Avoidance techniques in atopic eczema

We **recommend** to identify individual trigger factors in patients with AE, to avoid these in the future, with the aim of prolonging remission or clearance.

>75%  
(16/17)  
Expert Consensus

We **recommend** to avoid pollen, house dust mite and animal dander as much as possible to prevent exacerbation of AE in sensitized patients with a clear history of disease flares secondary to these triggers.

>75%  
(14/15)  
Expert Consensus

There is no need to restrict normal everyday physical activity in patients with AE.

>75%  
(17/19)  
Expert Consensus

We **recommend** avoiding irritant clothing (e.g. wool with coarse fibers) to prevent an exacerbation of AE in patients with sensitive skin.

>75%  
(16/17)  
Expert Consensus

We **suggest** that patients with AE learn strategies to cope with stress (e.g. educational programmes).

In selected cases, counselling or psychotherapy is **suggested**.

100%  
(16/16)  
Expert Consensus
We recommend the avoidance of tobacco smoke for the prevention of AE.

Pollen avoidance

Pollen-related flares can be observed in sensitized atopic patients. Exacerbation of AE may occur after either direct skin contact or inhalation of pollen allergens. Whether pollen avoidance leads to the prevention of flares in AE, has formally not been shown yet.

A reduced concentration of pollen indoors may help to prevent flares in patients highly sensitized to pollen. Keeping windows closed during high pollen peaks or the restrictions of outdoor activities in high pollen containing areas (e.g. lawn mowing) may be helpful. Frequently ventilated indoor spaces in rainy weather or at night/early morning, as well as the use of special pollen filters in air conditioners may also be advised. Skin contact with pollen-vectorized clothes or pets should be avoided. High-altitude climate may be recommended due to its lower pollen count compared to average living areas. These measures may however be difficult to maintain.

House dust mite avoidance

House dust mite (HDM) -related flares may occur in AE patients. Some house dust mite allergens identified by specific IgE or skin prick testing are enzymatically active compounds, which can destroy the cutaneous permeability barrier and may evoke the development of eczematous inflammation in sensitized atopic individuals.

The evidence on HDM avoidance techniques in the prevention of atopic flares is somewhat controversial. Measures of reducing exposure include mattress encasing, the use of adequate indoor ventilation (filter, well-aeration), and the avoidance of wall washing on high temperature. HDM, a common indoor allergen occurring in dust, may be reduced by cleaning regularly. Complete eradication by e.g. encasing is not possible.

Animal dander avoidance

When allergies to furry animals are evident, their avoidance is recommended. Particularly the exposure to cat allergens may be a risk factor for the development of inflammatory skin lesions as well as respiratory symptoms in sensitized patients with AE. There may be an exception for dogs due to a suggested general protective effect of dog-keeping in the development of AE.

Exercise/perspiration/physical activity

In AE patients heat and excessive sweating is one of the main factors reported to exacerbate itch. When excessive sweat is left on the skin it can lead to occlusion of the sweat pores and formation of keratin plugs which in turn may cause local inflammation and itch. Some of the components of sweat include histamin, antimicrobial peptides and proteases which can induce itch. Sweat can also facilitate the penetration of allergens through the defective atopic skin barrier leading to mast cell degranulation. As sweat is important for skin homeostasis it is not possible to avoid sweating completely. However, it should be washed off with consistent application of emollients as soon as possible to avoid inducing itch. The evidence concerning physical activity as a trigger for AE is
conflicting and incomplete.\textsuperscript{9} Although physical activity often leads to sweating, it is important for both physical and mental health, and AE patient should not be advised to avoid it.

**Clothing**

In patients with AE certain fabrics such as wool can cause a tingling sensation, skin irritation and itch. The evidence is not completely clear on which fabrics to recommend for use and which to avoid. Clothing-related exacerbation can be subjective.\textsuperscript{12} There is no evidence from high quality studies that certain fabrics improve the severity of AE.\textsuperscript{11, 13} In general, textiles with coarse fibres, such as certain wool garments and occlusive clothing leading to overheating should be avoided. Otherwise, the choices of clothing should be based on individual preferences. Most AE patients tolerate silk and cotton well, whereas contact with wool is frequently irritating.

**Psychological stress**

There is good evidence that AE is associated with depression, anxiety and reduced QoL.\textsuperscript{14, 15} It is difficult to investigate whether the psychological stress is a cause or consequence of the AE exacerbation, and in many case it is probably both. There is a positive correlation between maternal stress and offspring AE.\textsuperscript{16, 17} Although evidence from larger studies is lacking, patients report that stress induces itch and flaring of the disease.\textsuperscript{18, 19} (see chapter psychological intervention)

**Pollution**

In a systematic review of environmental epidemiologic studies about air pollution and AE acceptable evidence was found that, based on small-scale exposure measurements (so-called PM 2.5., i.e. particles with less than 2.5 µm diameter), especially truck traffic emissions increased AE prevalence. PM 2.5 are primarily comprised of organic carbon compounds, nitrates, and sulfates. For large-scale exposures to larger particles (PM10) or SO2 the review did not find an effect on AE prevalence.\textsuperscript{20, 21} Additional environmental risk factors for AE identified in single studies\textsuperscript{22} were carbon monoxide (CO) exposure during first trimester, CO exposure within past 12 months to CO level > 1 ppm above annual CO, high total volatile organic compounds (TVOC) in infant’s bedrooms at 6 months and AE at 36 months, and nitric oxide (NO2); the latter found as risk factor for AE in four different studies. So far the role of pollutants as trigger factor of pre-existing AE has not properly been described.

**Tobacco smoke**

The association of AE with active smoking was found to be significant in a metaanalysis (OR 1.87, 95% confidence interval 1.32-2.63). This association remained significant when looking at only children, only adults and by geographic region. Moreover, the effect of exposure to passive smoke on AE flares is small but also significant (OR 1.18, 95% confidence interval 1.01-1.38). Passive smoke was associated with the prevalence and severity of AE both in children and adults.\textsuperscript{23} The results of a recent registry study of 908 patients with AE suggest that the intensity of lesions and the Patient Global Assessment Score (PGA) were higher in smoking patients (n=352) than in non-smoking patients (n=556). However, physician-assessed disease severity (o-SCORAD and EASI scores) did not differ between smokers and non-smokers in this study.\textsuperscript{24}
References


