Allergic rhinoconjunctivitis, allergic asthma, and atop-ic eczema constitute the classical triad of atopic dis-eases. Whereas atopic respiratory disease can be diag-nosed with certainty by allergological methods, there is no definite marker of atopic eczema. Its diagnosis is based on clinical criteria: chronic or chronically relaps-ing, intensely pruritic, characteristically distributed eczematous skin lesions of variable morphology in the presence of an atopic diathesis.

22.1 Epidemiology

Epidemiologic studies on atopic eczema are hampered by the lack of a definite diagnostic marker, by the fluctuant course of the disease, and by the impact of environmental factors on disease manifestation. Available data indicate that atopic eczema is a major health problem, more prevalent among young people than among adults. Both sexes are affected, with a slight predomi-nance in females. Occurrence of the disease is associat-ed with the socioeconomic status.

Atopic eczema occurs worldwide and in all races. However, the International Study of Asthma and Allergies in Childhood (ISAAC) found a high worldwide variation of disease frequency ranging for younger children between 1% in Iran and 16% in Japan and Sweden. For 13- to 14-year-old children, prevalences were 1% in Albania and 17% in Nigeria. Overall, preva-lence seems to be higher in Australia and Northern Europe than in Asia and Central or Eastern Europe. Less data are available on the frequency of atopic ecze-ma in adults, point prevalences being about 1%–3%.

During the last decades the prevalence of the disease has increased considerably. For example, the cumula-tive incidence rate of atopic eczema among Danish twins up to the age of 7 rose from 3% for those born between 1960 and 1964, to 10% for those born between 1970 and 1974. Recent studies suggest that the increase could be leveling off.

The frequency of the extrinsic or intrinsic type of atopic eczema among patients seems to depend on numerous variables, but meaningful data are not yet available.

22.2 Clinical Presentation

Several phases of atopic eczema can be discerned: the infantile phase, up to 2 years; the childhood phase, up to 12 years; the adolescent/young adult phase, between 12 years and young adult life; and the late adult phase in older subjects. Itching is an essential and subjectively the most stressful feature in all phases of the disease.

Infantile atopic eczema not rarely develops already during the first three months of life. The disease starts on the face (cheeks, forehead) and scalp, with an ery-thematous and papulovesicular eruption, frequently developing to oozing and crusted lesions (cradle cap). Lesions may extend to involve other skin areas, particularly the extensor aspects of the limbs and the trunk. The course is chronically persistent or relapsing, healing may occur by the end of the 2nd year of life.

In the childhood phase, flexural eczema develops with involvement of the antecubital and popliteal spaces as well as of the wrists. Other sites of predilec-tion are the face, the neck, the retroauricular areas, and the backs of the hands and the feet. The disease may extend to involve the entire skin surface. The skin lesions are less exudative than in the infantile phase. On a conspicuously “dry” skin, there are patchy to dif-fuse erythema, papules, and excoriated scratch marks.
Especially in flexural regions, lichenifications or lichenoid prurigo nodules develop. In the adolescent and adult phases, the predilection sites are virtually the same, lichenified eczema now being the predominating type of lesion. Lymphadenopathy may be present in severe cases.

There are special manifestations of atopic eczema that can be divided into morphological and site-specific variants. They are found associated with classical disease manifestations or they occur in isolation, in which case they may pose diagnostic difficulties. Also, some of them are not unequivocally related to atopic eczema, demanding exclusion of other causes. Important morphological variants are the following: papular variant of atopic eczema, patchy pityriasisiform lichenoid eczema (follicular eczema), nummular atopic eczema, prurigo variant of atopic eczema, seborrheic atopic eczema, and pityriasis alba. Site-specific variants comprise, e.g., exfoliative and angular cheilitis, median fissuring of the lower lip, retroauricular intertrigo, infraauricular or infranasal fissuring, eczema of the lower eyelids, nipple eczema, pulpitis sicca, juvenile plantar dermatosis, and vulval eczema. UV-provoked atopic eczema also falls into the category of site-specific manifestations.

The burden of atopic eczema is enormous and usually underestimated. The disease influences psychological and social development and interferes with everyday activities as well as scholastic or professional achievements. Patients and family members or partners are affected by disease burden. There are also substantial financial costs to the patient and to society.

### 22.3 Histopathology

Atopic eczema is not a disease that can be diagnosed histologically, as the findings are nonspecific and shared by other forms of dermatitis or eczema. In acute lesions, there is spongiosis, acanthosis and parakeratosis, and the dermis shows a superficial, perivascular, predominantly lymphohistiocytic infiltrate with varying numbers of eosinophils.

Chronic lichenified lesions are characterized by a moderately dense lymphohistiocytic infiltrate around the vessels, varying thickness of the papillary dermis, sometimes acanthosis, and focal (hyper-)parakeratosis. Spongiosis is usually absent. The dermal infiltrate is dominated by macrophages; in addition there are eosinophils and T cells. Increased numbers of mast cells are present in the dermis.

### 22.4 Diagnosis

Diagnosis of atopic eczema is based on clinical criteria. Chronic or chronically relapsing, usually intensely pruritic eczematous skin lesions with a characteristic distribution in the presence of an atopic diathesis constitute the diagnosis. Age-related manifestation as well as morphological and site-specific variants are to be considered.

Information on atopy status can be obtained from a personal or family history, from skin or laboratory testing for (IgE-mediated) immediate type sensitization to common environmental allergens, and from physical findings that may be manifestations or sequelae of atopic diseases, associated conditions (e.g., keratosis pilaris), or physical features not related to manifest disease. The latter stigmata of atopy comprise especially dry skin, hyperlinearity of the palms and/or soles, infraorbital fold, white dermographism, facial pallor, orbital darkening, Hertoghe's sign, and low hairline. Depending on the criteria used, up to 50% of the population can be found to be atopic. Thus, the course and presentation of the skin disease itself are much more important to the diagnosis than the atopy status.

In view of the host of features characteristic of, but never specific for atopic eczema, various proposals of criteria have been made to establish a reliable diagnosis of the disease, e.g., the well-known criteria proposed by Hanifin and Rajka or the United Kingdom Working Party's diagnostic criteria for atopic dermatitis. They provide a helpful framework, particularly for nondermatologists, but their application cannot substitute for the proficiency of a trained dermatologist. There are also numerous systems to assess disease severity of atopic eczema, a widely accepted one being the Scoring Index of Atopic Dermatitis (SCORAD).

There is a plethora of differential diagnoses of atopic eczema. Particularly other eczematous diseases (seborrheic eczema, nummular eczema, allergic or irritant contact eczema), infectious diseases (scabies, dermatophytosis, candidiasis), immunologic disorders (dermatitis herpetiformis, pemphigus foliaceus, graft-ver-