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# Eczema and Contact Dermatitis

by John Berth-Jones, Eunice Tan and  
Howard I Maibach

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# **Eczema and Contact Dermatitis**

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## Introduction

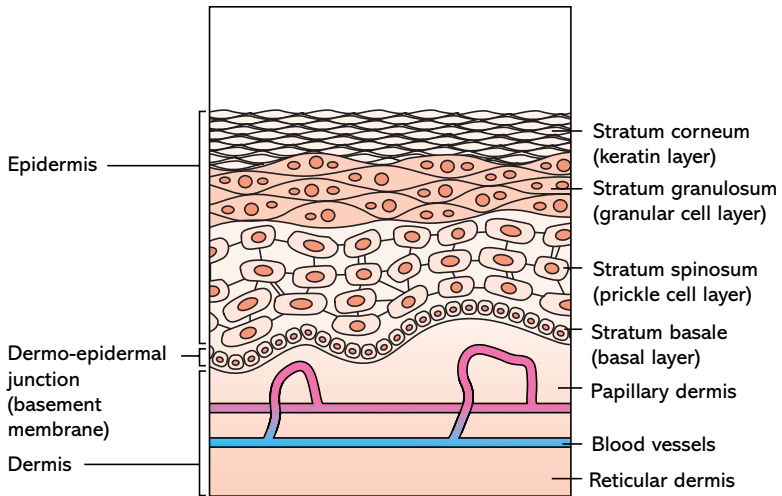
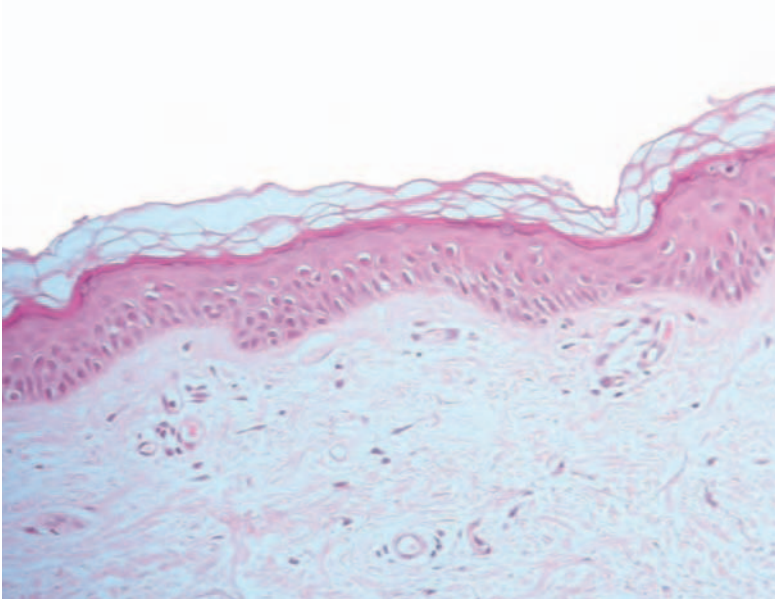
Eczema (also known as dermatitis) encompasses many common and important skin diseases. These affect patients of all ages. They include atopic eczema – the intensely itchy and distressing disease seen mainly in infancy and childhood – through industrial contact dermatitis – a major source of handicap and time lost from work in the adult population – to asteatotic eczema – the itchy dry skin associated with aging. Indeed, there can be few people who are not affected by some form of eczema at some point in their lives.

Largely because of the high incidence of eczema, physicians in many different medical disciplines are regularly involved in its management. This text is intended to help by familiarizing the non-dermatologist reader with the etiology, diagnosis and treatment of the various forms of this disease.

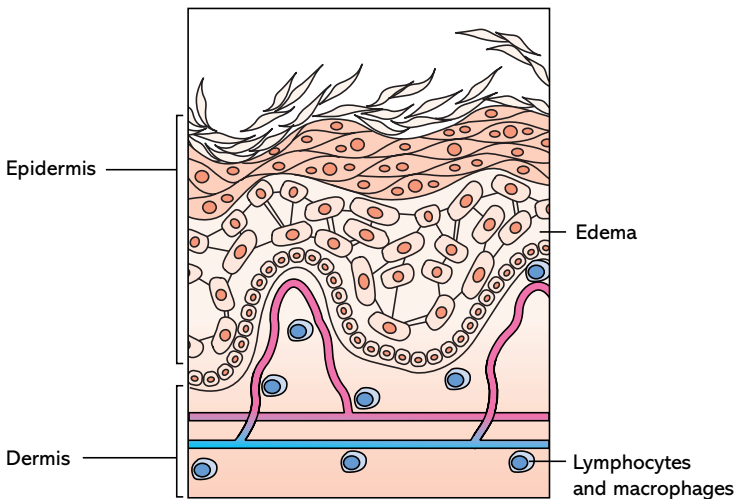
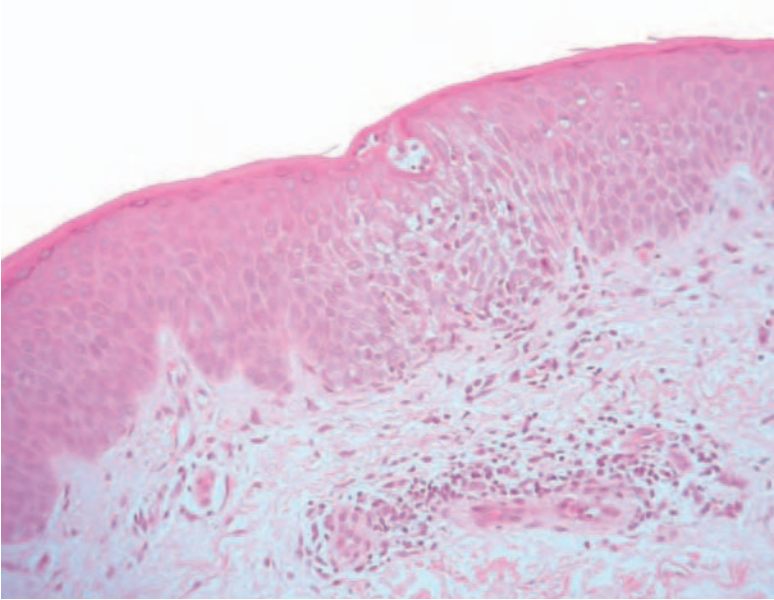
The different types of eczema have such diverse etiologies that it is not immediately obvious what they have in common. They all share a range of clinical and histological features. They are characterized, in the acute phase, by itching and erythema. Sometimes, especially on the hands and feet, vesicles or bullae may appear. Dryness, excoriation, exudation, edema, induration, scaling, lichenification and fissuring develop later. Lichenification, a sign of chronic inflammation, is a thickening of the skin initially manifested by a subtle accentuation of surface markings and progressing to marked induration and roughening of the skin surface. The histological features of eczema include edema of the epidermis (often called spongiosis), which may progress to vesicle or bulla formation, and inflammation of the dermis (infiltration with lymphocytes and macrophages). The histological appearances of normal and eczematous skin are shown in Figures 1 and 2, respectively.

A good starting point in the classification of eczema/dermatitis is the definition of two groups:

- endogenous, meaning the eczema is not believed to be due to contributing factors outside of the body
- exogenous, meaning outside influences have a significant contributory role.



**Figure 1** Histological appearance of normal skin. Photomicrograph reproduced courtesy of Dr N Bajallan, George Eliot Hospital, Nuneaton, UK.



**Figure 2** Histological appearance of eczematous skin. Edema develops between the keratinocytes in the epidermis (spongiosis), the epidermis thickens (acanthosis) and inflammatory cells infiltrate the dermis. Photomicrograph reproduced courtesy of Dr N Bajallan, George Eliot Hospital, Nuneaton, UK.

TABLE 1

**Classification of eczema/dermatitis**

**Endogenous**

- Atopic
- Seborrheic
- Discoid/nummular
- Pompholyx
- Venous
- Asteatotic (eczema craquelé)
- Juvenile plantar dermatosis
- Erythroderma

**Exogenous**

- Allergic
- Irritant
- Photosensitive

An example of this classification is given in Table 1. In reality, the situation may not be so simple, and eczema commonly has a mixed etiology. For example, atopic eczema may be aggravated by exposure to irritants or by allergy to topical medications.

Traditionally, endogenous dermatitis was given the term ‘eczema’ and exogenous dermatitis was termed ‘dermatitis’. Indeed, in an industrial setting the term dermatitis is often viewed as indicating a condition resulting from occupational exposure to irritants and may be seen as a diagnosis associated with litigation. However, the terms dermatitis and eczema are now used interchangeably. Eczema is derived from the Greek word meaning ‘to boil’ and refers to the itching and heating of the skin and to the appearance of small blisters. Dermatitis simply means inflammation of the skin.

## Epidemiology

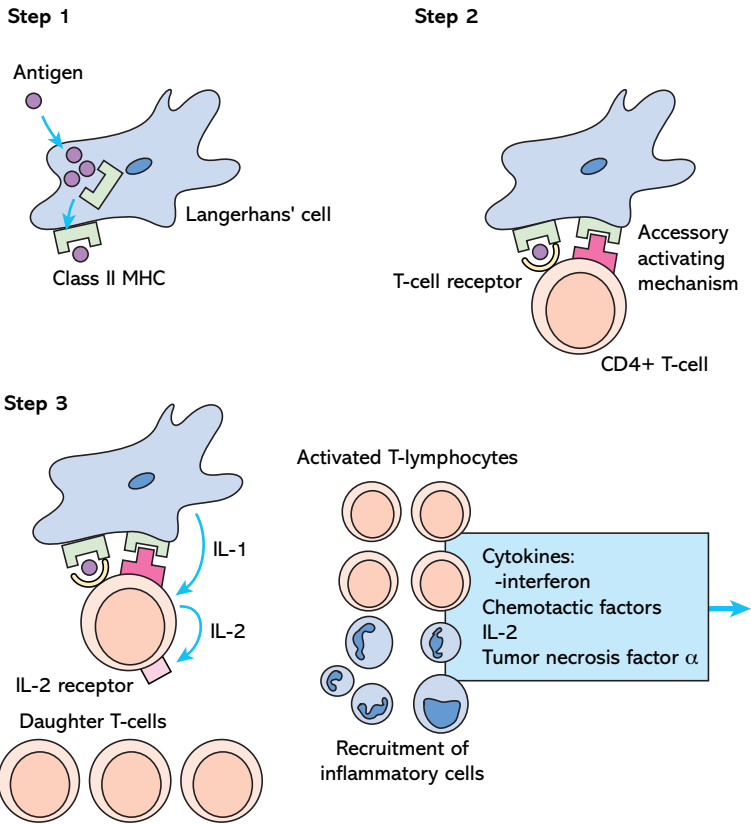
Allergic contact dermatitis (ACD) is seen predominantly in adults and is less common in children and the elderly. This age distribution is probably due more to patterns of exposure to potential allergens than to any constitutional changes in the immune system. The most common form of contact allergy is nickel sensitivity (Figure 1.1), which probably affects at least 10% of women.



**Figure 1.1** Nickel dermatitis caused, in this case, by contact with metal earrings.

## Pathophysiology

The development of ACD requires two phases. The first is a sensitization phase during which contact with the allergen results in the development of a specific immune response. This sensitization process may occur rapidly after brief contact with the allergen but often follows years of repeated contact. Once sensitization has occurred, subsequent contact results in the second phase of the reaction – the elicitation phase. This is a delayed (type IV) hypersensitivity reaction (Figure 1.2) which gives rise to the dermatitis at the site of contact. The delay between contact and the development of dermatitis in sensitized individuals is typically 48–72 hours, but reactions may occasionally develop within a few hours, or a longer delay of a week or more may be observed.



**Figure 1.2** Cell-mediated immune (type IV) reaction. In step 1 the antigen is taken up by antigen-presenting cells (e.g. Langerhans' cells in the skin), processed and bound to class II major histocompatibility complex (MHC). In step 2, these complexes migrate to the paracortical area of the draining lymph node via the afferent lymphatics. The MHC and antigen are recognized by T-cell receptors on CD4+ T-cells. In step 3, interleukin-1 (IL-1) is released by the antigen-presenting cell. The T-cells secrete IL-2, express IL-2 receptors and proliferate. Daughter T-cells, including memory cells, are generated. On re-exposure to the same antigen, the memory T-cell recognizes the antigen after it has been processed in steps 1 and 2. T-lymphocytes become activated and secrete cytokines, which induce the components of inflammation.

## Epidemiology

Irritant contact dermatitis is a common form of contact dermatitis, accounting for up to 75% of exogenous dermatitis. Those who work in constant contact with oils or solvents and those who have their hands constantly in water or detergent are particularly vulnerable. People in certain occupations, such as nurses, hairdressers and those who work in engineering, are therefore the most frequently affected. Irritants are also encountered at home, so people who spend their days looking after young families often develop irritant hand dermatitis. Individuals with a history of atopic eczema, even in childhood, seem to remain sensitive to the development of irritant dermatitis of the hands, and this may become a real problem if they enter the wrong employment.

In an industrial setting, irritant dermatitis should be largely preventable in all but the most vulnerable individuals. Work should be planned in such a way that constant or repeated exposure to irritants is minimized by using the least irritant materials possible, by installing appropriate shielding and by use of protective gloves and clothing. Inadequate care is often exposed when several employees working on the same task all develop dermatitis; this is the subject of much litigation.

## Pathophysiology

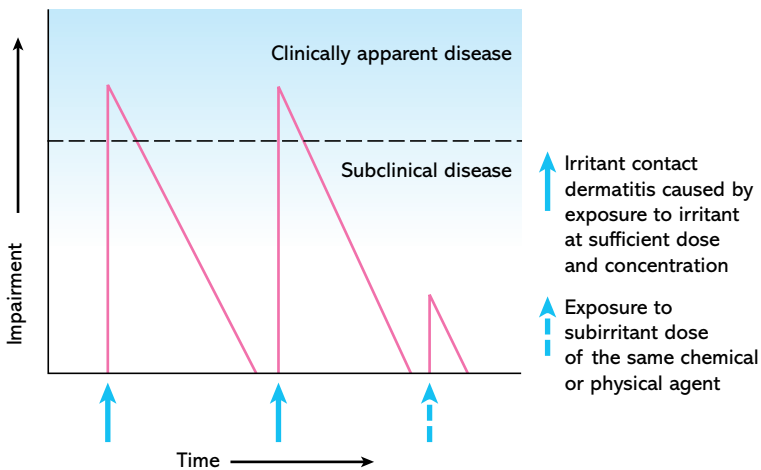
Irritant dermatitis results from physical or chemical damage to the skin. In contrast to allergic dermatitis, the mechanism is not primarily immunologic, and inflammation develops as a secondary event. Chemical damage to the skin can be caused by acids, alkalis, detergents, solvents, antiseptics, adhesives and industrial oils used for cooling or lubrication. Physical causes include friction, contact with abrasive powders and heat. All individuals will develop dermatitis if exposed to these irritants with sufficient intensity, although there is considerable variation between individuals in the amount of exposure required. Dermatitis most commonly develops as a result of repeated mild

exposure which has a cumulative effect, but acute reactions are also seen, caused by a single intense exposure to the irritant.

### Clinical presentation

**Acute irritant contact dermatitis** develops as a result of a single overwhelming exposure to an irritant or caustic chemical or a series of brief chemical or physical contacts (Figure 2.1). This response may be enhanced by occlusion. There is immediate stinging or burning. The initial reaction is usually limited to the area of contact. The most frequently affected sites are the finger webs and dorsa of the hands, especially under rings. The clinical features range from transient erythema or chapping to edema, inflammation, pain, vesiculation and, in severe cases, exudation, bullae and necrosis.

**Cumulative irritant contact dermatitis** develops as a result of repeated chemical or physical insults (Figure 2.2). The dorsal aspects of the hands (Figure 2.3), finger webs, face and eyelids are the most frequently affected sites. Initially there are localized patches of dry, slightly inflamed, chapped skin. Painful fissures may develop on the palms and



24 Figure 2.1 Acute irritant contact dermatitis.