More Than Just the Top Layer: How Age, Environment, Treatments, and Genetics Affect Atopic Dermatitis

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Abstract:

Atopic dermatitis (AD), also known as eczema, is a common disease in children. However, as most children mature, they lose symptoms. AD can present itself at any time in life, with different severities, reactions, and location on the body. Its presence is due to an overactive immune system. In this project, multiple factors that affect the presence and management of eczema have been assessed. Specifically, age and how it can change treatments used, the effect of environmental factors, types of treatments and their uses, and the impact of genetics were investigated. By integrating findings across this research, people will be able to gain a more comprehensive understanding about the progression and management of eczema and the multiple individual factors that affect its presence. This foundational work will help support future efforts in prevention, education, and target personalized forms of therapeutic management for patients with eczema.

Introduction:

For millions worldwide, waking up to itchy, inflamed skin is a daily reality. According to the NIH, atopic dermatitis (AD), more commonly known as eczema, is the most common chronic inflammatory skin disease globally (2024). The body reacts to unharmful antigens,and causes an overactive immune response, leading to symptoms such as skin inflammation, itchiness, swelling, rashes, bumps, and increased sensitivity (Avena-Woods, 2017). Due to increased sensitivity, certain people have specific triggers which can make symptoms worse. AD affects children and adults globally, disrupting sleep, daily activities, and quality of life. Despite its prevalence, there is no single cause, and its course varies widely due to a complex interaction of factors that affect its severity and incidence.

One evident factor is age, seeing how as children mature they often begin to lose symptoms of AD. According to the NIH, around 30% of adults have AD, while only 2-10% of children have AD (2024). Age impacts the maturity of the skin barrier and immune system, which leads to differences in prevalence and treatments of AD.

Environmental factors can also affect the presence and severity of eczema. Factors like prolonged sun exposure, increased air pollution, humidity, temperature, and climate change can all affect the presence of AD symptoms, and ultimately how they are managed through treatments.

Certain inherited genes can also play a role in eczema incidence. Genetic mutations can cause a defect in the skin barrier, which can make it more prone to reactions, and ethnicity can also play a part in the presence of eczema.

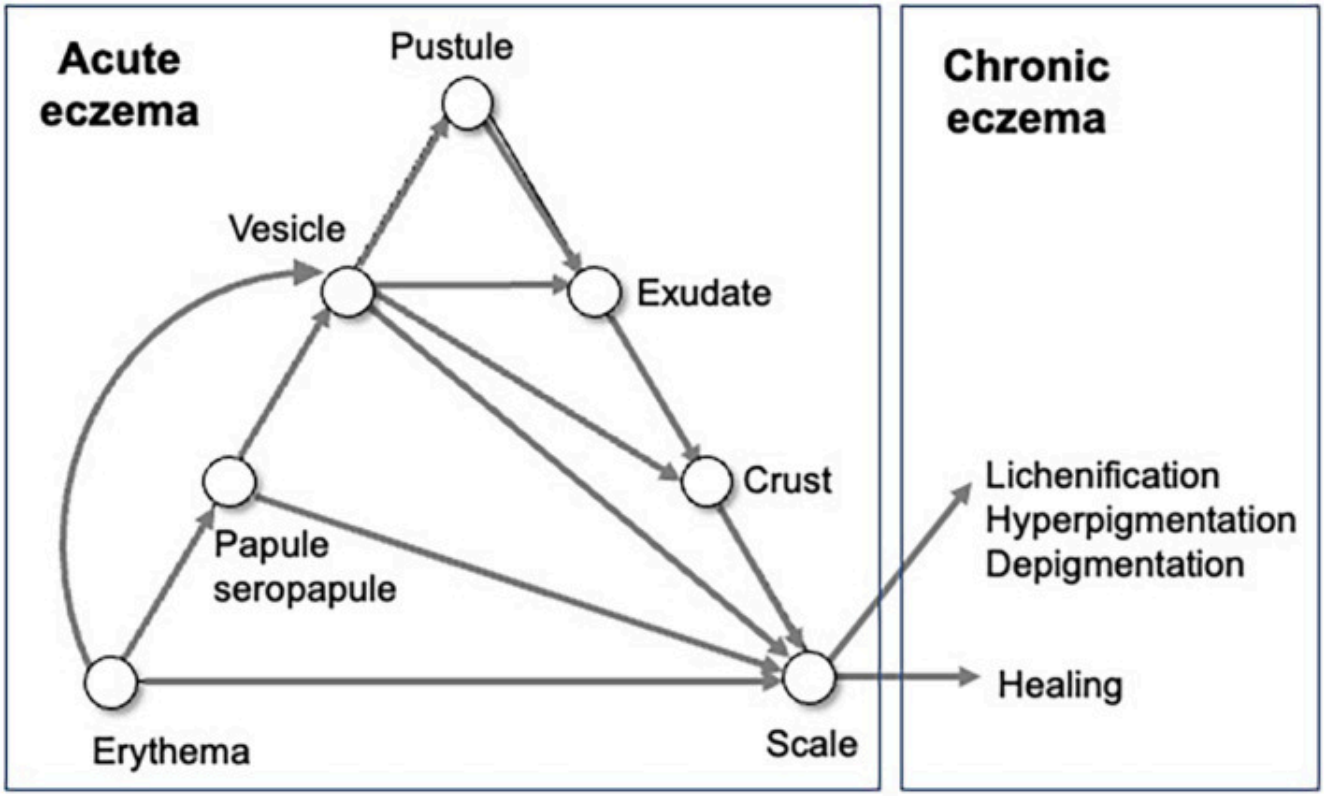
AD has multiple treatments, including ones for everyone. Some of these include simple moisturizers, daily broad-spectrum sunscreen (SPF 30+), gentle products, and emollients. There are also more specialized treatments that may need to be prescribed, such as steroids. The severity of eczema affects what type of treatment is used.

People struggle with AD every day and often have no clear starting point for controlling it. It is a multi-faceted disease influenced by factors people can and cannot control, and knowing what works in conjunction with those factors is essential for effective management. This review addresses these complexities through four specific aims. The first aim is to identify which

treatment ingredients are most effective in children compared to adults, and examine differences in AD incidence between age groups. The second is to evaluate how changes in environment, particularly humid versus arid climates, affect eczema's frequency and severity. This review also aims to investigate why topical corticosteroids are the most common treatment for all types of eczema and explain their mechanism of action, and assess how genetic factors, including skin barrier defects, impact eczema onset and severity. This leads me to introduce my research question: How do age, environmental factors, genetics, and treatments (such as topical corticosteroids, creams, and lotions) influence the presentation and management of atopic dermatitis?

Overview of Atopic Dermatitis

AD commonly involves the epidermis (topmost layer), the dermis, and/or the fat underneath the skin, often referred to as subcutaneous fat (Avena-Woods, 2017). Additionally, AD can be identified through three morphological features: multiple-pinpoint condition, polymorphism, and itch (Tokura, 2024). Multiple-pinpoint condition refers to all the small areas of focus, pinpoints, where papules or vesicles might occur (Tokura, 2024; Sohn, 2011). Polymorphism in AD means that it can display itself in different ways, such as erythema, papule, vesicles, scale, and crust, which often makes it difficult to diagnose compared to other skin diseases (Sohn, 2011). AD is also often more itchy compared to other inflammatory skin diseases (Tokura, 2024).

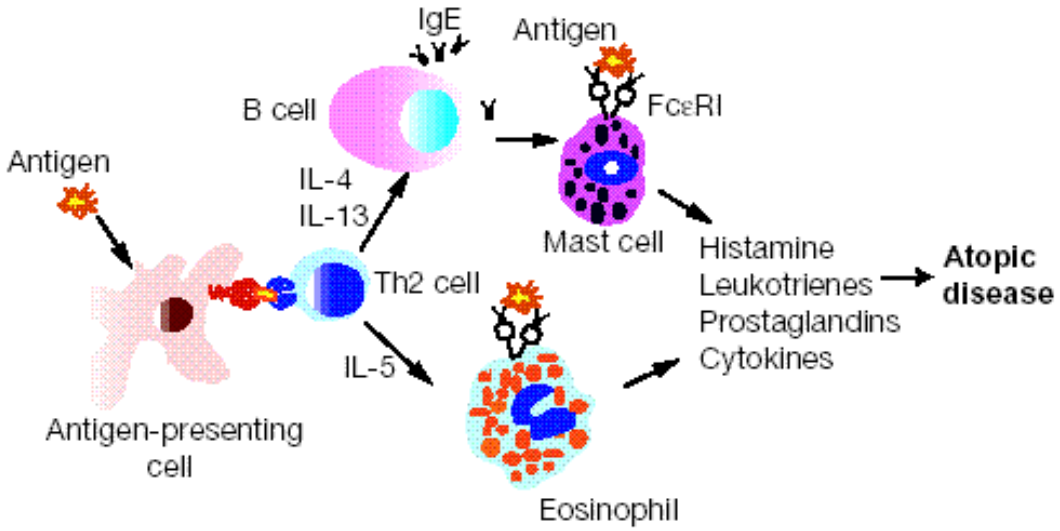


"Figure 1," Eczema Triangle (Tokura, 2024)

Figure 1 above shows the Eczema Triangle, which shows how certain changes seen on the epidermis can lead to others. Some of these changes are caused by inflammation in the dermis, while others are from inflammation in the epidermis. Each of these changes may vary from pinpoint to pinpoint because they may not all appear at the same moment, but may follow the same order of changes. This causes different pinpoints to have different epidermal changes, leading to polymorphism in the appearance of eczema (Tokura, 2024). In all cases of acute eczema, it begins with erythema due to histamines and other chemical mediators causing blood vessels in the dermis to dilate. After going through other epidermal changes, acute eczema always ends with scaling, where it then heals or leads to lichenification, where the skin becomes thicker due to excessive itching, hyperpigmentation, or depigmentation. Chronic eczema lasts for months or years, while acute eczema is similar to a flare-up (Sohn, 2011). AD is a type of eczema that encompasses both acute and chronic eczema (Tokura, 2024).

Pathophysiology and Immune Response

The cause of these flare-ups is due to the immune system overreacting to antigens on the skin. The immune system flags the antigen as foreign, but does not establish that it may not be harmful and overcompensates. As illustrated in Figure 2, the antigen is captured by a type of immune cell known as the antigen-presenting cell. It then activates the Th2 cell, which is a type of helper cell that coordinates the overall immune response, sending a chemical signal to both a B cell that creates antibodies and eosinophil, which is a type of white blood cell. When the B cell receives the chemical signal in the form of an interleukin (IL), it then begins to release an antibody called an Immunoglobulin E (IgE) which has a special Y shape (Darsow, 2008). The IgE then binds one side to a mast cell containing granules, and the other side is bound to the antigen (Darsow, 2008). The IgE binding to the mast cell causes the cell membrane to break and the granules release histamine and other chemical mediators which fight the antigen. Histamine and chemical mediators are what cause symptoms such as itching, so an increase in them causes more of a reaction. In eosinophils, a type of allergy cell, the same process occurs leading it to dispense chemical mediators.



"Figure 2," IgE cascade when an antigen is present (Corry, 1999)

Genetic Factors and Skin Barrier Function

A weakened skin barrier has been linked to be a reason why some people are more susceptible to AD (Avena-Woods, 2017). Inheriting filaggrin (*FLG),* which is a gene/protein important for skin barrier function, with a loss-of-function mutation that makes the gene/protein works less well or not at all can lead to gene-environment interaction in allergic sensitisation (Stefanovic, 2021; Avena-Woods, 2017). Allergic sensitisation (the immune system becoming primed to overreact to harmless substances) lowers the threshold for a type-2 (Th2) allergic response to common triggers. This consequently leads to extra chemical mediators in the body even when they are not needed. Further exposure of this allergen to the body leads to inflammation, which can eventually cause chronic inflammation (Stefavonic, 2021). Additionally, immune dysregulation, including the activation of the IgE cascade in Figure 2, can lead to a weakened skin barrier (Kim, 2019; Darsow, 2008). The epidermal lipid layer, a part of the skin barrier, aids in water retention and defects can lead to excessive drying which can lead to more inflammation and irritation (Rawlings, 2004). Defects in certain proteins in the skin barrier can also affect penetration into the skin, causing further drying and making it more difficult to manage (Kim, 2019; Rawlings, 2004). However, dryness and a weakened skin barrier can also occur later on to a person that has AD. Being born with that innate mutation can make a person more sensitive or likely to develop AD. While FLG mutations are linked to increased AD risk, the extent to which environmental factors can trigger disease onset in mutation carriers remains unclear.

Another mutation that can affect the presentation of AD is a gain-of-function mutation (a change that makes a gene overactive) in IL-4R (Kim, 2019). IL-4R is a receptor found in B cells that signals the body to create more mast cells. When Il-4R has a gain-of-function mutation, the receptor is on for the lifetime of the cell (Kim, 2019). This makes the body have extra chemical mediators present, so when there is an antigen present, extra chemical mediators like histamine are released causing flare-ups and a greater reaction. Overall, while FLG and IL-4R variants elevate risk, they act as modifiers, and not destiny; how specific exposures alter onset and severity in carriers remains uncertain and warrants further study (Stefanovic, 2021; Kim, 2019).

Demographic, Environmental, and Age-related Factors

Ethnicity can also play a role in an individual's susceptibility to getting AD. In fact, AD prevalence is reported to be higher among Black and Asian populations than White populations; proposed explanations include differences in barrier biology, environmental exposures, and access to care (Kaufman, 2018). Due to different ancestors and environments, the skin barrier has genetic differences, adapting to environmental factors in each respective area. Black and Asian populations have adapted to more hot environments as compared to White populations. They also have more melanin, which leads to less vitamin D production, and less protection from environmental factors. However, environmental factors in these areas can also play a role, such as a more arid climate which can exacerbate symptoms. It is also possible that access to care may be less in Black and Asian populations as compared to White populations, so AD is treated later, or mislabeled (Kaufman, 2018).

Environmental factors can also trigger flare-ups. Some of these factors include low humidity, UV exposure, air pollution, and water hardness (Stefanovic, 2021; Avena-Woods, 2017). Although low UV exposure is associated with higher AD prevalence, the optimal level for skin health without increasing cancer risk has not been established. AD is more common in areas with low humidity, low temperature, and low levels of UV exposure (Stefanovic, 2021).

These areas are known as places with arid climates, and arid climates have been linked with further inflammation (Avena-Woods, 2017). Low UV exposure also plays a role because UV exposure is necessary for vitamin D production. Vitamin D is necessary for the skin barrier as it regulates the production of proteins on the epidermis (Bikle, 2013). It also helps with management of AD by reducing inflammation with less mast cells and IgE being produced, and aiding in the immune system's response (Stefanovic, 2021; Bikle, 2013). Research also suggests that vitamin D has increased gut microbiome diversity (Stefanovic, 2021).

Being exposed to air pollution at a young age can prime the cutaneous immune system to be more sensitive to irritants in the air, increasing the chance for AD (Stefanovic, 2021). Certain pollutants in the air, such as volatile organic compounds, have also been linked with increased water loss in the skin barrier (Stefanovic, 2021). This can lead to hypersensitivity and cause flare-ups, while further harming the skin barrier (Stefanovic, 2021).

Differences in the hardness of water can play a role as well. For example, hard water has been linked with higher changes of AD. A study conducted by Enquiring About Tolerance (EAT) demonstrated that *FLG* mutations are increased three-fold in infants that are exposed to hard water, even if they did not have AD at that moment(Stefanovic, 2021). Current studies point toward these mutations being caused by barrier disruption, which allows immune dysregulation and indirectly promotes mutational processes, but there is still not enough research as to why hard water causes skin barrier disruption directly (Stefanovic, 2021).

Age is also a factor, considering how much more common AD is in children as compared to adults. Most children begin to lose symptoms of AD as they mature, while others do not (Fisal, 2025). Symptoms and types of eczema also are impacted with maturity. Most children experience acute eczema, while adults also experience the symptoms of chronic eczema on top of acute eczema. In children, acute eczema can progress to healing, but for some it might progress to lichenification, hyperpigmentation, and/or depigmentation as seen in Figure 1. This also explains why some adults after not seeing symptoms since childhood can experience one flare-up and then have it lead to chronic eczema (Fisal, 2025).

Due to the fact that children are still not fully mature and are still developing their immune system as well as their skin, treating AD in younger children can become more difficult as compared to adults, especially in more severe cases. Children can not use powerful steroids that directly suppress the immune system so the steroids do not impair the immune system. Even if topical steroids are deemed necessary, they are used in low potency and in shorter periods of time as compared to adults (Stacey, 2021). Adults are more likely to use topical steroids as a form of treatment because they have less risk factors (Stacey, 2021).

Treatment Approaches

Treatments can come in many different forms for AD, whether they are a simple over-the-counter treatment or a steroid that needs to be prescribed by a doctor. The most simple and commonly found treatments are moisturizers. Moisturizing the skin is very important for someone that has AD because it helps with dryness and flakiness that can cause irritation and inflammation (Chong, 2015). Some moisturizers also contain ceramides, which are an essential part of the skin barrier (Chong, 2015). Additional ceramides can aid with strengthening an already weakened skin barrier. Wearing sunscreen is also crucial as it prevents sunburns, and any additional dryness, redness, and irritation. Although UV exposure is important because it increases vitamin D production, it is equally as important to protect the skin from excessive sun

exposure, which can lead to unwanted side effects and can also lead to other conditions like skin cancer.

Other forms of managing AD include body washes aimed for people with AD. These products are fit for more sensitive skin by avoiding common allergens and using gentle ingredients. These minimize and prevent reactions that might be more common for people who are sensitive to the products they use currently. They also contain ingredients like ceramides which help moisturize and restore the skin barrier, and not strip the skin of its natural oils.

Despite these products, there is also the use of steroids in more severe cases. The most common ones are topical corticosteroids. They are often used to help with inflammation (Stacey, 2021). Corticosteroids turn off certain inflammatory pathways, while still being relatively safe. Inflammation is one of the most common and irritating symptoms of AD and can be chronic. Being able to control or at least help with this symptom is important for many people because it helps with quality of life. There are multiple potencies of corticosteroids from high to low potency (Stacey, 2021). As seen in figure 3, most cases of atopic dermatitis either need high or medium potency steroids (Stacey, 2021). More resistant forms of AD might need high potency corticosteroids, while less resistant or areas of more sensitive skin might use medium potency or in some cases low potency corticosteroids.

Skin Conditions Responsive to Topical Corticosteroid Treatment		
High-potency steroids (groups I and II)	**Medium-potency steroids (groups III, IV, and V)**	**Low-potency steroids (groups VI and VII)**
Alopecia areata	Anal inflammation (severe)	Dermatitis (diaper)
Atopic dermatitis (resistant)	Asteatopic eczema	Dermatitis (eyelids)
Bullous pemphigoid	Atopic Dermatitis	Dermatitis (face)
Discoid lupus	Dermatitis (severe)	Intertrigo
Dyshidrotic eczema	Infantile acropustulosis	Perianal inflammation
Hyperkeratotic eczema	Intertrigo (severe, short term)	Phimosis
Labial adhesion	Lichen sclerosus (vulva)	
Lichen planus	Nummular eczema	
Lichen sclerosus (skin)	Scabies (after scabicide)	
Lichen simplex chronicus	Seborrheic dermatitis	
Melasma	Stasis dermatitis	
Nummular eczema		
Poison ivy (severe)		
Psoriasis		
Vitiligo		

Figure 3, Skin conditions and Potency of Steroid (Stacey, 2021)

Although topical corticosteroids are so frequently used, they also have cutaneous (skin-related) and systemic adverse effects (Stacey, 2021). These include easy bruising, contact dermatitis, hyperpigmentation, hypertension, and skin thinning (Stacey, 2021). These are more common with frequent use, high potency, and thin skin (Stacey, 2021). Sensitive areas or areas with thin skin, including children who have undeveloped skin so their skin is thin, often are treated with low potency corticosteroids (Stacey, 2021). The length of use is determined by the potency, with higher potency ones being used for around 3 weeks, and low potency corticosteroids used for no more than 12 weeks at a time (Stacey, 2021). Topical corticosteroids can also come in multiple forms including ointments, creams, lotions, gels, and foams, which differ from use depending on patient preference and whether that area of the skin is hair-bearing (Stacey, 2021). Ointments usually have less ingredients, so they are better for people who have sensitive skin (Stacey, 2021). However, they leave a residue on the skin, so creams, lotions, and gels might be better for people who do not like a residue on their skin (Stacey, 2021). Foam works best for areas that are hair-bearing because it can blend in easily (Stacey, 2021). Despite adverse effects, topical corticosteroids are still considered first-line and come in different formulations.

There are other types of treatments, however. One of them is calcineurin inhibitors. Topical calcineurin inhibitors (tacrolimus, pimecrolimus) inhibit calcineurin, blocking T-cell activation/IL-2 transcription; they are useful on sensitive areas or for maintenance. (Safarini, 2023). Another is phototherapy, in which controlled UV light exposure aids in reducing inflammation and slowing skin cell growth.

Methods:

This paper is based on other pieces of research from credible sources and papers. We conducted a narrative literature review using PubMed and Google Scholar. Searches covered 2013-2025 as well as earlier landmark studies and were limited to English. We then cited the sources into a Google Doc containing all the sources and wrote a brief literature review for each source encompassing the major ideas in it and how they could be tied to the four aims mentioned in the introduction.

Results:

Age

AD prevalence is higher in children than adults (~30% vs 2-10%), with partial remission in adolescence reported in multiple cohorts (Fisal, 2025). Additionally, pediatric treatment studies favored lower-potency, shorter-duration corticosteroids to minimize adverse effects in children (Stacey, 2021). Studies also observed that adults can also experience relapses of AD after even one flare-up in adulthood (Fisal, 2025).

Environment

Low humidity, low UV exposure, air pollution, and hard water are associated with higher AD risk in children; effects may be stronger in FLG carriers (Stefanovic, 2021). Environments with low humidity and cold temperatures were associated with higher flare frequency (Stefanovic, 2021). Low UV exposure, subsequently leading to low Vitamin D levels, have also been reported to worsen AD outcomes (Bikle, 2013; Stefanovic, 2021). Air pollution ($PM2.5/NO_2$) exposure correlated with increased AD incidence (Stefanovic, 2021). Water hardness was also associated with increased AD risk in children (Stefanovic, 2021).

Treatments

Types of treatments include moisturizers, body washes, sunscreen, calcineurin inhibitors, phototherapy, and the most common type for serious cases, topical corticosteroids (Stacey, 2021). Emollients have been reported to help people with AD reduce inflammation and itchiness, resulting in reduced flare frequency (Araviiskaia, 2022). Topical corticosteroids improved the Patient Oriented Eczema Measure (POEM) score after moderate flare-ups, after 1 week up to 24 weeks in a sample of 32 patients (Halewijn, 2024). Emollients showed moderate to very good improvement in ~61% of participants in [Wilhelm 1998] (van Zuuren, 2017). Calcineurin inhibitors showed efficacy for sensitive areas (face/flexures) with fewer steroid-related skin changes (Safarini, 2023).

Genetics

Genetic mutations resulting in increased IgE antibodies as well as skin barrier defects have been reported to increase the risk of development of AD (Stefanovic, 2021; Kim, 2019). FLG (filagrin) loss-of-function mutations were associated with increased AD risk (Stefanovic, 2021). Variation in IL-4R/Th2 pathway genes correlated with elevated IgE and more severe disease as well (Kim, 2019). Ethnic differences were also reported with higher rates in Black and Asian populations compared to White populations (Kaufman, 2018).

Analysis/Discussion:

This review suggests that AD is shaped by interacting age, environmental, treatment, and genetic factors. Consistently, children show higher prevalence; low humidity, pollution, and low UV exposure exacerbate symptoms; topical corticosteroids and emollients remain first-line; and FLG/Th2-pathway variants increase risk.

AD is more prevalent in younger people as compared to adults, which can also lead to differences in treatment. These differences might happen for a couple of reasons, such as a mature skin barrier and immune system that are more well equipped to fight and have more effective responses. These heightened reactions lead to the need for more potent steroids as compared to children, despite the fact that they have more side effects. Over time, the skin barrier and immunity become weaker due to age, which can also affect the prevalence of AD as well as treatment options.

Environmental factors, including climate, humidity, and pollution can lead to barrier dysfunction and inflammation due to an introduction of antigens. These antigens are what cause a reaction, and they can be increased during certain seasons depending on what factors a person with AD is sensitive to. Seasonal patterns in these factors can also lead to seasonal based flare ups for someone with AD.

Management of AD has become more accessible, now coming in multiple different treatments and approaches. To directly stop a current reaction, topical corticosteroids are used locally to aid with inflammation and the immune system's response. To help prevent future flare ups, emollients are important as well as to soothe and moisturize the skin. Alternative options might be effective for people who are sensitive to other options, or if other options are ineffective. They also might use different treatments based on the side effects of each. Personal preference plays a big role in what type of treatment is best for an individual.

Although genetic mutations are a significant factor for AD, they are not a deciding factor on the presence of AD. They only increase the risk for the presence of AD, but do not actually

cause it. The environment is what causes these flare ups, and these factors are only accentuated by these genetic changes.

All of these factors might be different in everyone's lives, but there are still some general practical implications that can be drawn from this research. When using treatments, potency and duration should be considered in relation to age. Emollients should be used regularly to help soothe the skin as well as help with future flare ups. During winters, due to more dryness, humidifiers can be employed in homes and getting enough sun exposure safely. Pollution can also be avoided when possible.

There were also some limitations in this research. As a narrative, English-only review without a formal risk-of-bias tool, findings may reflect selection and heterogeneity. This could have also affected findings on other countries, such as in Asia.

Finding a way to prevent AD would be extremely helpful, especially to those who struggle with it in their day to day lives. There are multiple ways which unwanted symptoms can be diminished, but not fully prevented or stopped.

Conclusion:

This paper examined how age, environmental factors, genetic predisposition, and treatments choices influence the presentation and management of atopic dermatitis. AD is more common in childhood, with partial improvement for many during adolescence; some have persistent or recurrent disease into adulthood. Management differs by age and site: children typically use lower-potency, shorter-duration steroids; adults often tolerate medium-potency options on many body sites. Environmentally, lower humidity and temperatures worsen flares, as well as lower ambient UV exposure correlates with higher AD prevalence (with safe sun practices still essential). Treatment of AD comes in multiple forms, but emollients should be used regularly, topical corticosteroids in more serious forms, and non-steroid options for sensitive areas. Genetic mutations such as FLG mutations and immune-pathway variants may increase the risk and severity of AD, but they are not deciding factors.

Understanding these factors at a personal level is important to patients and caregivers as it aids in finding personalized approaches to help manage systems and enhance quality of life. Some practical implications that can be applied for most people with AD include being aware of how age can impact potency and duration of steroid use. Genetics do play a role as risk factors, especially in the skin barrier so barrier protection should be prioritized. Using emollients daily helps in protecting the skin barrier, and they can also aid during dry seasons. Using humidifiers also aids in dry weather, and UV protection is important for daily life.

Because this was a narrative review, results may be influenced by study heterogeneity and selection of sources. This research can be expanded upon, with future work clarifying gene-environment interactions and comparing real-world effectiveness of steroid and non-steroid regimens. Priorities include clarifying gene-environment interactions, comparative effectiveness of steroid vs non-steroidal regimens, long-term safety data, and strategies to reduce inequities in access and patient education. Education on Atopic Dermatitis should be more accessible and understandable to a common person who may not have much prior knowledge on the topic. Integrating barrier care, targeted anti-inflammatories, and environmental strategies, tailored to age and genetic risk, can meaningfully reduce flares and improve daily life for people with AD. Individuals should be able to take away what may apply to them and apply that to their own lives to enhance their daily life.

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