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Eczema Across the Lifespan: A Comprehensive Review of Current Research and Future Directions

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Abstract :

Eczema, or atopic dermatitis (AD), is a chronic inflammatory skin condition affecting approximately 200 million people worldwide, with its incidence rising significantly in recent decades, particularly in high-income countries. It typically begins in the first year of life but can manifest at any age. Eczema often follows a relapsing–remitting course, requiring ongoing treatment to manage symptoms. While many children with eczema experience improvement or resolution by late childhood, a substantial proportion continue to be affected into adulthood, with flare-ups occurring even after long periods of remission. The aim of this review is to provide a comprehensive examination of eczema (atopic dermatitis) across the lifespan, with a particular focus on its epidemiology, pathophysiology, and impact on different age groups, including children, adults, and the growing population of older adults. This review seeks to explore the current understanding of the disease's progression, the diverse life-course trajectories of eczema, and the implications of these trajectories for treatment and management. Additionally, the review aims to highlight the gaps in existing research and identify future directions for improving the prevention, management, and quality of life for individuals affected by eczema at various stages of life.

Key words : Eczema, Atopic Dermatitis, older adults .

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Introduction :

Eczema, also known as atopic dermatitis, is a chronic inflammatory skin condition that affects approximately 200 million people worldwide (1, 2). The incidence of eczema has risen significantly over the past decades, particularly in high-income countries (3). It most commonly develops in the first year of life, although onset can occur at any age (4, 5). Eczema usually follows a chronic

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relapsing–remitting course and maintaining disease control may require ongoing treatment (6).

While many eczema-affected children will experience resolution or improvement by late childhood (4), a substantial proportion of people will have ongoing eczema into adulthood, and flare-ups can occur even after long periods of remission (5, 7). Itch, discomfort, and visible skin lesions result in disturbed sleep, social embarrassment, and affect the quality of life of those affected and their families (8). When moderate to severe, the psychological impact on children and adults is often profound (9-11).

From 2020 to 2024, the global population of older adults (65+) has shown a consistent increase, starting from 727 million in 2020 and reaching an estimated 801 million in 2024. This trend highlights the growing aging population worldwide (United Nations, 2022). In the Middle East, a similar upward trajectory is observed, with the number of older adults rising from 13.5 million in 2020 to an estimated 15.5 million in 2024 (World Bank, 2023). Specifically, in Qatar, the older adult population has grown from 29,000 in 2020 to an estimated 33,000 in 2024 (Qatar Planning and Statistics Authority, 2023). These figures underscore the demographic shifts occurring both globally and regionally, indicating significant increases in the elderly population.(26)

The prevalence of eczema among older adults varies but generally falls within a specific range based on different studies. the prevalence of eczema among older adults aged 75-99 ranges from 7.0% to 9.3% (The epidemiology of atopic dermatitis in older adults, 2021), the prevalence of atopic dermatitis (a common type of eczema) in Qatar varies between 5% and 30%, depending on the age group. While specific figures for older adults are not provided, the prevalence among adults is reported to be around 5% (Hamad Medical Corporation, 2024).while affecting an estimated 15-20% of children worldwide (Allergy & Asthma Network, 2023). According to (Practice Update, 2023), the current prevalence of eczema in children is about 6.0%. (27)

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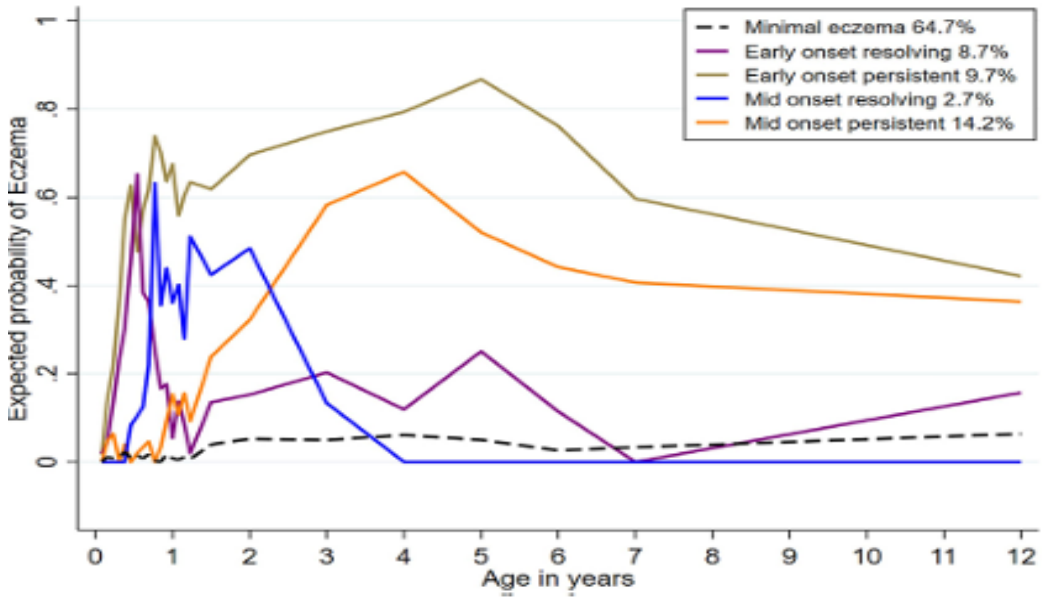

Figure (1). Onset of Eczema by Age in Years.

This figure illustrates the longitudinal trajectories of different eczema subclasses from birth to age 12 years, identified through a secondary analysis of an Australian randomized trial involving infants (11) and a birth cohort study (12) with 620 participants. Adapted from Lopez DJ, Lodge CJ, Bui DS, et al. (2022). Establishing subclasses of childhood eczema, their risk factors, and prognosis. *Clinical & Experimental Allergy*, 52, 1079-1090.

Traditionally, the early onset of atopic dermatitis (AD), compared to other allergic diseases such as allergic rhinitis and asthma, led to the hypothesis that AD progresses to allergic rhinitis and then to asthma in a linear progression sequence (9, 10). Although the average onset of disease among populations may generally develop this way, it is now evident that many individuals do not follow this pattern. Longitudinal clustering analyses of patients with AD show diverse life-course trajectories, including AD occurring in isolation, AD with rapid resolution, persistent AD, and various combinations involving AD with rhinitis, asthma, or food allergy (Figure 1). These data suggest that the pathogenesis of allergies may not always conform to a traditional sequential progression model (13-16). Thus, whether preventing AD definitively prevents the development of other allergic diseases remains a hypothesis with strong biological plausibility but, as of yet, insufficient

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supporting clinical outcome data. This review, therefore, focuses on the prevention of AD rather than the prevention of the progression from AD to other allergic diseases.

Theories of Pathogenesis of Atopic Dermatitis (AD):

The pathophysiology of AD is complex and multifactorial, with the initial events leading to the onset of the disease remaining uncertain. Efforts to prevent AD focus on enhancing the skin barrier, addressing immune dysregulation, and controlling allergen exposure. This section discusses the relevant mechanistic data and theories associated with these strategies.(23)

The Role of Exposome Components during Prenatal and Perinatal Period

1-Perfluoroalkyl and Polyfluoroalkyl Substances (PFASs)

PFASs, often referred to as "forever chemicals," are a large group of compounds used in a variety of products for their non-stick or stain-resistant properties. These substances are commonly found in industrial products such as lubricants, surfactants, and fire-fighting foams, as well as everyday items including non-stick cookware, greaseproof paper, food packaging, carpets, furniture, waxes, paints, clothing, and personal care products like shampoo, eye makeup, nail varnish, and dental floss. Prenatal exposure to PFAS compounds, such as perfluoro octane sulfonate (PFOS), perfluorooctanoic acid (PFOA), perfluoro hexane sulfonate (PFHxS), perfluoro nonanoic acid (PFNA), and perfluoro decanoic acid (PFDA), has been detected in over 90% of pregnant women in the US, Europe, and Asia and is linked to an increased incidence of childhood atopic dermatitis (AD) in girls during the first two years of life [11]. Additionally, prenatal exposure to polychlorinated biphenyls (PCBs) has been associated with a heightened risk of asthma and eczema in offspring [12]. Prenatal PFAS exposure occurs via placental transfer and continues postnatally through breastfeeding, with connections to a higher risk of early-onset AD in children under five years of age [13,14]. Research has shown a correlation between prenatal PFOA and PFOS exposures and elevated IgE levels in cord blood, particularly in boys, leading to the hypothesis that PFAS may enhance allergen hypersensitivity [15]. Furthermore, in utero exposure to PFOA has been linked to an increased risk of developing AD as early as age two, especially in children with GSTT1-null or GSTM1-null genotypes that impair glutathione S-transferase (GST) activity, crucial for chemical detoxification.

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Children with GSTM1-null and GSTP1 Ile/Ile genotypes are also at higher risk for AD when exposed to prenatal smoke [16]. Finally, in a domestic setting, infants and young children face an increased risk of PFAS exposure due to their exploratory and hand-to-mouth behaviors.

2-Pollution

Air pollution, which involves the contamination of the indoor or outdoor environment by various chemical, physical, or biological agents that alter the natural characteristics of the atmosphere, can trigger the onset of atopic dermatitis (AD) in childhood. (22)Common sources of pollutants include domestic combustion appliances, motor vehicles, industrial installations, and forest fires. Major pollutants of public health concern are categorized into air pollutants (such as sulfur dioxide, nitrogen oxide, carbon monoxide, ozone, and volatile organic compounds), persistent organic pollutants (like dioxins), heavy metals (including cadmium, lead, and mercury), and particulate matter (PM). Exposure to lead during late pregnancy increases the risk of AD in boys at 6 months of age. Similarly, prenatal exposure to inorganic arsenic and combined exposure to inorganic arsenic and cadmium have been associated with a higher risk of AD in young children [17]. Mono-benzyl phthalate (MBzP) has been linked to an increased risk of developing eczema in early childhood [18]. Additionally, prenatal exposure to a combination of bisphenol A (BPA) and phthalates may be associated with AD in 6-month-old infants [19].

The US Environmental Protection Agency (EPA) classifies particulate matter (PM) according to particle size: PM0.1 (ultrafine particles, ≤0.1 μm), PM2.5 (fine particles, ≤2.5 μm), and PM10 (coarse particles, ≤10 μm) (25). PM can cause skin barrier dysfunction and generate reactive oxygen species, leading to oxidative stress, potential epigenetic changes, and skin inflammation through both direct and indirect mechanisms (28-29). Notably, there is an observed association between maternal exposure to traffic-related pollution and the prevalence of AD in offspring.(26)

3. Exposome in the Development of AD during Childhood and Teenage Life

3.1. Climate, UV Radiation, and Vitamin D

Atopic dermatitis (AD) often worsens seasonally and in response to changes in climate, including factors such as UV exposure, humidity, temperature, precipitation, and indoor heating. While a combination of high UV exposure and temperature seems to have protective effects against AD, high humidity and

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precipitation have been linked to increased risk. In early childhood, direct UV radiation exposure is beneficial in reducing the risk of eczema, whereas living in high latitudes, with reduced UV exposure, is associated with an increased risk [14,15]. UV radiation also stimulates the production of vitamin D, and a deficiency in vitamin D has been linked to a higher incidence and severity of AD symptoms. Supplementing with vitamin D3 has shown significant improvements in clinical symptoms, particularly in patients with winter-related AD [23]. Additionally, there is an inverse relationship between AD severity and serum vitamin D3 levels, with low maternal fish consumption and reduced vitamin D3 intake during pregnancy increasing the risk of AD in offspring (16-17).

Hispanic and black children are more likely to experience persistent AD compared to white children, with black children also being at higher risk of developing AD initially. In a large study of children living in urban areas in the US, black children were found to have the highest prevalence and severity of AD among the examined ethnic groups and were the least likely to achieve full disease control. In contrast, AD onset tends to occur later in life for Hispanic children [18]. Black children are also more exposed to known risk factors, including social factors such as living in older or rental homes, frequent relocation, tobacco exposure, lower household income, and lower parental educational levels—factors that also impact Hispanic children. Individuals with darker skin types generally have lower levels of 25(OH)-vitamin D, and low maternal levels of 25(OH)D during pregnancy increase the risk of AD in their offspring. Compared to non-Hispanic whites, certain non-white racial subgroups experience higher rates of AD incidence and persistence. (34)A longitudinal cohort study of black children with AD aged 0 to 2 years found that, despite lower vitamin D levels in black participants, the allergic sensitization load was linked to FLG expression in non-lesional skin in non-black children, but this association was not observed in black children with low vitamin D levels [20,21]. Further research is needed to explore the environmental, socioeconomic, and genetic factors contributing to these observed differences.(34-35)

Impaired Skin Barrier:

An impaired skin barrier, a hallmark of established AD, may also play a role in the etiology of AD. Skin barrier function is primarily determined by corneocytes and the associated stratum corneum intercellular lipid matrix. The cytoskeleton of corneocytes, formed by keratin-filaggrin bundles, provides mechanical resistance to environmental stressors (18). The intercellular lipid matrix, consisting of equal

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molars of ceramides, free fatty acids, and cholesterol, prevents water loss (evaporation/dehydration) and the penetration of allergens and irritants into the skin (19). Consequently, genetic defects in the epidermal barrier, such as the loss of function of the gene encoding filaggrin, are associated with the development of AD (21, 22).

Prospective birth cohort studies have observed that impaired skin barrier function, indicated by higher levels of trans epidermal water loss, precedes overt clinical signs and symptoms of AD (23, 24). A small number of studies have also noted that abnormal early life lipid profiles can precede the development of AD (25-27). Genetic, mechanical (e.g., scratching), chemical, allergen, or irritant-induced epidermal barrier disruptions may trigger keratinocytes in the deeper layer of the skin to release interleukin-33 (IL-33), interleukin-25 (IL-25), and thymic stromal lymphopoietin, among other signals, leading to subsequent inflammation and sensitization to allergens.(37-38)

Table (1): Effectiveness of Various Interventions in Preventing Atopic Dermatitis in Early Life: A Comprehensive Review

Intervention	Effect Size (Risk Ratio [95% CI])	Participants, n (Studies)	Population Studied	Follow-up Age	Certainty (Quality) of Evidence (GRADE) as Reported by Authors
Skin intervention (emollients/moisturizer) among infants	1.03 (0.81-1.31)	3,075 (7 RCTs)	High-risk infants	6-36 months	Moderate (inconsistency)
Probiotics in mothers only	0.69 (0.38-1.26)	2,159 (7 RCTs)	Mixed population	1-6 y	GRADE assessment not done by study authors
Probiotics in infants only	0.85 (0.62-1.17)	1,884 (11 RCTs)	Mixed population	1 months to 9 y	GRADE assessment not done by study authors
Probiotics in infants and mothers	0.65 (0.49-0.86)	4,739 (12 RCTs)	Mixed population	18 months to 11 y	GRADE assessment not done by study authors
Prebiotics in infants	0.68 (0.40-1.15)	2,030 (6 RCTs)	Mixed population	3-24 months	Low (bias and inconsistency)
Synbiotics in infants	0.44 (0.11-1.83)	1,320 (2 RCTs)	High-risk infants	6 months	GRADE assessment not done by study authors
Vitamin D supplementation for pregnant women	0.85 (0.67-1.08)	2,074 (4 RCTs)	Mixed population	6-36 months	Moderate (imprecision)
Vitamin D supplementation for infants	0.84 (0.64-1.11)	942 (2 RCTs)	Mixed population	12-30 months	GRADE assessment not done by study authors
Partially hydrolyzed formula	OR = 0.84 (0.67-1.07)	5,372 (12 RCTs)	High-risk infants	0-14 y	Moderate (risk of bias)

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Intervention	Effect Size (Risk Ratio [95% CI])	Participants, n (Studies)	Population Studied	Follow-up Age	Certainty (Quality) of Evidence (GRADE) as Reported by Authors
eHF Casein	OR = 0.55 (0.28-1.09)	3,374 (7 RCTs)	High-risk infants	0-14 y	Very low (risk of bias, inconsistency, imprecision)
eHF Whey	OR = 1.12 (0.88-1.42)	3,374 (7 RCTs)	High-risk infants	0-14 y	Very low (risk of bias, inconsistency, imprecision)
Egg introduction at 4-10 mo	0.87 (0.68-1.12)	1,368 (2 RCTs)	Mixed population	12 months	GRADE assessment not done by study authors
Cow's milk introduction <4 y	<4 y: 1.14 (0.87-1.49)	6,798 (12 RCTs, 5 qRCTs)	Mixed population	5 y	Low (imprecision and indirectness)
Cow's milk introduction 5-14 y	5-14 y: 1.05 (0.9-1.23)	6,798 (12 RCTs, 5 qRCTs)	Mixed population	5 y	Low (imprecision and indirectness)
Prenatal omega-3 polyunsaturated fatty acid supplementation	RR = 1.09 (0.82-1.46)	1,926 (6 RCTs)	High-risk	6 months to 6 y	GRADE assessment not done by study authors
Prenatal blackcurrant seed oil supplementation	RR = 0.70 (0.51-0.96)	313 (1 RCT)	Mixed population	6 months to 6 y	GRADE assessment not done by study authors
House dust mite allergen avoidance	1.08 (0.78-1.49)	3,040 (7 RCTs)	High-risk infants	1-8 y	GRADE assessment not done by study authors
Bacillus Calmette-Guerin	0.88 (0.79-0.98)	4,383 (2 RCTs)	Mixed population	13-18 months	GRADE assessment not done by study authors
Prenatal albendazole	hazard ratio = 1.58 (1.15-2.17)	2,507 (1 RCT)	General population	5 y	GRADE assessment not done by study authors
Prenatal praziquantel	hazard ratio = 1.15 (0.83-1.58)	2,507 (1 RCT)	General population	5 y	GRADE assessment not done by study authors
Prenatal antibiotic	RR = 1.28 (1.06-1.53)	2,098 (5 NRS)	General population	6 months to 4 y	Low (inconsistency, publication bias)

The comprehensive review of various interventions to prevent atopic dermatitis (AD) in early life reveals mixed results. Skin interventions with emollients or moisturizers showed a slight, non-significant benefit in high-risk infants, with moderate evidence. (40)Probiotics, especially when given to both infants and mothers, demonstrated a significant reduction in AD, while prebiotics and symbiotic showed non-significant effects with low to unassessed evidence. Vitamin D supplementation in pregnant women and infants indicated non-significant benefits. Hydrolyzed formulas showed varied effectiveness, with partially hydrolyzed formulas having moderate evidence of non-significant benefit and extensively hydrolyzed formulas showing very low certainty of effectiveness. Introducing

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allergenic foods like eggs and cow's milk early did not significantly impact AD prevention. Maternal dietary supplements, specifically prenatal blackcurrant seed oil, showed potential effectiveness in reducing AD, while omega-3 supplementation did not.(39) Dust mite avoidance, Bacillus Calmette-Guerin vaccination, and prenatal antimicrobial treatments produced inconsistent results, with Bacillus Calmette-Guerin vaccination showing some promise. Overall, while some interventions like probiotics and specific dietary supplements show promise, others require further investigation to confirm their efficacy.(43)

Although many possible interventions and potential risk factors (not reviewed here) have been studied, what can clinicians and patients do now? The process of translating evidence into recommendations for individual patients and populations follows defined standards .(41-42) Effectively addressing the prevention of allergy requires balancing benefits, harms, values, preferences, certainty of evidence, and contextual factors such as acceptability, feasibility, resource implications (including cost and time), equity, and practical considerations. Among the interventions listed in Table I, probiotic and vitamin D supplementation for pregnant mothers and/or infants, and avoiding unnecessary antibiotic exposure, may modestly reduce the risk of childhood AD. (45-44)However, the uncertain effects, combined with concerns about feasibility and burdens (both practical and financial), may make the decision to use supplements sensitive to individual preference. The small preventive effects of newborn BCG vaccination for AD also have uncertain applicability, accessibility, and acceptability in non-tuberculosis-endemic areas. Patients concerned about developing AD should be reassured that if it occurs, there are robust AD treatment guidelines that provide multiple safe and effective therapies aligning with patient values and preferences.

Future Directions of Atopic Dermatitis (AD):

Currently, the strongest predictors of atopic dermatitis (AD) risk are based on genome-wide association studies, which are typically unavailable outside research settings.(49) In the absence of AD risk prediction tools, studies often rely on a family history of allergic disease for inclusion criteria.(46) Developing valid, accurate, and accessible risk prediction tools would enable families to make informed decisions about their child's AD risk and identify those who may benefit most from preventive interventions. Additionally, it is crucial to consider the best methods for measuring AD in clinical trials and population-based studies. Measuring AD outcomes can be challenging due to the multiple definitions of AD and varying measures of AD

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severity. Ideally, interventions should aim to prevent the most severe and persistent forms of AD, as the benefits of preventing mild or self-limiting disease are less clear. (47)It is also important for studies to assess outcome effects after the intervention has ended to distinguish between true prevention of disease and merely delaying the detection of AD (treatment of existing AD rather than its prevention). We have provided several examples where early preventative effects were lost after follow-up. (48)

The Impact of Antihypertensive Drugs on the Increasing Prevalence of Eczema in older adults :

Recent studies have suggested a potential link between the use of antihypertensive drugs and an increasing rate of eczema. Antihypertensive medications, commonly prescribed to manage high blood pressure, include various classes such as beta-blockers, calcium channel blockers, diuretics, and ACE inhibitors. These medications can impact the immune system and skin barrier function, potentially leading to dermatological side effects.(49-50) Some evidence indicates that certain antihypertensive drugs may exacerbate or trigger eczema in predisposed individuals. For instance, beta-blockers have been reported to aggravate eczema symptoms by influencing inflammatory pathways and skin barrier integrity. Similarly, diuretics can cause dehydration of the skin, leading to dryness and increased susceptibility to eczema. As hypertension and eczema are both prevalent conditions, understanding this connection is crucial for clinicians to manage and mitigate potential risks, ensuring that patients receive effective treatment for hypertension without compromising skin health. Further research is needed to elucidate the mechanisms underlying this association and to develop strategies for preventing and managing eczema in patients requiring antihypertensive therapy.

4. Lifestyle Factors—Smoking and Alcohol Consumption

Although there is no consistent evidence that alcohol consumption directly causes eczema or flare-ups, adults with atopic dermatitis (AD) tend to have higher rates of cigarette smoking and are more likely to have consumed 12 or more alcoholic beverages, either in low or high quantities. Heavier current drinking has been associated with eczema across all racial groups compared to those who have abstained throughout their lives or currently drink lightly. Interestingly, eczema was

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linked to a higher likelihood of ever drinking 12 or more alcoholic beverages among whites (adjusted Odds Ratio [aOR], 1.15; 95% Confidence Interval [CI], 1.14–1.15), blacks (aOR, 1.46; 95% CI, 1.46–1.47), and American Indians (aOR, 5.92; 95% CI, 5.83–6.01), but not among Asian-Americans (aOR, 1.00; 95% CI, 0.99–1.00) [20].

The relationship between smoking, both active and passive, and adult AD remains controversial. Early and ongoing cigarette smoking, along with exposure to environmental cigarette smoke during childhood, have been linked to a higher incidence of adult AD and continued smoking in adulthood. Both current and former smoking are significant risk factors for adult AD compared to non-smoking. Moreover, the number of cigarette packs smoked per year is significantly associated with adult AD, suggesting a lifelong cumulative risk for current smokers. Additionally, non-smokers with adult AD reported significantly higher exposure to environmental tobacco smoke. Therefore, adults should be discouraged from smoking to prevent AD in themselves and their family members [21].

Data from German registries indicate that smoking patients with AD experience a higher disease burden and a distinct pattern of lesion distribution, with a 2.5 times greater likelihood of foot involvement. While the overall scoring of atopic dermatitis showed no difference between smokers and non-smokers, the severity of lesions—such as oozing, crusting, and excoriations—along with patient global assessment scores (PGA) of AD severity, were higher in smokers. Smokers also reported more intense pruritus and fewer weeks with well-controlled AD compared to non-smokers. Additionally, smokers had elevated total Ig E levels and were more likely to have an early diagnosis of asthma. Interestingly, the majority of smokers in this study were males in their forties (58.7%) [22]. Conversely, a study on US women did not find a significant association between current smoking and the incidence of AD [23].

Further research from Germany suggests that adults with AD may have higher rates of problematic drinking, drug use disorders, internet addiction, and gambling problems compared to the general population [24].

5. Stress

Psychological stress has long been recognized as a factor that can influence the course of AD. While chronic stress typically exacerbates pathogenic immune responses, acute stress triggers the body's efforts to restore homeostasis by activating the sympathetic axis (SA) of the autonomic nervous system, the hypothalamus–

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pituitary–adrenal (HPA) axis, and the neuronal neuroectodermic axis (NNA). This activation leads to vasoconstriction, neurogenic inflammation, and the release of pro-inflammatory neuromodulators followed by the activation of the anti-inflammatory cholinergic axis (CA) of the autonomic nervous system [25].

Adults with AD show hyperresponsiveness of the SA, leading to the transient release of cortisol from the adrenal glands into the bloodstream, along with the release of corticotrophin-releasing hormone (CRH), adrenocorticotropic hormone (ACTH), and cortisol from skin cells. These processes favor the Th2 immune response and inhibit the Th1 response. Endogenous glucocorticoids (GCs) can impair stratum corneum cohesion, epidermal barrier homeostasis, and innate immunity in normal skin [26].

Acute stress has been shown to worsen the clinical manifestations of AD in both adults and children, and there is a correlation between psychosocial stress and the onset or exacerbation of AD. Patients with AD generally have a significantly higher sympathetic tone compared to healthy controls at rest and are less capable of managing acute stress [27]. The impact of maternal psychological stress during pregnancy on the development of AD in offspring has been previously noted, but psychological factors continue to play a role in AD into adulthood. Stress and sleep disturbances have a bidirectional relationship with AD, acting both as potential causes and consequences of the disease [28, 29]. The mechanisms underlying the psychological aspects of eczema across all ages involve stress responses, glucocorticoid secretion, immune dysregulation, and the development of scratching behavior in response to pruritus [30, 31].

Pregnancy itself can influence the course of AD, as hormonal changes and immune modulation during this period can exacerbate or alleviate symptoms. The management of AD in pregnant women is crucial not only for the mother's health but also for reducing the risk of AD in the offspring. Effective early-life interventions, such as the use of probiotics, dietary modifications, and careful skin care, can potentially mitigate the severity of AD later in life.(23)

As individuals age, the cumulative effects of early-life AD, coupled with the natural aging process, can lead to a more challenging disease course in older adulthood. The skin becomes more vulnerable, and the immune system's ability to regulate inflammation diminishes, often leading to chronic and persistent AD. Understanding the connections between early-life factors and the manifestation of AD in older adults underscores the importance of a comprehensive, lifelong

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approach to managing this condition, starting from pregnancy and continuing through all stages of life.(15)

Conclusion :

The comprehensive review of various early-life interventions to prevent AD reveals mixed results. Skin interventions using emollients or moisturizers provided a slight, non-significant benefit in high-risk infants, supported by moderate evidence. Probiotics, especially when administered to both infants and mothers, significantly reduced the incidence of AD, while prebiotics and symbiotics showed non-significant effects with low to unassessed evidence. Vitamin D supplementation in pregnant women and infants, along with the use of hydrolyzed formulas, offered non-significant benefits, with varying levels of evidence. The early introduction of allergenic foods, such as eggs and cow's milk, did not significantly impact AD prevention. While prenatal dietary supplements, like blackcurrant seed oil, showed potential effectiveness in reducing AD, omega-3 supplementation did not. Other interventions, such as dust mite avoidance, Bacillus Calmette-Guerin (BCG) vaccination, and prenatal antimicrobial treatments, produced inconsistent results, though BCG vaccination showed some promise. The relationship between atopic dermatitis (AD) in older adults and its onset during pregnancy and early life highlights the lifelong impact of this chronic condition. AD often begins in infancy or childhood, influenced by genetic factors, environmental exposures, and immune system development. For those who experience AD in early life, the disease may persist or re-emerge in later years, particularly as the skin undergoes changes associated with aging. The early-life factors that contribute to the development of AD, such as prenatal exposure to allergens, maternal diet, and environmental triggers, can set the stage for the condition's persistence into older adulthood.

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Conflicts of Interest:

The authors declare no conflict of interest.

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