultant for the Workman’s Safety Insurance Board. Considered a leading expert in Allergic Skin Disease she is often asked to speak or give advice on Cosmetic and Toiletry products.
Active in research and teaching, she has been teaching University of Toronto Dermatology, Allergy and Family Medicine residents at her clinic at St. Michaels Hospital since 1999. She is also a member of CREOD the Centre for Research Expertise in Occupational Disease and has published articles on different aspects of Allergic Skin Disease.

Her memberships include The Canadian Medical Association, Canadian Dermatology Association, American Dermatology Association and American Contact Dermatitis Society.

Dr. Skotnicki is a contributing author for Allergy Living Magazine where she answers patient's questions quarterly. She has also been quoted in Flare, Glow and Toronto Life Fashion. Her appearances include Breakfast TV, CTV, Shop Toronto and CBC radio. She has also been a consultant for Oil of Olay, Neutrogena, Lubriderm and is an active member of the Dove Advisory Board. She was named 2007 and 2008 spokesperson for KAO, a company that represents Jergens, Biore and Curel.

Considered to be a leading Dermatologist with interests in Allergic Skin Disease, Cosmetic Dermatology and General Dermatology she started the Bay Dermatology Centre in 2006 in an effort to have a full service Dermatology Centre with a focus on the patient and not procedures.

This medical discussion paper will be useful to those seeking general information about the medical issue involved. It is intended to provide a broad and general overview of a medical topic that is frequently considered in Tribunal appeals.

Each medical discussion paper is written by a recognized expert in the field, who has been recommended by the Tribunal’s medical counsellors. Each author is asked to present a balanced view of the current medical knowledge on the topic. Discussion papers are not peer reviewed. They are written to be understood by lay individuals.

Discussion papers do not necessarily represent the views of the Tribunal. A vice-chair or panel may consider and rely on the medical information provided in the discussion paper, but the Tribunal is not bound by an opinion expressed in a discussion paper in any particular case. Every Tribunal decision must be based on the facts of the particular appeal. Tribunal adjudicators recognize that it is always open to the parties to an appeal to rely on or to distinguish a medical discussion paper, and to challenge it with alternative evidence: see Kamara v. Ontario (Workplace Safety and Insurance Appeals Tribunal) [2009] O.J. No. 2080 (Ont Div Court).
INTRODUCTION

Contact Dermatitis (CD) includes both Allergic and Irritant Contact Dermatitis as distinct entities. Within Irritant Contact Dermatitis there are several subtypes. CD is one of the most common skin diseases, with a lifetime prevalence of between 1-10% in industrialized societies. CD is characterized by a chronic course with relapses upon contact with allergens or irritants. Management is complicated by lack of effective and reliable diagnosis. There is no cure for CD. CD represents the most common cause of occupational skin diseases in industrial countries and therefore is of major importance in occupational medicine.

DEFINITIONS:

IRRITANT CONTACT DERMATITIS (ICD)

ICD can be defined as a non-immunological, non-specific reaction of the skin to an irritant. It is now recognized that this definition is too simplistic. New evidence does suggest that our immune system plays a role in eliciting ICD, via multiple parallel pathways. In essence an irritant causes toxicity or damage to epidermal skin cells which results in inflammation by natural immunity. Natural immune responses comprise of cells or mechanisms that defend a person from infection or damage, in a non-specific manner. The response to damage or infection is nonspecific and this immune response does not confer long-lasting or protective immunity to the host. There is some evidence that endogenous (genetic predisposition) factors influence the susceptibility to ICD. These include age, sex, anatomic site and history of eczema including atopic (genetic) eczema. Susceptibility to irritation decreases with age, and is more common in women and, on the face.

Patients who have an “atopic diathesis” which means a genetic predisposition to develop one or all of the following: hay-fever, asthma and atopic eczema, are more susceptible to irritants on the skin, airway and mucosal surfaces (eyes, nose, mouth). An example would be smoke irritating and exacerbating asthma and water and soap irritating eczema. If you don’t have this genetic predisposition in your personal or family history, you are less likely to get irritated by these exposures. Some studies dispute that a history of hay-fever and or asthma will lead to greater skin irritation in the absence of atopic eczema.
Types of ICD:

The literature varies but a recent review described 10 subtypes of ICD.\(^5\)

**Acute Irritant Contact Dermatitis**

Acute ICD is caused by exposure to a potent irritant such as a strong acid or alkali. The skin response includes erythema (redness of the skin due to capillary dilatation), edema and possible necrosis (death) of skin cells. This occurs very soon after exposure. The healing occurs rapidly after the exposure and often takes up to 4 weeks. The prognosis is very good but scarring can occur. An example would be exposure to wet cement causing a “cement burn”.

**Delayed Acute Irritant Contact Dermatitis**

Some chemicals, such as dithranol and benzalkonium chloride, have the potential to cause a delayed inflammatory response, approximately 8-24 hours following the initial exposure.\(^6\) The symptoms are similar to acute ICD, and the prognosis is good. It is important to have knowledge of this subtype as the delayed nature of the dermatitis may lead the clinician to misinterpret the eruption as ACD.

**Irritant Reaction**

Patients exposed to wet work, such as hairdressers, may develop erythema, scaling, vesicles or erosions on the backs of their hands with repeated exposures. Hardening of the skin occurs after healing, and the prognosis is generally good. If exposures are not decreased however cumulative ICD may develop.

**Subjective or Sensorial Irritation**

Contact with an irritant produces a sensory discomfort, usually manifesting as a stinging, burning or itchy sensation, in the absence of clinical and histological (the structure of cells) evidence of skin lesions. The threshold to develop this reaction varies between people. Lactic acid and propylene glycol are good examples of subjective irritants. The outcome from this irritant reaction is good.

**Non-erythematous Irritation**

This subtype refers to irritation of the skin which shows some pathologic changes but the skin looks normal.\(^7\) The symptoms are similar to those of subjective irritation. Individuals experience discomfort with many chemicals. This form is believed to commonly occur with exposure to consumer products that have a high content of surfactants, such as cocamidopropyl betaine. The prognosis is variable. This subtype of ICD is suggested to play a role in people who develop “sensitive skin”. Both neural and vascular components, contribute to the discomfort.
Cumulative Irritant Contact Dermatitis

This is the most prevalent type of ICD. It is the result of multiple subthreshold insults induced by weak irritants. The repetitive nature of the irritants does not allow the skin to recover leading to persistent dermatitis or chronic dermatitis. The clinical features include redness and dryness followed by a thickening of the skin called hyperkeratosis. The threshold varies again between individuals. It is very important to remember that with Cumulative ICD the symptoms do not occur immediately after the exposure. This is similar to Allergic Contact Dermatitis (ACD) and therefore the two often cannot be distinguished without the help of Patch Testing (see page 18 for description). Another very important point is that exposure to weak irritants occurs not only at work, but also at home, adding to the complexity of identifying contributing factors.

Traumatic Irritant Contact Dermatitis

This is an uncommon form of ICD. It occurs after a very acute or sudden exposure to an irritant similar to a chemical burn. It is characterized by incomplete healing of the original insult followed by a nummular or circular eczema-like lesion. It has a chronic course and is sometimes recalcitrant (stubbornly resistant) to therapy.

Pustular and Acneiform Dermatitis

Exposure to metal, tars, oils and chlorinated agents can result in a pustular (relating to small, circumscribed elevation of the skin, containing purulent material) and acne form (resembling acne) dermatitis especially in atopic (allergic) patients (patients with Atopic Dermatitis, Hay fever and or Asthma).

Frictional Dermatitis

Frictional Dermatitis is caused by a shearing force acting horizontally to the surface, rather than pressure or temperature. It can be defined as an eczematous process in which physical frictional trauma contributes to the cause of a dermatitic process. It is confined to the locations of frictional trauma. It is common on the hands, especially the dominant hand, but can occur anywhere on the skin where repetitive frictional forces occur. It is under-diagnosed due to the lack of recognition of the potential for physical friction to induce eczematous changes in the skin. Repetitive friction can produce redness, scaling, occasionally vesicles (small, circumscribed elevation of the skin containing fluid.) and hyperkeratosis (thickening of the skin) and fissures or cracks in the skin. These changes are most common on the fingers especially the sides and tips as well as the palms. It is often delayed in onset sometimes by years. Published occupations associated with Frictional Hand Dermatitis include repetitive handling of small metal components, paper, plastic, cardboard, fabric and driving.

Prognosis usually is good. Cases of Frictional Dermatitis improve once removed from the friction. However, in an occupational setting, modification of the job to decrease frictional forces cannot always be accomplished. Protective Equipment, in particular
gloves, are an important treatment consideration and one where more study needs to be done. Gel Impaction Gloves have shown some promise at prevention of shearing frictional impact on the skin and improvement of Frictional Hand Dermatitis on the job.\textsuperscript{10}

**Picture 1:** Frictional Hand Dermatitis in Custodian
Hyperkeratotic Hand Dermatitis

Hyperkeratotic Hand Dermatitis can be classified as a subtype of ICD but irritation does not always have to be a precipitating event. It was found in two different studies to represent about 2% of all hand dermatoses. It is most common in males aged 40-60. This condition is clinically similar to Frictional Hand Dermatitis and is characterized by hyperkeratotic plaques symmetrically on the proximal or middle parts of the palms and/or soles. Itch can be a factor and painful fissures usually develop. There is usually an absence of psoriatic (eruption of reddish, silvery-scaled round flat raised skin) nail or scalp changes. Over 50% of patients who develop Hyperkeratotic Hand Dermatitis have hard manual work at the time of onset. It has been postulated that chronic mechanical trauma contributes to the cause. The occupations associated included construction workers, forest workers, machinists, mechanists and paper handlers. Prognosis is very poor. Unlike patients who suffer from Frictional Hand Dermatitis these patients do not improve once removed from work environment and often have permanent disability.

Picture 2: Hyperkeratotic Hand Dermatitis in Assembly worker
Allergic Contact Dermatitis

Allergic contact dermatitis (ACD) is caused by a wide range of chemicals (potential allergens) after prolonged or repeated contact with the skin. It is less common than ICD and is estimated to cause about 20% of occupational skin disease.

Its prevalence in Europe is about 20% of the population; the most common allergens are nickel, fragrances and preservatives. The most frequently and consistently reported agents in cases of allergic occupational contact dermatitis include cobalt, chromates, cosmetics and fragrances, epoxies, nickel, plants, preservatives, resins and acrylics.¹²

By definition ACD is an adaptive immune response toward chemicals penetrating the skin. It is a type IV, delayed or cell-mediated immunological reaction. In common with other forms of allergy, ACD occurs in a two phase process. Sensitization occurs on the initial exposure to the chemical resulting in immunity to the allergen followed by elicitation on re-exposure. If exposure to the specific allergen (chemical substance) is most likely to occur in an occupational setting, then the resulting dermatitis is considered to be work-related. Most importantly, in ACD most contact allergens produce a sensitizing allergic reaction in only a small percentage of exposed individuals, unlike ICD. Numerous factors may contribute to the allergen sensitization including temperature, humidity, genetic predisposition, and previous or concurrent skin irritation. The most important factor in an individual has been shown to be recent or present skin damage from trauma or irritation at the site of contact of the potential allergen. This often explains why some workers have minimal skin issues for months to years then “suddenly” develop an allergic contact dermatitis.

Patho-Physiology of Contact Dermatitis Irritant Contact Dermatitis

ICD is most commonly known to be a non-specific immunologic response. However, as mentioned the immune system is felt to now play a role. A genetic polymorphism shown in ICD may lead to a novel approach to detect susceptibility to ICD.¹³ Essentially, a chemical irritant or toxin damages the keratinocyte. The keratinocyte is the main cell in the outermost layer of the skin, the epidermis. This damage induces the activation of inflammation mediators. These mediators turn on genes in the keratinocyte which result in the production of proteins. These proteins called cytokines (proteins derived from cells) activate the immune system in the skin, in particular the T cells. All of these mediators and cells result in dilation (opening) of blood vessels in the area. This results in swelling and infiltration of more inflammatory and immune cells to the area.

Allergic Contact Dermatitis

ACD is a specific immunologic response. This is also called an adaptive immunological response. Adaptive immunologic responses are classified into 4 Types, based on the
Allergic Contact Dermatitis versus Irritant Contact Dermatitis

Gel and Coombs classification. ACD is a type IV delayed hypersensitivity immunologic response.

Classification of Hypersensitivity

In the early 1970s, P.G.H. Gell and R.A. Coombs proposed a method of classification of hypersensitivity. Their classification is as follows:

1. Immediate hypersensitivity.
   
   It includes three types:
   
   a. Type I (anaphylactic hypersensitivity). It involves IgE, mast cells, basophils and mediators that induce muscle contraction. This would manifest as urticaria, angioedema and anaphylaxis (shock).
   
   b. Type II (cytotoxic hypersensitivity). It involves IgG, IgM, complement, and the destruction of host cells.
   
   c. Type III (immune complex hypersensitivity). It involves IgG, IgM, complement, and the formation of antigen-antibody aggregates in the tissues.

2. Delayed hypersensitivity.

   Described as Type IV.

   d. Type IV (cellular hypersensitivity). It involves lymphokines and T-lymphocytes. This is seen in Allergic Contact Dermatitis.

ACD is mediated by T cells and not antibodies. The response occurs in two phases, initially a sensitization and then an elicitation response.

Sensitization

An allergen which is a chemical of low molecular weight is applied to the skin. The allergen is then taken up by a cell in the skin/epidermis called the Langerhan cell. This cell then presents the allergen to the immune system in the skin, the T cell. The presence or absence of specific T cell in the skin is most likely genetically determined. Once the allergen is recognized by the T cell both cells are activated and mediators are produced which leads to an increase in T cell proliferation. An expansion of specific T cell clones is produced. These T cell clones or memory cells recognize this allergen. The individual is now sensitized or primed to respond when these circulating T memory cells are exposed to the antigen.

Elicitation Phase

The second phase or elicitation of the delayed type of hypersensitivity occurs on re-exposure. The allergen is applied to the skin and the Langerhan cells present it to the immune system in the skin. The primed or memory T cells become activated and are
produced in greater numbers. Inflammatory mediators are produced, vessels dilate and swelling in the area occurs as well as infiltration of more cells.

**Summary of Patho-physiology**

Although ICD is not allergen specific and ACD is allergen specific the end result of both from a cellular level is similar. There is activation of immune T cells in the skin and inflammation resulting in dermatitis clinically in the skin.

**Causation**

**A. Endogenous Factors:**

Endogenous factors can influence the susceptibility to ICD and ACD.

**ACD**

- ACD is somewhat less common in young children however a recent increase in documentation of ACD cases in children is being seen.

- ACD requires a specific immune response to the allergen presented on the skin. The presence or absence of specific T cells in the skin is most likely genetically determined.

- Concurrent dermatitis of the skin is known to increase the risk of sensitization to a potential allergen, therefore patients who have suffered from Atopic Dermatitis in the past or present are more susceptible to sensitization by allergens.

- Mucosal Atopy which includes Asthma and Hay fever does not have such a clear cut impact on the susceptibility to potential allergens applied to the skin.

**ICD**

- Susceptibility to skin irritation decreases with age

- CD appears to be more common in women, It is not clear if this is due to an increased susceptibility to ICD in women or greater exposure of women to irritants, in particular wet work. Further studies need to be done.

- Skin penetration varies with anatomic region. The face is the most permeable; it is three times more permeable than the back.

- Patients with altered barrier skin function are more prone to ICD. Existing dermatitis, regardless of type, enhances reactivity to various irritants in areas of body not already involved with dermatitis. This is very clear for atopic dermatitis.
A study published in Immunogenetics in 2000 described the first non-atopic genetic marker for irritant susceptibility in normal individuals. Genotyping for the TNF-308 polymorphism may screen for individuals at risk of developing ICD.\textsuperscript{13}

\textbf{B. External/Exposure Factors:}

\textbf{ICD}

ICD can be caused by various irritants including chemicals and physical or mechanical irritants. The potential for irritancy of a substance is determined by its chemical and physical properties. The size, ionization and fat solubility determine skin penetration. Chemicals that are mild irritants will require repeat exposure. At high levels of exposure, many chemicals will act as irritants. The concentration, volume, application length and duration of exposure on the skin will determine the outcome. The penetration of an irritant will increase with exposure volumes and duration of exposure. This often comes into play with workers who work long hours over few days such as 3 days on and 4 days off which would increase the likelihood of developing ICD as the volume and duration of exposure would be increased with this type of shift.

1. Skin Irritants:
   Almost all chemicals have the potential to cause ICD. Identifying one cause is often not possible. The most important and common skin irritant is wet work.\textsuperscript{14} Wet work is defined as the exposure of the skin to liquid for longer than 2 hours per day, the use of occlusive gloves for longer than 2 hours per day or frequent hand cleaning.\textsuperscript{15} Occupations at risk include hairdressers, food handlers and health care workers. Wet work is the most common skin irritant followed by exposures to soap, detergents, solvents and oils and acid or alkali substances.

2. Physical or Environmental Skin Irritants
   Adapted from: (\textsuperscript{16})

   \textit{Low Humidity}
   Low environmental humidity enhances skin irritation. Air that is warm, dry and mobile can cause itch and even eczematous changes. Air conditioning results in low humidity in the workplace. An example is Airline stewardesses, particularly those on long haul flights. They give a history of recurrent facial eruptions that improve away from the air-conditioned environment of the aircraft cabin.

   \textit{Heat}
   Heat leads to sweating and is a particular problem from those working in a factory environment. Examples include workers close to welding equipment, molten metals and glass or those working close to furnaces or incinerators. Many of these patients
suffer from facial dermatitis. Sweating can also facilitate the skin penetration of allergens. Heat, sweat and occlusion of gloves or clothing can cause irritation.

**Metal Materials**
Chronic and repeated exposure to metal material handled by employee’s results in Frictional Hand Dermatitis. The dominant hand and fingers are the most commonly affected sites. Metal shavings and dust can also produce a facial dermatitis.

**Paper**
The majority of workers exposed to paper as a physical irritant work in offices, post offices and banks. The repeated sifting and distribution of paper cause friction and dryness of the finger pulps leading to chronic dermatitis.

**Tools**
Chronic low-grade trauma and friction to the hands from the use of tools can cause a variety of ICD responses. Workers from manufacturing, construction, woodworking, maintenance and repair industries were particularly affected. The hands are the most common site in these occupations. The mechanisms of irritation were usually friction and chronic trauma.

**Fabrics**
Repeat handling of fabrics by workers in the textile industry cause friction and desiccation (dryness) particularly to the hands. The fabrics commonly included implicated include polyester, nylon and wool. Repeat handling of fabric sacks by warehouse workers can be a problem. The fiber friction, rigidity and fineness all contribute to the increased risk of cutaneous irritation.

**Plastics**
Repeat handling of small plastic objects or bags can cause Frictional Hand Dermatitis. Plastic dusts can be generated by filing-down and finishing which can lead to facial ICD.

**Dusts**
Dusts can be produced by airborne particulate matter from wood, metals, plastics, cement and plaster. The dusts often spread over considerable distances to affect individuals not close by or immediately involved in the process. Workers commonly affected include carpenters and builders. Mechanical friction from sanding down these materials often causes chronic dermatitis on the hands and the resulting dust causes dermatitis on the face.

**Wood Materials**
Friction from handling unfinished wood products in the carpentry and construction industry can lead to Frictional hand dermatitis.
Rubber
Rubber bands are widely used in office and warehouses. Workers can develop dermatitis on their dominant hand. Rubber used in the grips of sports rackets can lead to a hobby-related or aggravated friction and pressure on the palms.

Heavy Machinery
Machine operators in factories often have to manipulate heavy pieces of equipment repeatedly, often as part of assembly work. This can result in repetitive mechanical trauma to the hands from heavy physical work and then frictional hand dermatitis.

Fiberglass
Fiberglass is a manufactured fiber made from silicon dioxide with various metals and other elements. Fiberglass can cause folliculitis, and irritant symptoms including itching without rash, burning of eyes, sore throat and cough. Curiously, not all workers are affected suggesting an individual predisposition. Fiberglass over a diameter of 3.5 ppm (parts per million) causes tissue irritation. Dermatitis usually occurs on exposed body sites due to contact with fiberglass dusts produced by grinding, sawing, and finishing. ACD can also occur to chemicals added to fiberglass manufacturing and should be ruled out in new onset cases of dermatitis.

C. Allergic Contact Dermatitis

ACD is caused by chemicals known as allergens. Allergens are small molecules that bind to carrier proteins in the skin. Several factors can contribute to allergen sensitization and include increased temperature, humidity and previous or concurrent skin irritation.

ACD develops with repeat exposure to chemical allergens. Workers may contact a potential allergen many times over many months without any problems and then suddenly become sensitized and develop intractable ACD.

Elicitation of ACD in sensitized workers will depend on the duration and frequency of exposure, region of application and the presence of irritants.

It is not possible to discuss all the potential chemical allergens associated with ACD. Textbooks in Occupational Dermatology usually break down potential irritants as well as allergens by occupation and discuss both within each occupation. However, discussion of the common allergen chromium will help illustrate the role of the allergen, exposure parameters, how it relates to the development of ACD and possible control measures.

Chromium
Chromium is a steel gray hard metal. It is used in metal alloys (stainless steel is an example), plating, leather tanning, paint, anticorrosives, ceramics and chemicals. Historically the most important cause of contact allergy to chromium has been occupational exposure to cement. Two forms of chromium act as allergens; these
include Trivalent Chromium and Hexavalent Chromium. The existence of each depends on the pH and temperature of the cement. Cases of ACD to chromium in cement date back to 1908 and 1925. Patch testing with Potassium dichromate 0.5% in petrolatum is used to diagnosis ACD to chromium. In 1950 it was determined that the Hexavalent Chromium allergen was the main cause of ACD from cement. In 1979, it was suggested that the addition of iron sulfate to cement would reduce the amount of water soluble Hexavalent Chromium in cement to 2 ppm. In Denmark in 1983, legislation was passed that made addition of iron sulfate to cement compulsory. Since then in Denmark the prevalence of ACD to chromium among construction workers have decreased. In countries, including Canada that did not pass this legislation chromium continues to cause ACD in construction workers. The European Union on January 17, 2005, restricted the marketing and use of cement containing more than 2 ppm hexavalent chromium.17

**Personal Protective Equipment**

Personal protective equipment includes gloves, sleeves, coveralls, masks/ respirators, glasses and protective footwear. It is possible for workers to have adverse reactions to coveralls, sleeves and respirators but these are rare occurrences. More commonly the glove supplied or chosen for protection is inadequate or even is the cause of ICD or ACD.

**Table 1**

<table>
<thead>
<tr>
<th>Material</th>
<th>Good Protection</th>
</tr>
</thead>
<tbody>
<tr>
<td>Latex</td>
<td>biologic material, water-based solvents</td>
</tr>
<tr>
<td>Nitrile</td>
<td>solvents, oils, greases, selected acids and bases</td>
</tr>
<tr>
<td>Vinyl</td>
<td>acids, bases, oils, greases, peroxides, and amines</td>
</tr>
<tr>
<td>Polychloroprene</td>
<td>acids, bases, alcohols, fuels, peroxides, hydrocarbons, oils, greases, and phenols</td>
</tr>
<tr>
<td>Polyvinyl alchohol</td>
<td>aromatic and chlorinated solvents, ketones, esters, methacrylate</td>
</tr>
<tr>
<td>Viton (Dupont)</td>
<td>Chlorinated and aromatic solvents, aliphatic, alcohols</td>
</tr>
<tr>
<td>Butyl</td>
<td>ketones, aldehydes and esters</td>
</tr>
<tr>
<td>Gel Impaction gloves</td>
<td>repeat mechanical/physical trauma</td>
</tr>
</tbody>
</table>
Mechanisms by which gloved hands can be exposed to harmful substances. There are 3 primary mechanisms by which gloved hands can be exposed to chemicals.¹⁹

1. Contamination
2. Permeation
3. Penetration

In conclusion, it is important that the chosen glove be changed at appropriate intervals to minimize the likelihood of permeation and penetration of chemicals. Gloves should be checked regularly for evidence of physical damage. Lastly, excellent glove donning and removal techniques must be used to prevent the contamination of the glove interior.

ACD to gloves
Certain allergens in rubber gloves can lead to ACD. This does not include latex allergen. Latex allergen does not cause ACD. Rubber accelerators including Carbamates, Mercaptobenzothiazole and Thiuram are the allergens associated with
ACD in rubber gloves. There are new and emerging allergens in rubber gloves. With the increase in use of other forms of rubber this will continue to increase and can only be detected by testing the worker to the gloves they use on the job in their raw form. This is done by cutting a small one inch piece of the glove, wetting it with saline and covering it on the back for 96 or 120 hours.

**Picture 4:** Positive Patch test to Glove Raw Material
Clinical Picture

It is difficult and often impossible to make a clinical diagnosis of ACD vs. ICD based on clinical appearance. Clinically the classifications of acute CD, subacute CD and chronic CD are of generic usefulness and do not distinguish between ICD and ACD. The clinical variations do not necessarily correlate directly with new, recent or chronic exposure to a substance because of the interplay of many factors. Several subtypes of Irritant Contact Dermatitis have special clinical attributes which make them stand out from the other forms of CD.

**Picture 5:** Sub acute ACD from rubber gloves
Acute Contact Dermatitis

Acute CD is characterized by redness, papules (bumps) and vesicles. The appearance of small 1-3 mm vesicles and occasionally bullae is the hallmark of allergic contact dermatitis. Itching is often intense and may disrupt sleep.

Subacute Contact Dermatitis

Subacute CD is characterized by redness and itching but vesicles are seen as fine peeling, scale or a thickening of the skin. Itching continues and scratching causes small sores or excoriations. Linear fissures or cracks are common at sites of mechanical trauma, especially on the hands.

Chronic Contact Dermatitis

Chronic CD is minimally red or inflamed and is characterized by thickened skin that is scaling or shiny on the surface with cracking and fissuring at sites where skin is stretched by motion. Burning and pain of the fissures is more a complaint at this stage than itching.
Airborne CD

Airborne exposures to allergens and irritants generally cause dermatitis at exposed sites on the face, neck and arms. The scalp and eyelids can be involved. Areas under clothing are usually less involved but can be and depend on the fit, amount of sweating and mechanical factors. A recent review on airborne CD in occupational cases concluded the most frequently associated chemicals were fragrances, preservatives, (systemic) drugs (occupations that produce or make systemic drugs) and methacrylate and epoxy resins. 20

Picture 7: Airborne ACD to epoxy resin system in assembler

Acute Irritant Contact Dermatitis

This form of ICD often presents like a chemical burn or a large bulla or blister. It does not often have the eczematous scaly red pattern usually seen in CD.

Frictional Hand Dermatitis

This form of ICD caused by mechanical trauma to the skin has a few clinical distinctions from other forms of CD. It is usually more prominent on the dominant
hand where the mechanical stress is most evident. It may only involve the palm of the hands and often looks psoriasiform with thickened red scaly patches that are not itchy. Clinically it may only involve the sides of the fingers or the fingertips depending on what the mechanical stress is.

**Picture 8: Frictional Hand Dermatitis in office worker**

The natural or temporal history of the dermatitis is often a clue to whether the eruption is ICD or ACD. Classically ACD occurs after repeated exposures with the workers really having little to no skin issues until they “suddenly” develop ACD. A history of tiny 1-3 mm itchy vesicles is classic for ACD. This will initially improve minimally over the weekend and clear completely while away on holiday. As the exposure becomes more chronic patients rarely clear completely even after weekends or one week vacation. Traditional ACD is a 3 week disease eruption. For example, if you touch Poison ivy, a common contact allergen, you have an eruption which usually takes about 3 weeks to completely resolve. This is only after one exposure. Put another way, if a worker repeatedly develops eruptions that last several hours to several days or clears after one night this is not the natural history of ACD. This would be more classic for ICD.
It is more difficult to generalize a natural history for ICD as there are many subtypes but in general, an irritant applied to the skin generally causes an eruption that clears fairly quickly, usually after several days or one week. A classic example would be a patient who works in a factory and develops red, burning eruptions on the face that clear by the next morning. This would be consistent historically with an Airborne ICD. Airborne ACD would not clear by the next morning and last weeks. As irritants accumulate over days and months the eruptions become more chronic and do not clear after several days or weeks.

The time course of cumulative or chronic ICD is slow compared to ACD. It has been reported that even after the skin appears normal, it takes approximately 4 months or more for the barrier function to normalize. 21

In Frictional Hand Dermatitis, a form of ICD, the natural history is usually very prolonged with patients having mild dryness or callous formation from repeat mechanical trauma for months to years. After a time patients may then develop thickened, painful and fissured patches on palms, fingers or fingertips usually with the dominant hand more involved. The natural history is a very slow onset with a chronic and often recalcitrant course thereafter. Once removed from the repeat mechanical trauma these patients hands always improve. It may take weeks to months depending on the severity of the dermatitis.

Hyperkeratotic Hand Dermatitis is a very small subset, 2% of all forms of Hand Dermatitis, and may have a genetic predisposition. It is similar to Frictional Hand Dermatitis in that it is precipitated often but not always, by repeat mechanical trauma to the hands. However, once this form of Hand Dermatitis is precipitated these patients do not improve when removed from their work environment. This is really the distinguishing factor from Frictional Hand Dermatitis. They go on to have chronic and often disabling hand dermatitis that is very resistant to therapy. This form of hand dermatitis is not an uncommon cause of permanent disability.

Diagnosis

Establishing a diagnosis of Occupational Contact Dermatitis OCD involves two steps:

1. Recognizing the existence of an occupational exposure and;
2. Assessing whether that exposure represents a cause or aggravating factor.
Table 2. *Diagnostic criteria for occupational contact dermatitis.* 22

1. Onset of the eruption after the patient began work
2. Precise cause identified
3. Patient primarily exposed to the etiologic agent at work
4. Site of onset exposed to the causative agent at work
5. Distribution of lesions conforms with occupational exposure
6. Appropriate time between exposure to the causative agent and the development of lesions
7. Biological plausibility
8. Non-occupational dermatitis excluded
9. Other workers similarly affected
10. Process change before the onset of dermatitis
11. True positive patch-test reaction(s) in cases of allergic contact dermatitis.

Another list of criteria often cited in the literature is the Mathias criteria for establishing occupational causation and aggravation of contact dermatitis.23 The validity of these criteria has been studied over the years and they consistently prove to be very useful, in particular when validity of workplace exposure is questioned.24

Criterion 1: Is the clinical appearance consistent with contact dermatitis?

Criterion 2: Are there workplace exposures to potential cutaneous irritants or allergens?

Criterion 3: Is the anatomic distribution of dermatitis consistent with the form of cutaneous exposure in relation to the job task?

Criterion 4: Is the temporal relationship between exposure and onset consistent with contact dermatitis?

Criterion 5: Are non-occupational exposures excluded as likely causes?

Criterion 6: Does avoiding exposure lead to improvement of the dermatitis?

Criterion 7: Do patch tests or provocation tests implicate a specific workplace exposure?
To accomplish the above criteria the physician needs to take a comprehensive history, a complete exposure review of potential irritants and allergens at work, examination and patch testing. A visit to the workplace may also provide essential information in the investigation of suspected OCD.

Identifying all the probable hazardous agents in the workers environment is very important and a difficult task. The agent may be chemical, physical or biological. Physical factors such as heat, humidity, cold, vibration, radiation and mechanical trauma must be considered as well as chemicals. A qualified Occupational Hygienist is indispensable in reviewing the workers exposures and reviewing and summarizing Material Data sheets on all chemicals workers are exposed to.

**Patch Testing**

Properly applied and appropriately interpreted patch tests are the only scientific proof of ACD. There is no diagnostic test for ICD. Patch testing for OCD cannot be done until a complete exposure review and documentation of probable chemicals is determined. Patch testing will only be useful if the worker is tested with what he/she is being exposed to in their work environment. Series or trays of allergens for specific occupations and substances used by the patients are required. In some instances if the standard supplied occupational trays will not cover what the patient is exposed to, testing with the workers raw material in a diluted or raw form will be necessary. This requires an experienced Clinician and chemist to prepare the raw materials. This unfortunately can delay patch testing but will prevent false-negative testing.
Patch Test Trays

Several companies produce allergens in standardized percentages ready made for application. To determine the correct testing percentage each allergen is tested at various concentrations on 20 controls and the concentration that will elicit true positive ACD without ICD reaction is chosen. Some examples of occupational trays include:

1. Oils and Coolants
2. Hairdressing Tray
3. Bakers’ Tray
4. Mechanics’ Tray

Raw Material Patch Testing

- raw material can be tested in a diluted form. This requires obtaining the workers raw materials, for example paint or varnish, then diluting them in appropriate
vehicles such as petrolatum, alcohol or sterile water. Once various dilutes are made these custom allergens are tested on controls subjects to ensure they do not elicit an irritant reaction. Once controls are done they are then ready to test on the worker.

- Open or semi-open testing of raw material is a more controversial way of testing patient’s raw materials. Materials that have a Ph below 3 or above 10 are not tested in this manner. Any raw material that comes into contact with the worker’s skin on a daily basis such as an oil or paint could be tested in this manner. A small amount 1-2 ml (milliliters) applied with a cotton swab on a 1 cm squared section of the skin and allowed to dry completely. It is then covered with paper tape or left open. This area is then reviewed at 48 hours and 96/120 hours like routine patch testing. Reading of these results must be done cautiously and irritant reactions are not uncommon.

**Patch Testing Technique**

- patch testing involves the use of small chambers either made of plastic or aluminum. A small 0.5 cm strip of allergen in petrolatum or in water or alcohol is applied to the chamber. These chambers come in strips. The strips are placed on the patient’s clean back.

- the strips are removed in 48 hours and a preliminary read is done and recorded

- a final or delayed read is critical to patch test interpretation. The late reading is done usually at 96 hours or 120 hours. If only a 48 hour read is done patch test results are not valid and cannot be interpreted correctly. ACD is a delayed hypersensitivity reaction and must have a delayed read to be valid.

- patch tests should not be applied to a back that has active dermatitis. This can sometimes make patch testing difficult as many patients have chronic dermatitis. The dermatitis should be cleared with either time off work or systemic prednisone before testing can proceed.

- patch tests need to be kept dry for the entire 96/120 hours. This is often difficult in factory workers as they sweat. In these cases time off work will be required to complete patch testing.

**Patch test interpretation and Relevance**

The final day read at 96 hours or 120 hours is obviously of great importance. An experienced clinician is required to interpret the positive as true allergic responses or irritant reactions. Once positives are determined the last and most important step is to determine if that positive is the cause of or relevant to the patients work related eruption.
This again takes experience and knowledge of patient exposures. A patient may have a strong positive to Chromium, but it may not be present in their work environment. In this case the allergen is not relevant and he may have been sensitized from an earlier exposure.

Patients who have Atopic Dermatitis often develop several irritant reactions to patch test chemicals. An experienced Clinician will clinically be able to determine these as irritant reactions and not true allergic positives.

**Picture 10:** Positive Patch test results in dental hygienist 120 hour final read

Cross-reactions of allergens in Patch testing

Cross reactions between chemicals occur in many areas of medicine. An example more commonly known would be protein cross reactivity between bananas and latex. Allergy is an immune response as was discussed earlier. An antibody is produced against a very specific antigen. Antigens are usually proteins of some kind. If several proteins look similar then the antibody would recognize both. An example for ACD:

- hair colour dyes are from the chemical class called Azo dyes. There are many other chemicals from this Azo class including textile dyes, and some drugs. If a
hairdresser becomes allergic to hair dye she may also react allergenic to some clothing/textile dyes or some drugs because they are all chemically similar.

Differential Diagnosis

Some of the major considerations in the differential diagnosis of CD may at times coexist with CD. It is sometimes more helpful to consider all the contributing factors rather than assume there is only one possible diagnosis. Additional diseases to consider are determined by the clinical appearance at the time of evaluation, acute or chronic and include:

- Psoriasis
- Pustular eruptions including pustular psoriasis
- Id reaction from fungal infections on the feet. An “Id” reaction is red and scaly and can look like dermatitis.
- drug eruptions
- insect bites or infestations
- bacterial infections including folliculitis
- primary vesicular bullous dermatoses such as Dermatitis herpiformis, Porphyria cutaneous tarda
- Primary papulosquamous dermatoses including Lichen planus and Pityriasis Rosea

Risk Factors

The workers most commonly reported to be at increased risk of developing occupational contact dermatitis include agricultural workers, beauticians, chemical workers, cleaners, construction workers, cooks and caterers, electronics workers, hairdressers, health and social care workers, machine operators, mechanics, metalworkers and vehicle assemblers. Atopy also appears to be an independent risk factor for the development of occupational contact dermatitis. A history of atopic dermatitis, particularly in adulthood, appears to be an independent risk factor for the development of occupational contact urticaria.

Occupational contact dermatitis can present at any stage in the worker’s career, including apprenticeship.
Controversy in the medical community about Allergic Contact Dermatitis and Irritant Contact dermatitis

CD is not well taught in dermatology programs. Many institutions that have Dermatology programs do not have a Dermatologist who has expertise in the field of CD. In these cases the amount of training in CD is very little. Patch testing is an expensive and time consuming endeavor for both patient and doctor. Unfortunately it is not well remunerated in Canada. For this reason, many Dermatologists do not offer the testing in their offices.

Some Allergists patch test and often do not have the proper or in depth training to interpret and determine the relevance of positives.

For those physicians who do patch tests in their clinics; they usually only do a standard set of allergens which is usually inadequate for complex occupational cases. Complicated occupational cases require in depth exposure reviews and it is not possible or even probable that the clinician has the time to review and complete a thorough exposure review. Thus, in addition to a nurse, an occupational hygienist is necessary. Furthermore, if raw material testing is needed, a chemist and lab will be required.

In Canada there are only a handful of centers that have Patch test clinics capable of handling complex Occupational CD.

In conclusion, all of the above factors often lead to an incorrect diagnosis or a delay in the diagnosis for affected workers.

Issues Specific to the Tribunal

a. What is the relationship between exposure to sensitizers and ACD?

Not all patients will become sensitized to allergens. There is a genetic factor playing here as discussed above. Patients require T cells that recognize the allergen and this appears to be genetically determined. The presence of dermatitis of any type, as discussed, will increase one’s chances of becoming sensitized. There is only one mechanism of sensitization. It is a Type IV hypersensitivity reaction.

b. Can there be a delay between exposure and the onset of the dermatitis?

Yes, this was answered in the Natural history section. It depends on the clinical type but in general for both ICD and ACD there can be a long delay before the onset of dermatitis. This is not seen in acute ICD which is more similar to a chemical burn.
c. What is the relationship to latex?

Allergens presented to the body can result in one of four hypersensitivity reactions discussed above. Latex allergen results in a Type 1 immediate hypersensitivity reaction which is mediated by antibody IgE. The resulting clinical picture is not that of dermatitis but of urticaria (hives), angioedema (large circumscribed areas of subcutaneous edema) and anaphylaxis (hypersensitive reaction). Latex allergen does not result in a Type IV or delayed hypersensitivity reaction which results in dermatitis. This is a common misconception by both doctors and patients. Latex allergy from a glove can result in urticaria or hives on your hands, not a dermatitis.

d. What is the relationship, if any, to metal working fluids (MWF)?

Some chemicals act as both an irritant and a potential allergen. Metal working fluids can be irritating to the skin and also contain biocides or preservatives which are potential allergens. Often a patient can have both ICD and ACD from metal working fluids. For example a worker who tests positive to a particular biocide can change the MWF to another that does not contain the allergen. The worker may still have ongoing hand dermatitis based on the irritancy of the MWF.

The treatment of ACD is really avoidance of the allergen. Once allergic, always allergic and you simply must avoid the allergen. Treatment of ICD is more difficult. Management focuses on protecting the skin from the irritant or changing the way the irritant comes into contact with the skin though a change in handling or processing, decreased exposure, or improving protective equipment.

e. Can one distinguish between the rash related to ICD and that related to ACD?

As answered above, generally no. There are clues but patch testing should always be done to rule out ACD in most cases.

f. Are there various types of dermatitis caused by exposure to irritants?

Yes, they are discussed in detail above.

g. Can some substances be either a sensitizer or an irritant, depending on conditions or level of exposure?

Yes, answered in question d.

h. Aggravation of atopic dermatitis and other forms of dermatitis related to mental stress in the workplace.

There are several articles in the literature that show an association of worsening of forms of Atopic Dermatitis/eczema with stress.\(^{26, 27}\) There appear to be neurological mediators associated with flaring of the dermatitis. This may also in the future prove to be a possible route for treatment of Atopic Dermatitis.
There are many subsets of Atopic Dermatitis; I would consider Dyshidrotic Hand Dermatitis and Nummular Dermatitis to be a type of genetic or atopic form of dermatitis.

The association of stress flaring these forms of dermatitis is very well accepted clinically by dermatologists across the world. We are just learning the scientific reasons for this.

Other forms of dermatitis including Seborrheic dermatitis, allergic and irritant dermatitis can also be worsened by stress in the workplace but the studies and science here are less studied. This conclusion mainly comes from clinical practice.

i. Non-cutaneous complaints such as a burning sensation of the eyes and/or throat as well as tingling sensations related to various workplace exposures.

The description of the above symptoms would classify a diagnosis of Airborne Irritant Contact Dermatitis. As discussed in the ICD section, there are 10 subtypes of irritant responses. This would fall into the subjective or sensorial subtype.

“Subjective or Sensorial Irritation”

Contact with an irritant produces a sensory discomfort, usually manifesting as a stinging, burning or itchy sensation, in the absence of clinical and histological (the structure of cells) evidence of skin lesions. The threshold to develop this reaction varies between people. Lactic acid and propylene glycol are good examples of subjective irritants. The outcome from this irritant reaction is good.

This can be from direct contact of chemicals or airborne. If chemicals are volatile (easily evaporated at normal temperatures), the chemical in the air can land on the skin of the face, mainly cheeks, forehead, ears, eyelids. This is often exacerbated by dusty environments as the volatile chemicals will combine with dust in the air and increase the concentration of the volatile chemical in the air. For the subjective or sensorial subtype of ICD, patients do not see a rash but just experience symptoms of itch, burn or stinging. Volatile chemicals will possibly irritate the eye, nose and throat, leading to increased watering of the eyes and nose, dry irritated throat, congestion and headache.

j. When can Contact Dermatitis lead to Permanent Disability?

The difficulty with contact dermatitis becomes avoiding the chemicals that are causing the irritation in ICD or allergy in ACD. It is possible in many cases for patient to return to work with modification of their jobs, modification of job schedule (i.e. less shifts back to back), increased protective measures, allergen avoidance and sometimes change of departments etc. In some cases this is not possible as the job requires exposure.
Example: a worker who works at a nickel mine and becomes allergic to the metal Nickel or a nurse, chef, beautician or baker with exposure to wet work and glove wearing. If a clinical nurse working a 12 hour shift can only endure 8 hours of hand washing before her hands breakdown and crack, it becomes difficult for her to continue clinical work. She would have to change to clinical research or administration which requires no patient contact and therefore no hand washing.

Airborne exposures often lead to permanent disability. Workers who become allergic to ubiquitous allergens like fragrances may have difficulty working in open spaces with other workers unless a fragrance free policy is enforced. They may also have issues walking through a mall or going to any public space.

More specific airborne occupational allergens such as epoxy resins would only impair the worker at his/her job. Example: assembler of airplanes who becomes allergic to epoxy and has subsequent ACD to contact and airborne epoxy resins systems. This worker would no longer be able to work in the airplane production plant but could find work in another field that does not expose him to epoxy resin systems.

Finally, three specific clinical forms of Hand Dermatitis, once precipitated by irritants, can become chronic and refractory to treatment even when the patients are taken out of their work environment and at times when they are not doing anything at all. These include Dyshidrotic Hand Dermatitis, Hyperkeratotic Hand Dermatitis and Traumatic Irritant Hand Dermatitis. The latter 2 clinical subtypes are forms of ICD.

Dyshidrotic Hand Dermatitis is a clinical subset of hand dermatitis where patients develop small vesicles (water blisters) on the palms and sides of the fingers. They can be very itchy and in some patients the vesicles very large. It is generally considered in the Dermatology literature to be a primary genetic form of hand dermatitis. However it is my clinical experience that many patients with Occupationally associated Dyshidrotic Hand Dermatitis may be precipitated on the job especially where there is significant wet work, glove wearing or solvent exposure. The patients may have an atopic diathesis which means genetically prone, but many have never suffered from the condition before their exposure. Once they develop it, the hand dermatitis often continues after removal from workplace. This diagnosis more than others poses an issue with claims as it is generally considered to be genetic but once precipitated can be debilitating not only for work but activities of daily living.

k. Please provide a list of common irritants and list of common sensitizers that have been noted in various occupations.
<table>
<thead>
<tr>
<th>Occupations</th>
<th>Common Irritants and Sensitizers</th>
</tr>
</thead>
<tbody>
<tr>
<td>Agricultural Workers</td>
<td><strong>Irritants</strong>&lt;br&gt;- soaps, detergents, pesticides, disinfectants, solvents, and petrolatum products, fertilizers, grains and other plant products&lt;br&gt;<strong>Allergens</strong>&lt;br&gt;- rubber in gloves, hoses, pesticides, chromate in leather, milk preservative, cement and preservatives in creams and ointments</td>
</tr>
<tr>
<td>Construction Workers</td>
<td><strong>Irritants</strong>&lt;br&gt;- cleansers and solvents, dirt and refuge, wet cement, fiberglass, resins. Physical mechanical irritants from repeat trauma to the hands.&lt;br&gt;<strong>Allergens</strong>&lt;br&gt;- chromate in cement, leather gloves and boots and wood preservative, rubber in gloves and tools, epoxy resin in paints and adhesives, colophony in pine dust soldering flux, nickel from tools, formaldehyde from resins and metal cleansers.</td>
</tr>
<tr>
<td>Food Handlers</td>
<td><strong>Irritants</strong>&lt;br&gt;- wet work, soaps and detergents, spices, vegetable and fruit juices, garlic and onion, flour and dough&lt;br&gt;<strong>Allergens</strong>&lt;br&gt;- rubber gloves, flavours, essential oils, food preservative and some spices&lt;br&gt;<strong>Contact Urticaria</strong>&lt;br&gt;- from certain foods</td>
</tr>
<tr>
<td>Hairdressers</td>
<td><strong>Irritants</strong>&lt;br&gt;- wet work, shampoos, and permanent wave solutions&lt;br&gt;<strong>Allergens</strong>&lt;br&gt;- nickel from scissors and clips, rubber gloves, formaldehyde in shampoos and cosmetic hair products, hair dye, fragrances, permanent wave solution</td>
</tr>
<tr>
<td>Housekeeping</td>
<td><strong>Irritants</strong>&lt;br&gt;- wet work, cleansers, disinfectants and bleach. Physical mechanical irritants from brooms, floor cleansers etc.&lt;br&gt;<strong>Allergens</strong>&lt;br&gt;- rubber gloves, fragrances, preservative in cleaning products and creams, gluteraldehyde, choloxylenol and benzalkonium chloride disinfectant allergens</td>
</tr>
<tr>
<td>Occupations</td>
<td>Common Irritants and Sensitizers</td>
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<td>-----------------</td>
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</tr>
</tbody>
</table>
| Machinists      | **Irritants**  
- metalworking fluid MWF, metal chips, soaps and detergents.  
Physical mechanical irritants.  
**Allergens**  
- metals, rubber in gloves and anticorrosives in MWF, preservatives in MWF, colophony an emulsifier in MWF, fragrances in cutting oils, chromeate- anti-corrosive. |
| Medical Workers | **Irritants**  
- wet work/hand washing, soaps, detergents, disinfectants, ethylene oxide  
**Allergens**  
- rubber in gloves and tubing, preservatives, chromeate in sutures, antimicrobials, methacrylate, fragrances, tricolsan, gluteraldehyde, chloroxylenol and benzalkonium chloride disinfectants in hand washes and other medical solutions |
| Office Workers  | **Irritants**  
- wet work, developers, fixers, bleaches. Physical mechanical irritants from repeat paper use.  
**Allergens**  
- rubber gloves, developers, formaldehyde, photographic chemicals. |
| Printers        | **Irritants**  
- solvents, inks, abrasive soaps and cleaners  
**Allergens**  
- chromeate in fountains solutions, rubber in gloves, epoxy resin, Phenylenediamine and colophon in ink, photographic chemicals, printing ink (raw material in 1% petrolatum) |
| Textile Workers | **Irritants**  
- acids, alkalies, fiberglass, detergents. Physical mechanical irritants from repeat touching of fabrics.  
**Allergens**  
- chromeate in mordant, rubber in rubberized textiles, epoxy resin as adhesive, dyes, formaldehyde in fabric finishes, nickel in tools, textile raw material, soaked in water for 10 minutes and left on for 5 days. |
Other Definitions

**Urticaria**

Urticaria, also known as hives, is a skin reaction due to the release of a chemical in the body called histamine. It is a Type 1 immediate hypersensitivity reaction. Histamine causes swelling, redness and itching. Urticaria is most commonly an adaptive immune response. Allergen bound IgE antibodies bind to mast cells. Once the IgE antibodies bind to the mast cells they release histamine which results in the urticarial lesions.

Urticaria in most cases results from the ingestion of an allergen (for example peanuts) resulting in lesions. Clinically urticaria or hives are red, swollen itchy lesions without any scale and they resolve over 24 hours.

**Contact Urticaria and Occupational Skin Disease**

Contact Urticaria can occur but is less common. Some allergens when put on the skin, or contact the skin can induce Urticaria. An example would be Latex. Latex allergen when applied to the skin in an allergic patient can result in a hive like lesion.

Occupational contact urticaria is associated with proteins in food and latex gloves, especially in healthcare workers.\(^{12}\)

Occupational contact urticaria accounts for between 1-8% of reported cases of occupational disease.\(^ {12}\)

The workers most commonly reported to be at increased risk of developing occupational contact urticaria include bakers, farmers, health and social care workers and those in the food preparation industries.\(^ {12}\)

**Definition of Spongiotic eczema**

Pathology is a subspecialty of medicine that deals with disease through the analysis of tissue, cell or body fluid samples. Pathology has its own medical terms and definitions. Spongiosis or spongiotic is a pathologic term to describe the hallmark of eczema or dermatitis. You will not see spongiosis in any other diagnosis except eczema/dermatitis.

The pathology definition of spongiosis is intercellular edema (abnormal accumulation of fluid) in the epidermis. It is characteristic of eczematous dermatitis. Clinically it will manifest as intraepidermal vesicles (fluid containing spaces), juicy papules or lichenification (thickening of the epidermis).

In summary, if a patient has a biopsy of their skin and it shows spongiosis then the patient has eczema/dermatitis. It does not tell us the cause of the eczema.
Eczema

Eczema and Dermatitis define the same thing and can be used interchangeably. The definition of Eczema/Dermatitis is inflammation of the skin.

References


