Updates in Clinical Dermatology
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Wanda Robles

Skin Disease in Travelers

Springer

Updates in Clinical Dermatology

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Skin Disease in Travelers

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Foreword

One of the major features of our changing world is the regular movement of individuals and groups from one region to another. In some cases, this reflects the ease of travel leading, in turn, to large numbers of tourists leaving their home environment to visit different regions and people. There has also been an increase of movement for a different reason, in that wars, civil unrest and natural disasters have led to large numbers of people fleeing their homes and seeking refuge in other countries; in other cases, the motivation for travel is to escape from a cycle of poverty and deprivation. Whatever the reasons the consequences are that the full range of human disease follows these trends leading to people presenting with illnesses in another country where health workers are often unfamiliar with disease common or endemic in a different part of the world. This is not simply restricted to encounters with infectious diseases; it also extends to physical reactions to different climates, hot and cold, as well as the different elements that make up those climatic zones from jungles to mountains to seas and lakes. All these environments are associated with the need to address different and unfamiliar diseases, presentations and treatments.

The skin, being the largest organ of the human body and the site exposed to the natural environment, is often the first place where these diseases are seen. They range from infections, which are normally confined to specific regions now presenting in outside their normal endemic area to the consequences of overcrowding and deprivation amongst large mobile populations which also lead to dermatological diseases such as scabies and leishmaniasis.

This book addresses all these issues. Written by subject experts in the different areas, it draws a comprehensive picture of a range of infections presenting in the skin caused by bacteria, virus, fungi and parasites as well as the skin signs that develop as a consequence of encounters with different climates; these include cold injury as well as infections and trauma associated with swimming in unfamiliar seas. It addresses the pathology, the clinical manifestations and treatment of these imported diseases and, as such, will be useful for both dermatologists and non-dermatologists; it addresses the full

range of conditions experienced by travellers and migrants. This book is a welcome addition to more conventional works on skin disease, while helping to inform those encountering the new and less familiar manifestations of skin disease that result from travel.

Professor (Emeritus) of Cutaneous Infection Roderick Hay
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Preface

With the increased ability of people to travel around the world, a great number of skin diseases, mainly infectious, can be acquired by travellers. These may represent a challenge not just to the medical community but particularly to dermatologists. Signs and symptoms may be nonspecific. Furthermore, many infections by different organisms may share a similar set of signs and symptoms making definitive diagnosis even more difficult. Whenever possible, this is achieved only in isolation or demonstration of the causative organism. However, this can be very difficult if the diagnosis is not considered, on clinical suspicion by the physician. Though infectious skin diseases should be high in the differential diagnosis at presentation of any traveller with skin disease, there are also some conditions produced by physical agents, for example, exposure to extreme temperatures: heat or cold.

The aim of this book is to raise awareness in the medical world in order to consider a potential infectious cause for many of the skin rashes in travellers, as well as over exposure to different climatic conditions. It also aims to give advice on potential ways of prevention but foremost to acquaint the physician with the different signs and symptoms of skin diseases in travellers, as well as the best available treatments. I very much believe this book will be a valuable contribution to all medical communities including young and experienced physicians as well as junior and senior dermatologists.

Contributors are from 14 different countries, all of whom have a special interest and outstanding expertise in their particular field or disease, confirming that this book in the series is an international effort. I am very grateful to my mentor Professor Rod Hay and my colleague John Berth-Jones for encouraging me to take on this task.

London, UK — Wanda Robles

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Influence of the New Environment on the Skin

Environmental Related Skin Disorders in Immigrants and Tourists

A. L. Nguyen and S. Badeloe

Key Points

- A new environment can induce a wide variety of skin disorders in people travelling between different global climates (tropical, arid, temperate, cold, and polar).
- Skin disorders can occur in travellers due to interaction between genetic and biological traits of the individual, on the one hand, and changes in environmental factors (i.e. physical factors, biological and immunological factors, social and cultural factors), on the other hand.
- Physical environmental factors in a climate include low humidity and dry environment, hot and humid environment, sunlight/ultraviolet radiation, cold environment, and water hardness.
- Changes in biological and immunological environmental factors can induce new infectious skin disorders in travellers, such as varicella zoster virus infection and strongyloidiasis.
- Social and cultural factors may lead to skin problems in the new environment; this includes habits such as sunbathing and skin bleaching.
- Climate change causes global warming, leading to higher average temperatures, changes in humidity and precipitation. This can lead to increased cutaneous infectious diseases, marine dermatoses, pollution-induced or aggravated skin disorders, and skin cancer.

Introduction

Worldwide travel has increased enormously; according to the United Nations World Tourism Organization, the estimated tourist arrivals were 25 million in 1950, which has increased 56-fold to 1.4 billion international arrivals per year in 2018. Europe is the most important touristic region and accounts for about half of the international travel, followed by Asia & Pacific (24.5%), America (15.5%), Africa (4.8%), and Middle East (4.6%) [1]. Not only tourists but also business travellers, researchers, volunteers, immigrants, refugees etcetera contribute to the worldwide travel, which can occur between different climatic zones across the globe.

The term ‘climate’ encompasses the composite or generally prevailing weather conditions in an area over a long time such as temperature, air pressure, humidity, precipitation, sunshine, cloudiness, and wind [2]. Global climates can be

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divided into five types according to the Köppen-Geiger climate classification system: tropical (A), arid (B), temperate (C), cold (D), and polar (E) (Fig. 1). In tropical climate, the mean temperature of all months exceeds 18 °C (64.4 ° F) combined with significant amount of precipitation. An arid climate is hot (mean annual temperature ≥ 18 °C) or cold (mean annual temperature < 18 °C) with little precipitation. A temperate climate has a temperature between 0 °C (32 ° F) and 18 °C in the coldest month and a temperature of >10 °C (50 °F) in the hottest month and with a variable amount of precipitation determining the dryness of a season. A temperate climate can be subdivided into several subclimates such as Mediterranean climate with dry summers, humid subtropical climates with hot humid summers and mild winters, and oceanic climates without a dry season. Cold climate has a temperature of >10 °C in the hottest month and ≤ 0 °C in the coldest month, which can be further subdivided in numerous subclimates without dry season, with dry summers, and dry winters. In polar climate, the temperature in the hottest month is ≤10 °C. Figure 1 illustrates different type of climates for all countries across the world according to the Köppen-Geiger climate classification [3].

All types of travellers including immigrants, refugees, tourists can travel across the world between countries with different climatic zones, for instance they can travel from a (sub)tropical climatic zone to a temperate, cold or polar climatic zone, and vice versa. Travelling between these five climatic zones can induce numerous infectious and non-infectious skin disorders due to several environmental factors, which can be subdivided in:

1. Physical factors, e.g. ambient temperature (heat, cold), degree of humidity (dryness, moistness), amount of precipitation, sunlight, ultraviolet (UV) radiation, and water hardness.
2. Biological and immunological factors, e.g. micro-organisms.

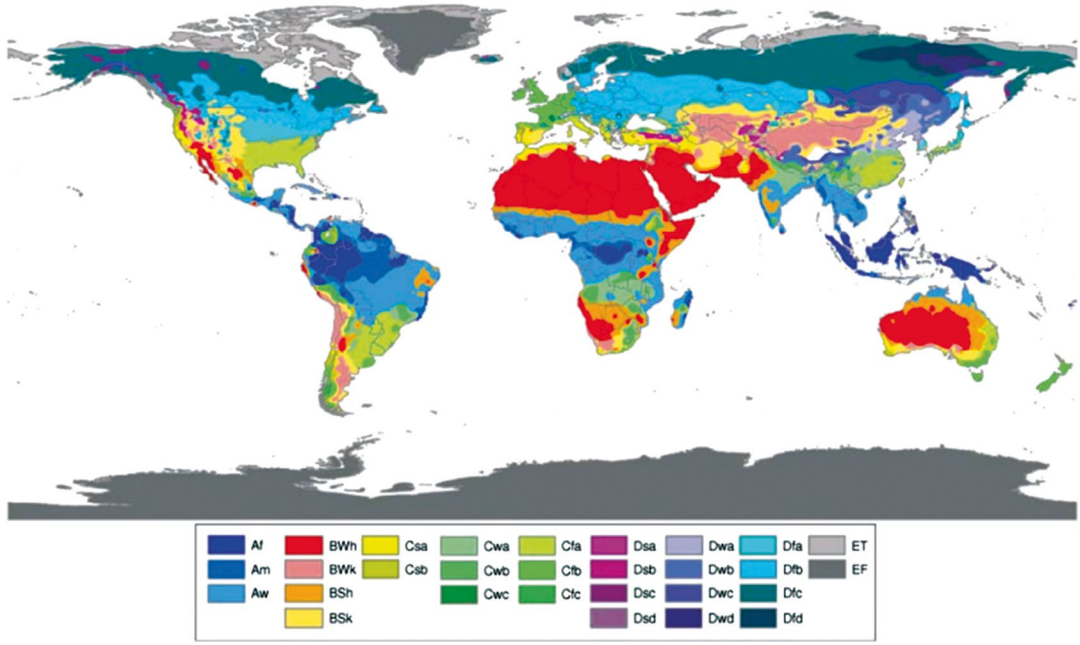

Fig. 1 Köppen-Geiger climate classification (1980–2016). Main climates: (**a**) (tropical): f (rainforest), m (monsoon), w (savannah) (**b**) (arid): W (desert), S (steppe); temperature h (hot) or k (cold) (**c**) (temperate) and (**d**) (cold): s (dry summer), w (dry winter), f (without dry season); temperature a (hot summer), b (warm summer), c (cool summer), d (very cold winter) (**e**) (polar): f (frost), T (tundra). Figure was adopted from *Peel* et al [3]

3. Social and cultural factors, e.g. cultural habits such as bathing habits and skin bleaching.

Other non-environmental factors determining the development of skin diseases are the structure and function of the skin, genetic constitution, and psychological factors, including behaviour. The interaction between factors present in a certain climatic zone, on the one hand, and biological traits and behaviour of the individual, on the other hand, can induce 'new' skin disorders in individuals that originated from different climatic zones.

This chapter discusses skin disorders induced by change in environment and climatic zone or to be more precise: the lack of adaptation to the new environment. We will also discuss specific skin disorders physicians should be aware of when treating immigrants and refugees, as they will experience a long-term and possibly a permanent change in climatic environment. In this chapter, we will discuss few disorders as an example of this topic. Tropical infectious disorders that can occur in (returning) travellers will be discussed in chapter "Cellulitis and Erysipelas".

Skin Function and Racial Differences

The skin primarily serves as a barrier against the external environment. The epidermis is the outer layer of the skin, which mainly consists of keratinocytes. It undergoes a continuous process of regeneration, proliferation, and desquamation. The skin barrier is maintained by formation of tight junctions and desmosomes between the keratinocytes. The stratum corneum is the outer layer of the epidermis and consists of corneocytes, lipids, and proteins. The tight lipid barrier and desmosomes between the corneocytes protect the skin, like a brick wall, to prevent trans epidermal water loss (TEWL) and penetration by micro-organisms (e.g. bacteria, viruses, fungi), allergens, toxins, and irritants. The corneocytes also contain a natural moisturising factor (a mixture of hygroscopic amino acids resulting from filaggrin breakdown), which is essential for skin hydration, water retention within the stratum corneum, and plasticity of the skin. An impaired skin barrier function is associated with increased TEWL and low corneum water content, which leads to dry skin. Another main function of the skin is thermoregulation. Skin blood flow plays an important factor in thermoregulation; vasodilatation and vasoconstriction of the cutaneous vasculature help regulate heat loss. Evaporation of water through skin and respiratory tract reduces excess heat from the body. However, in hot and humid conditions evaporation can become ineffective in releasing heat into the environment.

Structure and function of the skin in people originating from tropical countries are in some aspects different compared to people from a temperate, less sunny climate. Skin colour is a complex adaptive trait facilitated by a particular environment: natural selection influences genetic and phenotypic diversity in humans. In a wide range of geographical populations, several genetic differences associated with skin colour variation have been identified [4]. Skin colour is highly correlated with geographical latitude and ultimately UV radiation, therefore people living closer to the equator (0° latitude) tend to have darker pigmented skin [4, 5]. Darker skin contains larger and more numerous melanosomes, which are more evenly distributed throughout the epidermis, and also has a higher eumelanin to pheomelanin ratio. Eumelanin absorbs lights, prevents the formation of free radical species, protects against UV light, and lowers the risk of UV induced carcinogenesis [6]. Lighter skin has a lower melanin content, smaller melanosomes mostly located (supra)basal in the epidermis, higher pheomelanin content, and higher penetration of UV light to the upper dermis. People living at higher latitudes, north or south of the equator, tend to have paler skin to ensure adequate absorption of UV rays to maximize vitamin D synthesis in the basal layer of the epidermis, however they are also more prone to phototoxic reactions and UV-induced carcinogenesis [4–6]. In addition to skin colour variation in humans across the world, other anatomical differences related to skin type have been identified. For

instance, the TEWL appears to be greater in black skin compared to white skin and the desquamation rate is 2.5 times higher compared to Caucasians and Asians [7]. Also the stratum corneum of black people appeared to be more compact than that of white people, reflecting a stronger intercellular cohesion [5]. This may be responsible for the fact that continuous scratching in black people often leads to lichenification. Black skin also contains larger mast cell granules compared to white skin, which could account for differences in pruritus sensation between these groups [7]. The skin surface pH is lower in black people, and they appear to have larger apocrine sweat glands and in greater numbers compared to white people [5, 7]. Racial differences with regard to other skin properties such as water content, corneocyte variability, blood vessel variability, elastic recovery, lipid content, and surface microflora are inconclusive and often contradictory [7].

Skin Disorders Due to Physical Environmental Factors

A seasonal variation is often seen in a variety of skin disorders such as atopic dermatitis, psoriasis, acne, miliaria, polymorphic light eruption, lupus erythematosus, fungal dermatitis, and perniosis [2]. The conditions of a certain climate such as temperature and humidity influence skin disorders by affecting the skin barrier function. Several studies have reported that low humidity causes a decrease in skin hydration, initial increase in TEWL, decreased elasticity of the skin, increased dryness of skin and complaints such as pruritus and irritation [8]. Eventually, the TEWL will decrease under dry conditions and declining temperatures [9]. Furthermore, studies have shown that skin symptoms improve with increasing absolute and relative humidity. A low temperature decreases skin hydration, which leads to dry skin and increased sensation of itchiness [8]. Also short- and long-term effects of change in climate for, respectively, tourists and immigrants can influence symptoms of atopic dermatitis, for instance flares of atopic dermatitis occur more frequently under cold and dry weather conditions. A higher prevalence of paediatric eczema was seen in American states with low humidity, low UV exposure, low outdoor temperature, indoor heating, and increased precipitation. The combination of high UV exposure and high outdoor temperature with low indoor heating demonstrated a protective effect against eczema, whilst combined high humidity and precipitation with low UV exposure were associated with increased eczema prevalence [10].

In this paragraph, we focus on skin disorders that can develop in all types of travellers (including immigrants, refugees and tourists) due to a change in climatic zone, for instance from a cold climate to a (sub)tropical climate and vice versa. We shall discuss a few examples of skin disorders that can develop due to different physical environmental factors in a certain climate, such as low humidity and dry environment, a hot and humid environment, sunlight / UV radiation, cold environment, and water hardness (Table 1).

Low Humidity and Dry Environment

Dry Skin and Asteatotic Eczema

Dry skin or xerosis is one of the most common skin disorders in immigrants originally from a (sub)tropical climate migrating to a temperate climatic zone. Dry skin and subsequently asteatotic eczema (eczéma craquelé) can develop very soon after arrival, especially during wintertime due to low environmental humidity caused by cold, dry weather, and central heating. Dry skin can also develop or worsen due to prolonged and excessive showering and bathing, especially with hot water and excessive use of soap. Racial differences in skin properties of immigrants can also explain certain differences in susceptibility to the development of dry skin or asteatotic eczema. The increased TEWL in black skin could explain the increased frequency of xerosis cutis in individuals with black skin [7].

The signs of the dry skin syndrome range from mild to severe: dry scaly skin, ichthyosiform skin (Fig. 2), and asteatotic eczema (mildly infiltrated erythematous scaling plaques with

Table 1 Skin disorders related to physical environmental factors

Low humidity and dry environment	Hot and humid environment	Sunlight/ultraviolet radiation	Cold environment
Itch Dry skin Ichthyosiform skin Asteatotic eczema	Miliaria Bacterial infections Viral infections Fungal infections Yeast infections Parasitic infections Mycobacterial infections	*Idiopathic, probably immunologically mediated photodermatoses* – Polymorphic light eruption. – Juvenile spring time eruption. – Solar urticaria. – Chronic actinic dermatitis. – Actinic prurigo. – Hydroa vacciniforme. *Photodermatoses caused by exogenous sensitizers* – Phototoxicity including phytophotodermatitis. – Photo-allergy contact dermatitis. *Photoaggravated dermatoses*	Raynaud phenomenon Livedo reticularis Acrocyanosis Erythrocyanosis Perniones (chilblains) Cold erythema Cold urticaria Cold agglutinins Cold panniculitis Cryoglobulinaemia

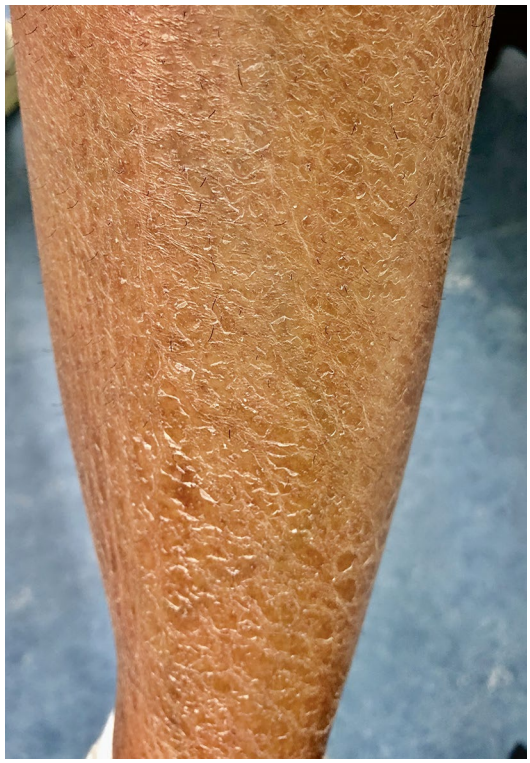

Fig. 2 Ichthyosiform skin

characteristic polygonal cracks and superficial fissuring). If the skin is dark, the erythema cannot be identified. Symptoms are a 'dry feeling', pruritus (sometimes severe and even causing sleep disturbances), and sometimes pain. The disorder most commonly involves upper and lower extremities, but can be localized anywhere on the body including the face, especially the lips. This diagnosis can be easily made on clinical grounds. However, it should be differentiated from other types of eczema, e.g. contact and atopic dermatitis, as other forms of eczema can be worsened by dry skin. A secondary infection can occur as a complication of the eczema.

Management of dry skin consists of humidifying the environment and adjusting bathing habits. Patients should decrease the frequency and duration of showering, use lukewarm (not hot) water, use oily-based products instead of soap, gently dry the skin with a towel; patting is better than rubbing and use of a hydrating ointment after drying the skin. Emollient ointments, with or without urea, should be used abundant and multiple times a day and continued as maintenance therapy to prevent recurrences. The eczema needs intermittent treatment with a topical corticosteroid ointment (class 2 or 3).

Hot and Humid Environment

We will discuss miliaria as an example of skin disorder that can occur in travellers in a hot and humid environment. A hot and humid environment can also induce skin disorders caused by bacteria (e.g. pyoderma, impetigo, erysipelas/cellulitis), fungi and yeast infections (e.g. pityriasis versicolor, mycoses), these will be discussed in chapter "Ecthyma".

Miliaria

Miliaria (prickly heat) is a disorder caused by blockage of the eccrine sweat duct. Miliaria is a common disorder in tourists visiting a hot and humid climate. It can develop within a few days after arrival. Three subtypes of miliaria can be distinguished dependent on the level of obstruction:

1. Miliaria crystallina: sweat duct blockage is located in the stratum corneum. It presents with thin-walled superficial, clear vesicles of 1–2 mm, that easily rupture. There is no inflammation or erythema. The lesions are asymptomatic.
2. Miliaria rubra: sweat duct blockage is located in the epidermis. This the most common type, which is characterized by itchy or stinging non-follicular papules or papulovesicles, on erythematous background. Miliaria pustulosa is a variant in which pustules form. A secondary bacterial infection, usually caused by staphylococci, can occur and may lead to sweat gland abscesses.
3. Miliaria profunda: sweat duct blockage is located at the dermo-epidermal junction, causing leakage of sweat in surrounding tissues. This presents as erythematous to skin-coloured non-follicular firm papules of 1–4 mm. Due to deep obstruction of the sweat ducts little to no sweating occurs at affected sites.

The lesions are most commonly localized in friction areas with clothing or occluded sites such as flexural areas, head-neck region, and upper trunk. Sweating is the most important means of heat regulation in a hot environment and blockage of sweating can cause hyperthermia, heat exhaustion and eventually heatstroke with malaise, nausea and vomiting, tachycardia, dyspnoea, and ultimately cardiovascular collapse. Miliaria is generally easily diagnosed; however, it must be differentiated from folliculitis, herpes simplex virus, varicella, acne, or insect bites.

Management of miliaria is primarily aimed at minimizing sweating and obstruction of sweat ducts by means of a cooler (air-conditioned) environment, cool baths, or showers combined with daily gentle exfoliation and wearing breathable non-occlusive clothing. Miliaria rubra can be treated with topical corticosteroids to decrease the pruritus and inflammation. Topical or oral antibiotics can be needed in case of a secondary bacterial infection. These disorders normally disappear within a few days after arrival in a cooler climate.

Sunlight/UV Radiation

Solar radiation is composed of three components of the electromagnetic spectrum: UV radiation, visible light, and infrared radiation. UV radiation can be subdivided based on wavelength into UVA (400–320 nm), UVB (320–290 nm), and UVC (290–200 nm). A higher wavelength can penetrate deeper into the skin. During a holiday in a (sub)tropical climate, short-term effects of UV radiation can cause sunburn (UVB > UVA) and tanning (immediate tanning: mostly UVA; delayed tanning: UVB). UVB exposure induces epidermal hyperplasia, which relatively protects the skin from UV radiation. Furthermore, UV radiation also leads to vitamin D synthesis, pro-inflammatory responses, and immunosuppression. UVC is filtered by the atmosphere and very little reaches the earth's surface. Photodermatoses can develop due to exposure to specific wavelengths of electromagnetic radiation. Skin disorders caused by solar radiation in tourists, and other travellers can be categorized in three main groups: idiopathic or immunologically mediated photodermatoses, photodermatoses caused by exogenous sensitizers, and photoaggravated dermatoses.

Idiopathic, Probably Immunologically Mediated Photodermatoses

The group of idiopathic, probably immunologically mediated photodermatoses comprises of the following more common disorders such as polymorphous light eruption, solar urticaria, chronic actinic dermatitis and actinic prurigo. As an example, we will discuss the occurrence of polymorphous light eruption in travellers.

Polymorphous Light Eruption

Polymorphous light eruption is a recurrent delayed-type hypersensitivity response to sunlight. It usually occurs in the spring and early summer in temperate climates, but it can also occur after sudden intense sun exposure during a holiday in a (sub)tropical climate. The occurrence is more frequent at higher latitudes. In the United States, blacks more frequently have polymorphous light eruption than Caucasians [11–13]. Symptoms present as symmetric pruritic skin-coloured to red papules, papulovesicles, and plaques on sun-exposed skin. In darker skin types, 1–2 mm pinpoint papules a more frequently seen, which constitute a morphologic pinpoint variant of polymorphous light eruption. In case of initial sunlight exposure, these symptoms can develop after 30 min up to 2–3 days, whilst after repeated sun exposure it can already present as soon as after 10 min up to several hours. After avoidance of sunlight, the skin eruption usually resolves in a few days to 2 weeks. The differential diagnosis consists of solar urticaria, cutaneous lupus, and photoallergic contact dermatitis.

The treatment for polymorphous light eruption consists of sun avoidance and adequate photoprotection (e.g. broad spectrum photoprotection, hats, clothing). It can also be preventively treated through photohardening of the skin, preferably with narrow-band UVB in early spring. A mild to moderate eruption can be treated with topical corticosteroids, whilst a severe eruption may require short courses of oral corticosteroids, antimalarial agents (i.e. hydroxychloroquine), or in rare cases azathioprine or ciclosporin.

Photodermatoses Caused by Exogenous Sensitizers

Travellers visiting a (sub)tropical climatic zone are exposed to a greater amount of UV radiation, which could lead to photosensitivity due to exogenous drugs and chemicals that can present as phototoxicity or a photoallergy.

Phototoxicity develops after exposure to UV radiation (UVA > UVB) and a photosensitive agent that has been ingested or applied to the skin, which causes direct tissue and cellular damage to the skin through a non-immunological mechanism. These reactions present as exaggerated sunburn, usually appear within minutes to hours after sun exposure. In severe cases, vesicles and bullae may develop. A phytophotodermatitis is a form of phototoxicity caused by contact with photosensitizing substances in plants. It is mainly caused by furocoumarin-containing plants such as (giant) hogweed, parsnip, dill, fennel, parsley, celery, lime, lemon, and fig. This can induce a reaction consisting of pruritic, burning or painful erythema, oedema, vesicles, and bullae in linear streaky configurations on sun-exposed skin, healing with post-inflammatory hyperpigmentation.

Photoallergy is a delayed-type hypersensitivity reaction to a photoallergen on sun-exposed skin. This type develops after 24–48 h of sun exposure and usually presents as a pruritic eczematous eruption on sun-exposed areas.

The extensive list of photosensitive drugs includes tetracyclines (especially doxycycline), diuretics, nonsteroidal anti-inflammatory drugs, sulphonamides, metformin, amiodarone, hydroxychloroquine, psoralens, retinoids, and azathioprine. Examples of topical photosensitizing agents are sunscreens, fragrances, nonsteroidal anti-inflammatory drugs, dyes, and psoralens. Phototests and photopatch test can be performed in order to identify the offending agent. These photosensitivity reactions should be differentiated from chronic actinic dermatitis, porphyria cutanea tarda, lupus erythematosus, or solar urticaria.

Management consists of cessation or avoidance of the offending drug or chemical, sun avoidance and adequate sun protection with broad-spectrum sunscreens and (UV-)protective clothing. Symptomatic treatment with emollients and topical corticosteroids may be needed to reduce pruritus and the inflammatory response.

Photoaggravated Dermatoses

Photoaggravated dermatoses are skin disorders that can be exacerbated by UV radiation or visible light. A change in climate to a (sub)tropical or sunny temperate climatic zone can cause exacerbation of existing skin disorders due to a greater exposure to sunlight. The list of photoaggravated

dermatoses is extensive and includes acne, atopic dermatitis, bullous pemphigoid, disseminated superficial actinic porokeratosis, erythema multiforme, Hailey-Hailey disease, Darier disease, dermatomyositis, Grover's disease, herpes simplex infection, lichen planus, lupus erythematosus, melasma, pemphigus, pityriasis rubra pilaris, and rosacea. Some skin disorders such as atopic dermatitis generally improve with sun exposure and also respond to treatment with phototherapy; however, a minority of patients with atopic dermatitis can experience aggravation due to sun exposure.

Management of these photodermatoses consists of adequate photoprotection and treatment of the underlying skin disorder.

Lack of Exposure to Sunlight and UV Radiation

Lack of exposure to sunlight and subsequent UVB radiation can lead to a vitamin D deficiency, which is common in Europe and the Middle East [14]. Non-western immigrants or refugees usually have darker skin, which requires more UVB to produce vitamin D. A higher latitude, winter season, and shorter duration and exposure to sunlight negatively influence the amount of vitamin D that can be produced in the skin [15]. A poor vitamin D status was observed in non-Western immigrants, therefore it has been advocated that vitamin D supplements should be considered in order to prevent the consequences of vitamin D deficiency such as rickets in children and osteomalacia in adults [14]. It is well known that sunlight exposure can reduce disease severity in several skin disorders such as atopic dermatitis, psoriasis, and vitiligo, whilst the lack thereof can lead to exacerbations [16].

Psoriasis

Psoriasis is a common chronic relapsing inflammatory skin disorder with a genetic component. Worldwide prevalence is estimated at approximately 1–3%; however, the prevalence is quite variable depending on ethnicity and geographic areas. The prevalence rate appears to be higher in whites compared with non-white ethnic groups. It has been noted that psoriasis is more common in people from South Asian descent than people from African descent. Also, within Africa, a wide variation has been reported with higher prevalence in Eastern Africa (1.9–3.5%) compared to western Africa (0.025–0.9%) [17]. Overall, psoriasis is more common in the colder northern climates than in tropical climates [18]. The clinical course of psoriasis in immigrants could be negatively influenced by environmental triggers in the new environment such as cold weather, low humidity, less exposure to sunlight and UV radiation, and psychological stress related to life in a new environment. Immigrants from (sub)tropical countries can also have their first episode of psoriasis after arriving in a temperate climate.

Chronic plaque psoriasis, the most common form of psoriasis, is characterized by sharply demarcated indurated erythematous plaques with a coarse silvery scale. The typical localizations are the scalp, extensor sides of elbows, knees, and sacral region, but lesions can appear on virtually any part of the body. In 30% of patients, the lesions are not or slightly itchy. In black patients, psoriasis lesions are less erythematous, more violaceous, or hyperpigmented, plaques appear to be thicker with more scaling and affected body surface area tend to be more extensive. In dark skin, active lesions more frequently resolve with hyper- or hypopigmentation, which can make it difficult to distinguish active lesions from post-inflammatory dyspigmentation [17]. The clinical characteristics are usually sufficient to diagnose psoriasis. However, darker skin can make it challenging to determine the right diagnosis; post-inflammatory hyperpigmentation, nummular eczema, neurodermatitis circumscripta, dermatophytosis, lichen planus, and parapsoriasis should be considered. Histopathological investigation can sometimes be helpful.

Treatment for psoriasis is diverse and consists of topical therapies (corticosteroids, vitamin D analogues, combination corticosteroid-vitamin D analogues, calcineurin inhibitors), phototherapy, systemic medications, and biological therapies. Physicians should be aware of the country of origin and travel history of immigrants and refugees

as they should be adequately screened for various endemic infectious diseases such as tuberculosis, human immunodeficiency virus, hepatitis B and C before starting immunosuppressive therapies.

Cold Environment

A cold, damp, and non-freezing environment with a shortage or lack of sunlight (visible and UV-radiation) can induce new skin disorders or worsen existing skin disorders in immigrants from (sub)tropical countries moving to a temperate climatic zone.

Cold-induced disorders consists of acrocyanosis, perniosis (chilblains), Raynaud's phenomenon, livedo reticularis, cold urticaria, cold panniculitis, and cryoglobulinemia. As an example, we will discuss perniosis in travellers. Other cold-induced skin disorders will be discussed in chapter "Cold Injuries".

Perniosis

Perniosis presents as cold-induced erythrocyanotic lesions on skin vulnerable to cold exposure. It is caused by an abnormal vascular reaction to cold in probably genetically predisposed persons. Immigrants or refugees from (sub)tropical countries, not using gloves and wearing inadequate footwear in cold seasons are prone to developing perniosis.

It is a common disorder that occurs during the cold months of the year. The clinical signs are erythematous to blue-violet macules, papules, plaques, or nodules on the hands or feet. It can also affect the nose, ears, calves, thighs, or buttocks. Sometimes blisters and ulceration can occur. The symptoms are pruritus, burning, and pain. It must be differentiated from other cold-induced disorders with similar symptoms, like acrocyanosis, chilblain lupus erythematosus, and lupus pernio (a variant of sarcoidosis). It typically resolves in 1 to 3 weeks.

The management of perniosis is primarily targeted at avoiding exposure to cold by wearing adequate and warm gloves, clothes, and footwear. In recalcitrant cases, treatment with nifedipine, a calcium antagonist with vasodilatory properties, can be effective in terms of accelerated clearance of existing skin lesions and prevention of new perniosis lesions [19].

Water Hardness

Hard water contains a high mineral content, typically calcium and magnesium ions. It has been hypothesized that exposure to hard water aggravates atopic dermatitis, as high concentrations of calcium and magnesium act as chemical irritants which can cause irritation and dryness of the skin [20]. Travellers moving to another country can be exposed (short or long term) to domestic water with a higher degree of water hardness. Several studies have demonstrated that higher water hardness is associated with an increase in atopic dermatitis during childhood [20–22]. Two studies have demonstrated that children exposed to hard water have an increased risk at atopic dermatitis when they have a positive atopic status or a filaggrin mutation [23, 24]. However, an observer-blinded randomized controlled trial of 12 weeks conducted amongst 336 children with moderate to severe atopic dermatitis supplied by hard water demonstrated no additional benefit of an ion-exchange water softener, regardless of filaggrin mutation [25]. Further prospective studies regarding this topic are needed.

Skin Disorders Related to Biological and Immunological Factors

The immune system, including the so-called skin immune system, plays a major role in defending the body against microbial intruders [26]. In a new environment with micro-organisms in the ecosystem which are immunologically unknown to the traveller, infectious diseases including skin infections can develop, which otherwise would not appear in the old environment. The so-called hygiene theory hypothesizes that exposure to pollutants or infectious diseases in poorer less developed nations

would protect against atopy [27]. According to this theory, epidemiological and laboratory studies have implied that the environment during early childhood is important for the risk of developing atopic disorders [28].

Skin disorders related to immunological and changed biological environment such as the occurrence of varicella zoster virus infection and strongyloidiasis in travellers such as immigrants and refugees will be discussed.

Varicella Zoster Virus (VZV) Infection

A primary VZV infection results in varicella (chickenpox) and is a highly contagious disease. In temperate, industrialized countries, it is a very common and self-limiting disease amongst healthy children [29, 30]. It has been reported that about 90% of primary VZV infections occur in children under the age of 10 years. It is more common in cooler winter and spring months. The incidence of varicella amongst children is lower in tropical climates compared to temperate climates. In the tropics, it more often develops in adolescents and adults. About 30% of individuals from tropical regions are susceptible to varicella at 20 years of age and 5–10% remain susceptible at 30 years of age [29]. Varicella outbreaks have been reported in immigrant populations in temperate climates. In a group of Tamil refugees in Denmark, 38% of the adults and 68% of the children developed varicella in the first few months after arrival due to lack of immunity [30]. Varicella is more severe in adults than in children and is associated with significant morbidity (e.g. secondary bacterial infection, pneumonitis, hepatitis, encephalitis) and mortality. Non-immune female immigrants or refugees are also at risk of acquiring varicella during pregnancy, with potential complications for mother and baby. Congenital varicella syndrome may develop during the first 20 weeks of pregnancy and neonatal varicella during 5–7 days before delivery to 2 days after, with a reported mortality of 30%. Therefore, serological screening of immigrants and refugees without a self-reported history of varicella and subsequent vaccination of seronegative immigrants and refugees has been advocated [29].

Varicella is spread by droplet-airborne transmission or by direct contact of skin and mucosa with the contents of blisters. The incubation period ranges between 10 and 21 days. After a prodromal phase of 2–3 days with fever, malaise and flu-like symptoms, the skin eruption appears. It is characterized by erythematous macules, papules, vesicles, pustules, and crusts, which present in different stages of development. It occurs mainly on scalp and face, which can progress to trunk, extremities, oral mucosa, and occasionally on other mucous membranes such as conjunctiva and genitalia. Total healing takes about 2–3 weeks. The eruption can be extremely itchy. On a dark skin, the initial erythematous macules are obscure, and after healing 'polka dot', hyperpigmented scars can be present for many months and sometimes even years.

In immigrants and refugees, with an unknown varicella history or serologic status, a varicella infection should be distinguished from a disseminated herpes zoster or herpes simplex infection. Herpes zoster infection (shingles) is caused by reactivation of latent VZV in sensory ganglia and most frequent occurs in older adults and immunocompromised individuals. Typically, this presents as an intense painful unilateral progressing erythematous macules, papules, and vesiculopustular eruption following a dermatomal distribution. In case of a disseminated herpes zoster infection, several dermatomes are involved or lesions can develop at distance from the primary affected dermatome. Early herpes zoster can occur in infants who have been exposed to VZV in utero or postnatally. A definite diagnosis can be made by performing polymerase chain reaction of vesicle fluid or by serological antibody assessment.

Non-immune adolescent or adult immigrants and refugees with uncomplicated varicella should be treated with oral antiviral therapy (e.g. acyclovir, valacyclovir) to reduce the severity of symptoms and risk of complications. Pregnant women, newborns, and immunocompromised individuals should be treated with intravenous acyclovir. In healthy children $\leq$12 years, varicella is typically self-limited old and therefore generally does not require antiviral treatment [31].

Strongyloidiasis

Strongyloidiasis is caused by an infection with the parasite *Strongyloides stercoralis*. About 30–100 million are estimated to be infected worldwide, however, this might be an underestimation. Strongyloidiasis occurs in warm, especially damp, climates and is endemic in (sub) tropical areas such as South-East Asia, sub-Saharan Africa, Latin America, mainly in the rural areas. Sporadically this also occurs in temperate areas such as North America, Southern Europe, Japan, and Australia. In the United states, the infection rates are high amongst those who resided in endemic areas (including immigrants, refugees, travellers, and military personnel) [32]. High risk of exposure to *S. stercoralis* is considered in immigrants from endemic areas, adopted children who have been living ≥1 year in a highly endemic area, and expatriates living >1 year in endemic countries and visiting rural areas [33].

Transmission of this infection occurs via skin contact with contaminated soil, faecal-oral transmission, and from person-to-person transmission through faecal contaminated fomites. More than half of cases can be asymptomatic or are associated with nonspecific symptoms. In an acute or chronic infection gastrointestinal symptoms, respiratory symptoms and dermatologic manifestations can occur. Thirty percent of patients have urticarial wheals and flares of migrating subcutaneous larvae anywhere between nipples and knees, particularly around anus and buttocks. The erythematous urticarial tracks can disappear within a day. Eosinophilia can occur up to two-thirds of a chronic infection. Chronic asymptomatic infection can sustain for decades due to the autoinfection cycle of the *S. stercoralis* in skin, lungs, and gastrointestinal tract within the human host. Immunosuppressed patients are at risk for developing a *S. stercolaris* hyperinfection (accelerated autoinfection within organs normally involved in autoinfection cycle) or disseminated infection (hyperinfection with spread of larvae to organs and tissues outside autoinfection cycle).

Serologic screening of asymptomatic individuals is warranted in immigrants and refugees from endemic areas, immunosuppressed patients, or candidates for immunosuppressive therapies with a high-intermediate risk to strongyloidiasis, epidemiologic exposure in military personnel [32, 33]. In patients with gastrointestinal symptoms additional stool testing (polymerase chain reaction or agar plate culture) can be performed, however, due to intermittent larval excretion the sensitivity is relatively low [32].

Treatment of strongyloidiasis consists of ivermectin 200 microgram/kg daily for 2 days, repeated at 2 weeks for immunocompromised patients or those who require immunosuppression. Amongst patients with hyperinfection or a disseminated infection, the mortality rate is 70–100% [18, 32].

Skin Disorders Related to Social and Cultural Factors

Different social and cultural factors related to an old environment can cause skin disorders in travellers moving between different climatic zones. For instance, sunbathing habits of tourists in (sub)tropical climates could lead to massive sunburn and subsequent complications. Whilst, veiled female immigrants or refugees wearing covering clothes often suffer from vitamin D deficiency in Western countries. We report on adverse reactions to skin bleaching as a common practice amongst travellers such as immigrants and refugees with skin of colour.

Adverse Reactions to Skin Bleaching

Worldwide, in several communities with dark-skinned inhabitants, a clear and light skin has been viewed as a cultural beauty ideal and deemed to represent a superior socio-economic status. Skin bleaching has been practiced in order to achieve this beauty ideal. Skin bleaching is therefore a common phenomenon in countries such as sub-Saharan Africa, Middle East, Asia (i.e. India, Philippines, Hong Kong), Southern, and Central America [34]. The skin bleaching practices are frequently continued as an "imported phenomenon" amongst immigrant populations in North America and Europe [35]. There are indications that due to "psychosocial pressure" the skin bleaching practise is intensified in the new environment by some individuals in certain

groups of immigrants. Mostly adult woman practice skin bleaching. The most common compounds that are used are hydroquinone (in several concentrations) and 0.05% clobetasol propionate. In this cosmetic context, these skin bleaching products can be obtained without a doctor's prescription from their country of origin, tropical convenience stores in immigrant countries, or online via the internet. The active ingredients may be indicated on the packages of the skin bleaching products, however, it can also be inaccurate or undocumented [34]. Inadequate use of skin bleaching products can lead to various adverse reactions.

Misuse of hydroquinone can lead to periorbital hyperpigmentation, irritant, and/or allergic contact dermatitis with subsequent post-inflammatory dyspigmentation and exogenous ochronosis. Exogenous ochronosis can develop due to long-term or excessive use of hydroquinone. It represents as reticulated and ripple-like sooty or blue-black pigmented macules, papules, and plaques on sun-exposed skin, also pigmented colloid milia can be seen. It must be differentiated from post-inflammatory hyperpigmentation and melasma. The histological picture is pathognomonic, with a dermal infiltrate and yellow-brown banana-shaped fibers in papillary dermis in the haematoxylin eosinophilic staining. Unfortunately, there is no effective treatment known for this disease.

Misuse of topical corticosteroids can induce local complications such as striae, skin atrophy, acne, folliculitis, purpura, persistent erythema, tinea corporis. Prolonged and excessive application of topical corticosteroids, especially on large body surface areas, can lead to systemic complications like hyperglycaemia, hypothalamic-pituitary-adrenal axis suppression, Cushing's syndrome an hypertension [35].

Awareness and adequate recognition by physicians of cosmetic skin bleaching practices amongst immigrants is important in order to prevent potential permanent local and systemic complications. Physicians should educate immigrants and refugees about the cutaneous and systemic complications including paradoxical hyperpigmentation of prolonged, extensive, and incorrect use of skin bleaching products. The use of these products should be immediately discontinued [35].

Climate Change and Skin Disorders

The global climate is changing. Skin disorders aren't only influenced by a change in environment due to travelling to another climatic zone, but they can also be affected by the overall change in the global climate and environment. Climate change describes the regional or global variation in climate over time in terms of temperature, humidity, precipitation, atmospheric pressure, cloud cover, and wind. Climate change has been considered to be the cause of global warming, leading to higher average temperatures, changes in humidity and precipitation. The average surface temperature has increased by 0.6 °C over the past 100 years and will probably increase by 2 °C by the end of 2100 [2]. Warming oceans, melting of arctic ice cap and rising sea levels contribute to more severe hurricanes and storms. Climate change overall also leads to a global rise in extreme climatic events such as floods, droughts, and wildfires [36]. These conditions are associated with increased cutaneous infectious diseases and expanding geographic range of vector-borne diseases (e.g. Lyme disease, malaria, leishmaniasis, dengue) [34], which will be discussed in chapters. "Ecthyma", "Boils or Furunculosis", and "Cellulitis and Erysipelas". Water-associated climate changes such as increasing ocean temperatures also cause increases in marine dermatoses such as jellyfish stings, seabather eruption, and Swimmer's itch [37], which will be further discussed in chapter "Marine Dermatoses". Environmental pollutants from fossil fuel emissions and wildfires can also negatively affect inflammatory disorders such as atopic dermatitis [36]. Ozone depletion increases UV radiation on earth, which leads to skin aging and increases the risk at skin cancer. It has been estimated that each 1% reduction in thickness of the ozone layer increases the incidence of melanoma by 1–2% and the risk for squamous cell carcinoma by

3–4.6% and for basal cell carcinoma by 1.7–2.7% [38]. Dermatologists should be aware that climate change could cause changing patterns in skin disorders.

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Part I

Dermatoses Caused by Infection: Bacterial Infections

Impetigo

Patricia Chang
and Monica Vanessa Vasquez Acajabon

Key Points

- Impetigo is the most common bacterial skin infection.
- Non-bullous impetigo and bullous impetigo.
- Primary and secondary impetigo.
- *Staphylococcus aureus* or *Streptococcus pyogenes.*
- Treatment: topical antibiotics such as mupirocin, retapamulin, and fusidic acid.
- For limited impetigo.

Impetigo, written for the first time in 1864 by Tilbury Fox [1], is an acute bacterial-origin dermatosis caused by *Staphylococcus aureus or Streptococcus pyogenes* that affects the superficial layers of the epidermis [2, 3]. It is also called impetigo contagious, impetigo bullosa, or impetigo of Tilbury fox [4].

It is highly contagious and self-inoculable [4, 5], and a linear distribution secondary to scratching is frequently observed [6].

Impetigo is among the first five causes of dermatological consultation in children [2, 4, 5]. A higher prevalence is observed in malnourished and immunocompromised patients, but the main risk factor is the alteration of the integrity of the skin due to trauma, bites or insect bites, preexisting dermatoses, pyogenic infections, and poor hygiene [2, 4, 7, 8], as well as diseases that affect the host's immune status, such as diabetes mellitus, HIV, autoimmune, hematological diseases, chemotherapy, steroid and biological treatments.

As for the ethiopathogenesis, it can be caused by S. aureus and S. pyogenes or beta hemolytic group A, or both. In immunocompetent patients, 60% of impetigo is due to staphylococcus, 20% to streptococcus, and 20% to both [4].

Streptococcal infections are more prevalent in the summer and in tropical or humid climates [4, 6]. Staphylococcal impetigo primarily affects newborns and school children [2]. In the case of infants, the highest frequency is explained by the lack of specific antibodies to neutralize the staphylococcal exfoliative toxin that acts against desmoglein (DSG) [4], which causes blisters due to epidermal separation. If it is very severe, it causes staphylococcal scalded skin syndrome (Ritter's disease) [4, 7, 8]. 60% of healthy people are staphylococcus carriers in nostrils, armpits, groins, and perineum, so their frequent participation as a primary site of infection is observed [8]. In addition, staphylococci of phage type 71 may reduce the population of group A streptococci [4].

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There are two main types of impetigo: the non-bullos which occurs in 70% of cases and is also called contagious impetigo, and the bulloso, which occurs in 30% of cases [9].

Non-bullous impetigo can also be classified as primary or secondary, which is the most common one [5, 6]. It is generally caused by S. aureus, but S. pyogenes may also be involved, especially in warm, humid climates (Table 1).

It is clinically characterized by blisters and pustules, which erode and subsequently dry out quickly and are covered by meliceric scabs. They are classified as primary and secondary impetigo, and according to their cause and morphology, in blister or staphylococcal, and crusty or streptococcal [3, 4].

Primary impetigo occurs by direct bacterial invasion of previously normal skin [6] and is mainly located on the face, around the natural holes: mouth, nostrils, ear pavilions, and eyes (Figs. 1, 2, 3, and 4) [2, 4], although in infants it predominates in the perineum, in the periumbilical region or in disseminated form [4, 8]. Vesiculopustular lesions predominate in the lower extremities, face, and can be extended by self-inoculation (Figs. 5, 6, 7, and 8) [4, 5, 7].

The secondary form, also called impetiginization, is preceded by an underlying pruritic dermatosis that alters the skin barrier [4, 6] (Figs. 9, 10, 11, and 12). It can appear on any part of the body. The lesions are erythema, blisters, pustules, and meliceric scabs [4]. The secondary form, also called impetiginization, is preceded by a pruritic dermatosis that alters the underlying skin barrier [4, 6]. Usually S. aureus is associated in secondary forms [6].

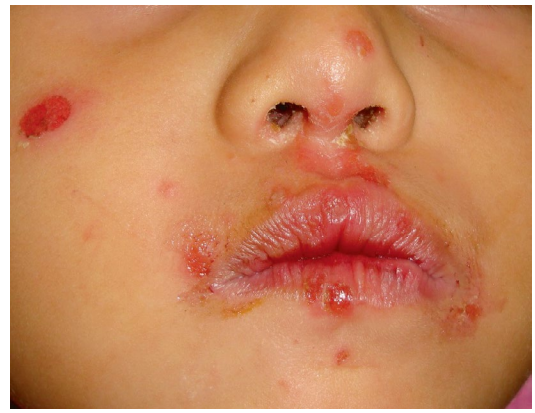

Fig. 1 Primary impetigo around natural holes

Table 1 Main types of impetigo

Bullous impétigo	Non-Bullous impetigo
Caused only by *S. Aureus*	*S. Aureus S. pyogenes*
Large, fragile, flaccid bullae that can rupture and ooze yellow fluid	Primary or secondary (more common form)
The pathognomonic collarette of scales on its periphery develops after the bullae rupture, leaving a thin, brown crust on the remaining erosions	Primary impetigo is a direct bacterial invasion of intact healthy skin
Exfoliative toxins produced by *S. Aureus* strains that cause loss of cell adhesion in the superficial epidermis That form the bullaes	Secondary impetigo is a bacterial infection of disrupted skin caused by trauma, eczema, insect bites, scabies, or herpetic outbreaks and other diseases. Diabetes or other underlying systemic conditions also increase susceptibility.
Trunk, axilla, and extremities, and in intertriginous (diaper) areas	Maculopapular lesions that transition into thin-walled vesicles that rapidly rupture, leaving superficial, sometimes pruritic or painful erosions covered by the classic honey-colored crusts
It is the most common cause of ulcerative rash on the buttocks of infants	Exposed skin of the face (nares, perioral region) and the extremities are the most affected sites
Systemic symptoms are uncommon but can include fever, diarrhea, and weakness	Systemic symptoms are unlikely
Topical antibiotics: Mupirocin, retapamulin, and fusidic acid	Topical antibiotics: Mupirocin, retapamulin, and fusidic acid
Resolve within 2 to 3 weeks without scarring	Resolve within 2 to 3 weeks without scarring

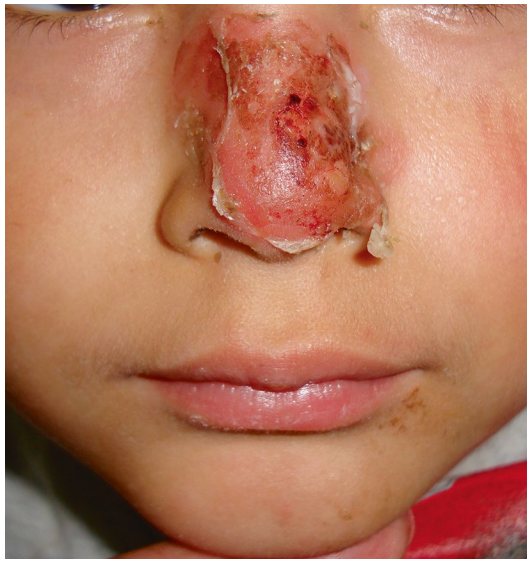

Fig. 2 Bullous impetigo on nose

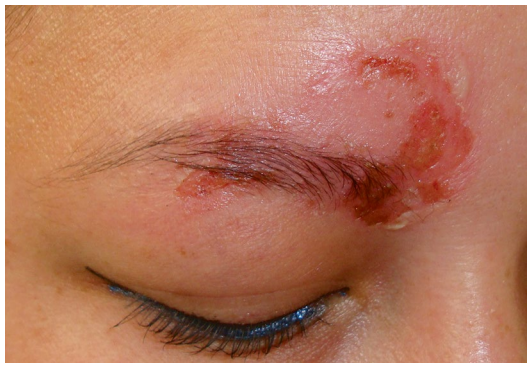

Fig. 3 Erosive pustular lesions on the right eyebrow

Some authors classify this entity as true blister or staphylococcal impetigo, which usually affects the face, buttocks, trunk, and perineum [1], and contagious impetigo of Tilbury Fox or non-bullous (the most common) [1, 4, 6, 7], the latter caused mostly by S. aureus and less frequently by S. pyogenes [8]. Other less frequent forms have been described, such as layer impetigo of the scalp, where there are scabs that agglutinate the hair; impetigo of the mucous membranes, consisting of erosive plaques on the lips and oral mucosa, and which, if affecting the corners, produces angular cheilitis and may be accompanied by phlyctenas keratitis; Circinated or geographical impetigo where squamous circles form; miliary impetigo in which micro vesicles and pyogenic intertrigos are observed [4].

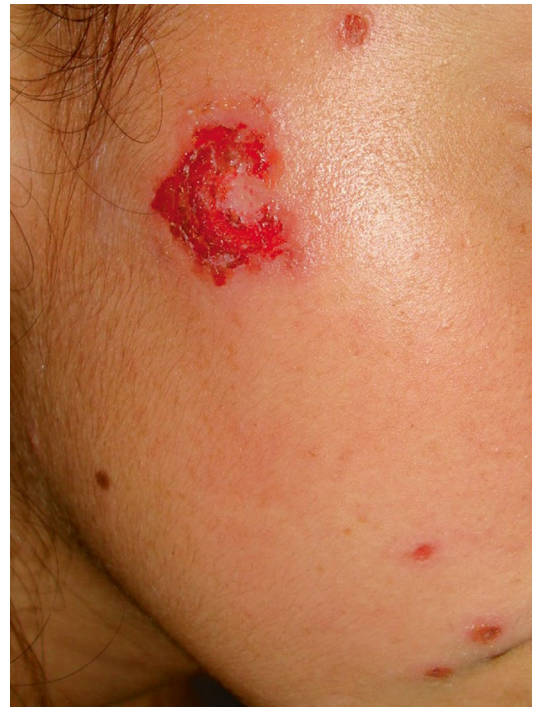

Fig. 4 Crusty lesions on cheek

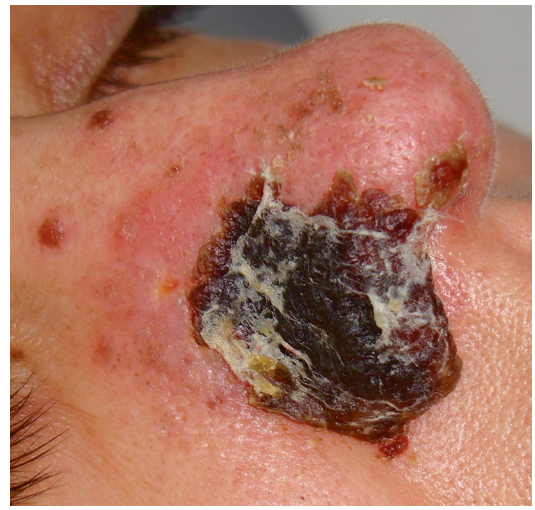

Fig. 5 Lesions with meliceric blood crust in a patient with sytemic lupus

The evolution is acute and tends to a spontaneous resolution in 2 to 3 weeks. It leaves an eroded skin that subsequently evolves to a pinkish spot,

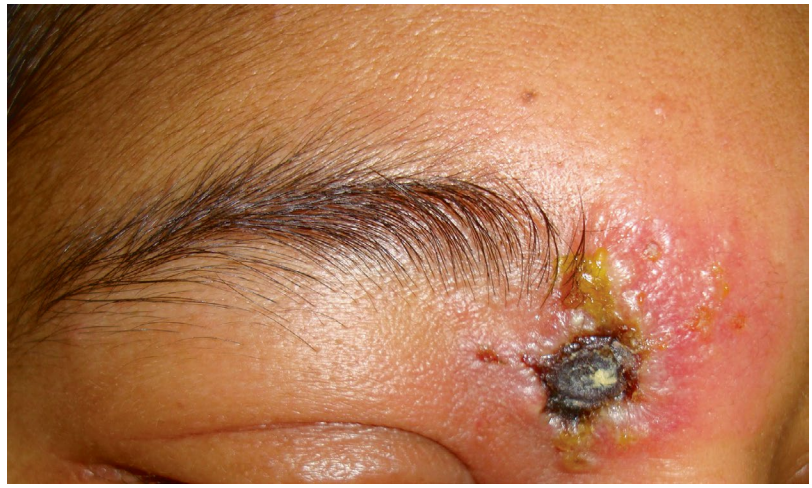

Fig. 6 Melicerous crusts surrounded by erythema on eyebrow

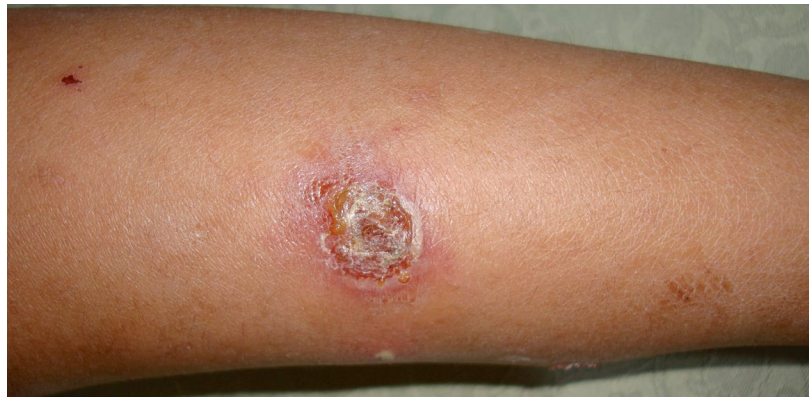

Fig. 7 Meliceric crust on leg

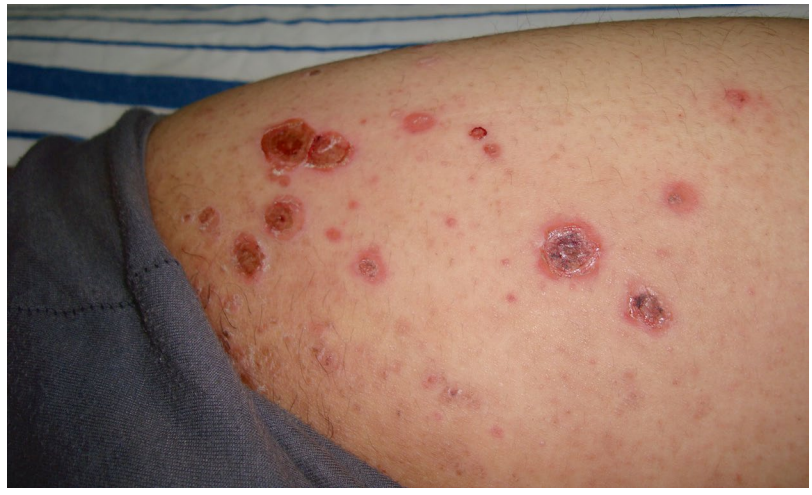

Fig. 8 Crusty lesions on leg