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Title: Prenatal and perinatal risk factors for atopic dermatitis in offspring - a literature review

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Abstract

Background: Atopic dermatitis (AD) is a chronic, inflammatory skin disease that significantly impacts quality of life. This condition most commonly affects children, occurring in 10-20% of the pediatric population. The pathophysiology of the disease is complex and characterized by the interaction of genetic factors, immune dysregulation, and environmental factors. The influence of prenatal and perinatal risk factors on the potential AD occurrence in offspring is crucial.

Aim: Assess the influence of prenatal and perinatal risk factors on the likelihood of AD occurrence in offspring, with particular emphasis on non-modifiable and modifiable risk factors, and the role of eliminating modifiable risk factors early in disease prevention.

Material and methods: A literature review was conducted using PubMed and Google Scholar. Original articles and reviews were analyzed. The studies included a pediatric population.

Result: Numerous proven prenatal and perinatal factors play a role in the development of AD in offspring. These can be divided into biological, environmental, behavioral, and clinical factors. A different classification takes into account modifiable and non-modifiable determinants.

Conclusions: Identifying modifiable risk factors that influence the risk of developing AD in offspring enables preventive measures to be taken, thereby reducing or eliminating them, and ultimately reducing the risk of the disease in offspring. Further research is needed to identify additional risk factors that can increase parents' awareness of prenatal and perinatal risk factors for AD in their offspring.

Keywords: Atopic dermatitis; Child; Pregnancy; Risk factors

Introduction

Atopic dermatitis (AD) is a chronic, recurrent inflammatory skin disease, primarily manifested by itching, eczematous lesions based on dermatitis, and a disruption of the skin barrier, which increases the risk of infection [1]. This disease significantly impacts quality of life, including through its impact on self-esteem and self-confidence, environmental stigma, and sleep disturbances caused by itchy skin and recurrent skin infections [2]. Studies show that AD most often occurs in children, with a prevalence of 10-20% in this age group, compared to 2.1-8.1% in the adult population. In the pediatric population, AD occurs in 30.48% of infants aged 1 to 12 months and 12.94% in children aged 1 to 7 years [1].

The pathophysiology of AD is complex and not fully understood, and determinants contributing to the development of the disease include genetic factors, immune dysregulation, and environmental factors. Genetic mutations, particularly in genes encoding structural proteins of the epidermis, such as filaggrin, are a recognized factor leading to impaired skin barrier function in AD [3]. The reduced immune response in AD involves the dominance of type 2 (Th2) helper lymphocytes, which also leads to epidermal barrier dysfunction. The influx of Th1, Th2, and Th17 lymphocytes into the skin, along with the cytokines they secrete, contributes to inflammation. The role of the skin microbiome, which is poorly differentiated in AD and characterized by the dominance of Staphylococcus aureus bacteria, which colonize lesions, exacerbating inflammation, is also important. These microorganisms dominate beneficial commensal bacteria such as Staphylococcus epidermidis [4]. Intestinal dysbiosis is also a significant risk factor for the development of AD. Metabolites produced by gut microorganisms affect the immune system by influencing T cell differentiation [5], and therefore any changes in the gut microbiome can modulate the function of host immune cells.

Environmental risk factors for the development of AD include diet, environmental pollutants, exposure to infections or medications, mode of delivery, and even the season of birth. Prenatal exposure shapes the gut microbiome and the child's immune system, while the postnatal environment may play a significant role in epithelial dysfunction and the development of allergies [6]. Therefore, it is crucial to understand the factors that increase the risk of developing AD in offspring and to implement preventive measures to reduce the likelihood of developing this disease in children.

Materials and methods

This study was conducted as part of a literature review examining the influence of prenatal and perinatal factors on the risk of developing AD in children. A systematic search of the PubMed and Google Scholar databases was conducted, and these sources were considered as the primary evidence base. The literature included reviews and research papers. The studies analyzed covered the pediatric population. Risk factors were assigned to specific categories for clarity and thematic organization. Both modifiable and non-modifiable factors were considered, and the pathophysiological mechanisms leading to the development of AD in children were described. Based on the collected data, protective factors were identified that, when administered prenatally and perinatally, may contribute to reducing the risk of AD occurrence in offspring.

Results

1. Per- and polyfluoroalkyl substances (PFASs)

Per- and polyfluoroalkyl substances (PFASs) are a group of compounds used in many industrial products, including lubricants and firefighting foams, but also in household and everyday products such as nonstick cookware, food packaging, baking paper, clothing, some furniture, cosmetics, and cleaning products.

Prenatal exposure to PFASs, through transplacental transmission to the fetus, is associated with the development of AD in girls during the first two years of life. Furthermore, postnatal exposure to newborns through breastfeeding leads to an increased risk of developing AD in children under 5 years of age. Infants are also exposed to PFASs in the home environment through exploration and hand-to-mouth contact [6].

The results of the conducted studies indicate a correlation between prenatal exposure to PFASs and elevated levels of IgE antibodies in the umbilical cord blood of fetuses, particularly males. Therefore, it has been hypothesized that PFASs may contribute to increased hypersensitivity to allergens.

Interestingly, exposure to PFASs and tobacco smoke during uterine life in children with the GSTT1-null and GSTM1-null genotypes is associated with a higher risk of developing AD in early childhood than in fetuses carrying the aforementioned genes.

The GSTT1 and GSTM1 genes encode glutathione transferases, which are involved in phase II detoxification. The absence of these genes is associated with weaker antioxidant defenses, which contribute to an increased risk of developing AD in early childhood.

2. Air pollutions

Air pollution can be caused by chemical, physical, or biological factors and poses a serious threat to public health. Chemical factors causing air pollution include gases such as sulfur dioxide, carbon monoxide, and nitrogen oxides; volatile organic compounds; heavy metals like cadmium, lead, and mercury; metalloids such as inorganic arsenic; and particulate matter. Several studies have examined the association between maternal exposure to chemical air pollutants during pregnancy and the subsequent risk of atopic dermatitis in children.

Exposure to lead in late pregnancy has been shown to increase the risk of developing AD in boys at 6 months of age. Furthermore, prenatal exposure to inorganic arsenic and concomitant exposure to inorganic arsenic and cadmium were associated with a higher risk of AD in young children [6]. In turn, the COCOA study documented the negative effect of lead and chromium in cord blood on the persistence and severity of atopic dermatitis [7].

In addition, particulate matter (PM) has been shown to cause skin barrier dysfunction and the formation of reactive oxygen species, resulting in induced oxidative stress, epigenetic changes, and skin inflammation [6]. Studies have shown that prenatal exposure to PM in the first trimester of pregnancy and skin barrier dysfunction were positively correlated with the occurrence of early-onset atopic dermatitis in offspring. Interestingly, high levels of exposure to PM with particles ≤2.5 μm in the first trimester, higher maternal prenatal stress, and male gender were associated with a higher incidence of AD at age 1 year. In turn, high levels of PM 2.5 in the first trimester of pregnancy, combined with low levels of vitamin D in cord blood, influenced the early onset of chronic atopic dermatitis, with the most sensitive period being the 6th to 7th week of pregnancy.

Prenatal exposure to tobacco smoke during pregnancy predisposes the mother to risk factors for developing AD later in life [6]. Furthermore, increased maternal exposure to fine PM particles synergizes with postnatal exposure to tobacco smoke and increases the risk of developing AD in infancy. Prenatal exposure to tobacco smoke is associated with elevated levels of miRNA-223 and impaired Treg cell function [8]. Infants exposed to

pollutants in utero who develop AD by the age of 1 year have lower levels of Treg cells in cord blood after birth [5]. This imbalance leads to increased immune activation and inflammation, which increases the risk of AD in offspring [8].

3. Gut microbiota

Disturbances in the development of the gut microbiome in early life and the associated disruptions in metabolite production may contribute to the development of AD in childhood [7]. Metabolites produced by gut microorganisms affect the immune system through their influence on T cell differentiation [5]. Also, intestinal dysbiosis leads to reduced production of bacterial metabolites with antioxidant properties, which leads to the generation of reactive oxygen species (ROS) and exacerbates inflammatory processes. In turn, elevated ROS levels perpetuate gut dysbiosis and damage the epithelial barrier, triggering systemic reactions and releasing bacterial and inflammatory mediators that reach the skin. This constitutes a chronic, self-reinforcing feedback loop that perpetuates inflammation and exacerbates the disease process [9]. Although the gut microbiome matures with age in both healthy children and infants with AD, in the latter, the gut microbiome has been observed to develop in a detrimental direction, characterized by abnormal production of short-chain fatty acids and increased IgE production [7].

Research has shown that the perinatal environment influences the development of an infant's gut microbiome. The mode of delivery has been shown to be significant. Vaginal infants have a rich and diverse microbiome. The feeding method also influences the gut microbiome of infants. Breastfeeding promotes the development of Bifidobacterium, which is beneficial for the gut microbiome. It has also been shown that infants, whose mothers receive perinatal antibiotic therapy, regardless of the delivery method, experience gut microbiota dysbiosis. Other factors influencing the infant's microbiome include gestational age at delivery. Preterm infants have impaired gut microbiota development—it is poorer and less stable. However, term infants develop a stable gut microbiota more quickly [5].

4. Maternal diet and antenatal nutrition

Maternal diet and prenatal nutrition can affect fetal development by influencing epigenetic and adaptive mechanisms, which in turn may impact the immune response and the development of AD later in life. Furthermore, nutrition during infancy also influences the development of atopy, which is related to the development of immunological tolerance.

BREASTFEEDING

Breastfeeding for the first 6 months of life is considered effective in preventing the development of AD. Breastfeeding for the first 4 months of life reduces the risk of atopy in the first 4 years of life [6]. Furthermore, as described above, breastfeeding affects the development of the infant's gut microbiota and increases the infant's immunological tolerance, reducing the risk of developing AD later in life [5].

VITAMINS

Vitamin E is a powerful antioxidant with lipophilic properties. The pathophysiology of AD involves inflammation and oxidative stress, and vitamin E protects cell membranes from lipid peroxidation, thus counteracting oxidative damage [10]. Inadequate vitamin E intake during pregnancy is associated with a higher risk of developing AD in children aged two years [6].

Vitamin A deficiency results in decreased immunity and increased susceptibility to skin inflammation and infections. There is evidence that Staphylococcus aureus colonization, along with concomitant vitamin A deficiency, plays a role in the pathophysiology of AD. Furthermore, vitamin A deficiency may increase the incidence of Staphylococcus aureus-related skin infections [10].

Vitamin D plays a significant role in regulating skin development, including the formation of the lipid layer and modulation of the immune system. Therefore, maintaining adequate vitamin D levels is crucial for the formation and maintenance of the skin barrier. Studies have shown a correlation between blood vitamin D levels and the risk of developing AD, with vitamin D levels being lower in AD patients compared to healthy individuals [10].

MICROELEMENTS

Zinc

A contributing factor in the development of AD is skin barrier dysfunction. Zinc is a trace mineral that may contribute to the repair of damaged skin by inhibiting the immune-inflammatory response and repairing epithelial integrity. However, maternal zinc intake during pregnancy has been shown not to protect against the development of AD in her offspring [10].

Iron

Studies have shown that iron deficiency is more common in children with AD, and AD is more common in children with anemia. It has been suggested that prenatal supplementation with folic acid and iron may reduce the risk of atopic dermatitis in children [10].

POLYUNSATURATED FATTY ACIDS

A mother's diet rich in oily marine fish and walnuts has a protective effect on the fetus due to the presence of polyunsaturated fatty acids, which beneficially affect the immune system and the functioning of cell membranes [6]. Essential unsaturated fatty acids are an important subgroup of polyunsaturated fatty acids. These include ω-3 alpha-linolenic acid and ω-6 linoleic acid. The body does not synthesize these acids, so they must be supplied through food.

Research on the association between ω-3 fatty acid intake and the incidence of AD in children is inconclusive. One study found that consuming ω-3 fatty acids during pregnancy did not alter the incidence of AD in children. However, another study found that maternal supplementation with ω-3 fatty acids provided little benefit in reducing the risk of allergies in offspring. It has been clearly demonstrated that prenatal supplementation of ω-6 fatty acids by women was associated with an increased risk of developing AD in children. Based on the above observations, it was concluded that the higher incidence of AD may be related to an imbalance in the composition of the diet, especially when the ratio of consumed ω-6 to ω-3 fatty acids is high [10].

SATURATED FATTY ACIDS

Scientific observations have shown an association between high saturated fatty acid (SFA) intake by breastfeeding mothers and an increased incidence of AD in infants [10]. Therefore, the consumption of fast food meals rich in SFA by breastfeeding mothers may be associated with the development of severe AD in their offspring.

ALCOHOL

Studies clearly indicate that alcohol consumption during pregnancy is associated with a significant and dose-dependent increased risk of AD in early infancy [6].

5. Prebiotics, probiotics, synbiotics

As mentioned in the previous sections, the pathogenesis of AD may be related to both disruption of the gut microbiota and dysregulation of the skin microbiome. Reduced gut microbiome diversity can trigger an immune response, contributing to the development of AD. Taking the gut microbiota-modulating formulas described below may contribute to a decrease in the incidence of AD in infants.

PREBIOTICS

It has been observed that adding dedicated prebiotic formulas to infant formula can lead to a reduction in the incidence of AD in infants at low risk of AD during the first year of life [10]. This may indicate a potential preventive effect of prebiotics in infants at low risk of AD. Another study revealed that adding similar formulas to a partially hydrolyzed whey formula did not prevent the development of atopy in high-risk infants compared to the same formula without prebiotics [10].

PROBIOTICS

A 2019 study found that probiotic therapy initiated prenatally and continued for the first six months after birth can reduce the risk of AD in infants and children at a later age [10]. Interestingly, the results of probiotic therapy administered solely postnatally appeared to have a negative effect; however, when probiotics were administered only to pregnant women, the effect was significantly more effective [10]. Furthermore, probiotic supplementation may also lead to a reduction in the risk of AD occurrence in children by age six with a high risk of allergies [10].

The most effective probiotic preparations are LP (Lactobacillus paracasei ssp. paracasei F19), Mix8 (Lactobacillus paracasei ST11, Bifidobacterium longum BL999), and Mix3 (Lactobacillus rhamnosus GG, Bifidobacterium animalis ssp. lactis Bb-12) [10]. Importantly, it has been observed that the influence of probiotic mixtures composed of multiple strains is better than that of single-strain preparations [6].

SYNBIOTICS

Breast milk can be considered a natural synbiotic because it contains probiotics (bacteria from the mother's intestines) and prebiotics (breast milk oligosaccharides), which may be the reason why breastfeeding is believed to be beneficial in preventing AD.

In summary, numerous studies have demonstrated the ability of probiotics, prebiotics, and synbiotics to prevent or alleviate the symptoms of AD, especially in women taking supplements during pregnancy.

6. Infections and antibiotics

Maternal infection during pregnancy is a clear risk factor for the development of AD in children [6]. Furthermore, exposure to antibiotics, both prenatal and in early childhood, particularly during the first two years of life, is associated with an increased risk of developing allergic diseases, especially AD [11]. Interestingly, exposure in childhood poses a greater risk than prenatal exposure [12]. Maternal antibiotic administration during labor, regardless of delivery method, has also been shown to result in gut microbiome dysbiosis, as mentioned above [5].

Antibiotic use has been shown to play a role in immune programming, leading to a decrease in Treg lymphocytes and an increase in the Th2 immune response, resulting in increased neonatal susceptibility to type 2 inflammation [5].

7. Delivery

As described above, the mode of delivery has an impact on infants' gut microbiomes. It has been shown that in infants born vaginally, the gut microbiome mirrors their mother's vaginal microbiome, with bacteria such as Lactobacillus, Prevotella, and Sneathia predominating. In contrast, infants born by cesarean section have a microbiome similar to that found on the skin's surface, with bacteria like Staphylococcus, Corynebacterium, and Propionibacterium dominating. Infants born vaginally have a rich and diverse gut microbiota. This microbiome provides protection and reduces the likelihood of allergic diseases such as AD developing [6].

8. Other risk factors

1. Psychological problems

There is conflicting data on the impact of psychological problems in pregnant women on the development of AD in children. Eun Lee et al. indicate that maternal psychological stress during the prenatal period increases the risk of AD developing in her offspring [7]. Similarly, it has been shown that psychological stress in the prenatal and postnatal period may lead to changes in epigenetic programming and the functioning of the immune system, which increases the susceptibility to AD in offspring [13]. On the other hand, Shuguang Chen and Sheng Chen report that mental disorders during pregnancy are associated with an increased risk of asthma in their children, but have no significant relationship with the development of AD in the offspring [14]. For this reason, it is important to prevent the occurrence of symptoms of psychological disorders. For example, physical activity is an effective method for alleviating anxiety symptoms [15]. Yoga, as well as aerobic and resistance exercise, has

been shown to relieve symptoms of depression and anxiety. Furthermore, physical activity reduces inflammation, contributing to improved health [16].

2. Diabetes and hypertension

Studies have shown that gestational diabetes in the mother is associated with an increased risk of AD in her children. However, no association has been found between hypertension during pregnancy and the AD occurrence in the offspring [17].

3. Season of birth

Papers have revealed that autumn and winter births (September-March) are associated with an increased risk of allergic diseases, particularly AD, in children [5].

4. Atopy in parents

Parental atopy, especially AD, is a risk factor for AD in offspring, and the likelihood increases with the number of atopic conditions in the parents and the number of parents affected by the condition [18]. The risk of disease is particularly increased in children during the first two years of life [19].

Discussion

The results of the analysis showed that many prenatal and perinatal risk factors can increase the likelihood of AD occurrence in children through various pathophysiological mechanisms. The first group comprises non-modifiable factors, such as genetic determinants (particularly filaggrin gene mutations), environmental aspects, like the season of birth, and clinical influences, including maternal prenatal illnesses or premature birth. The second group comprises modifiable risk factors: environmental determinants, such as maternal prenatal exposure to environmental pollutants, PFASs, and tobacco smoke; behavioral factors, including dietary habits and alcohol consumption during pregnancy; and clinical causes, like increased maternal exposure to infections during pregnancy and antibiotic therapy. These factors increase the risk of AD occurrence in offspring during the first period of life through their impact on skin barrier dysfunction, gut and skin microbiome dysbiosis, and immunological disorders [18]. The results described above are presented in tabular form (Table 1).

Table 1: Risk factors of AD developing in children classified according to modifiability, occurrence period, and category

Risk factor	Modifiability	Period	Category
Mother's industrial pollution and PFASs exposure	Modifiable	Prenatal	Environmental
Mother's tobacco smoke exposure	Modifiable	Prenatal	Behavioral

Mother’s eating and drinking habits (insufficient vitamin intake, high ratio of saturated to unsaturated fatty acids in the diet, alcohol consumption)	Modifiable	Prenatal	Behavioral
Mother’s infections and antibiotics are being taken	Modifiable	Prenatal	Clinical
Premature birth	Unmodifiable	Perinatal	Clinical
Cesarean section delivery	Modifiable/unmodifiable	Perinatal	Clinical
Maternal gestational diabetes, atopic dermatitis, and psychological disorders	Unmodifiable	Prenatal	Clinical
Autumn or winter season at the time of birth	Unmodifiable	Perinatal	Environmental
Formula milk feeding instead of breastfeeding	Modifiable/unmodifiable	Perinatal	Behavioral/clinical
Perinatal antibiotic therapy	Modifiable	Perinatal	Clinical
Genetic factors	Unmodifiable	Prenatal	Biological

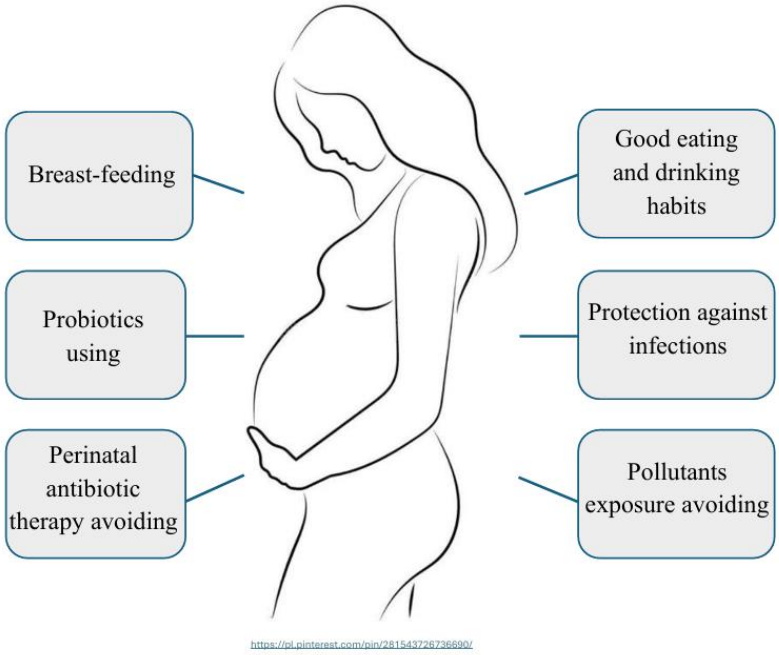
With knowledge of modifiable risk factors for AD occurrence in offspring, exposure to them can be reduced or eliminated very early in a child's life. The mother's prenatal diet plays a crucial role. It should be a rich source of vitamins, particularly A, D, and E, trace elements such as iron, and ω-3 unsaturated fatty acids. Furthermore, high intakes of ω-6 and saturated fatty acids have been shown to increase the risk of AD developing in offspring, so it is advisable to reduce their amount in the future mother’s diet. Alcohol consumption by pregnant women is also strictly prohibited. Breastfeeding plays a significant role in protecting newborns from the first days of life because breast milk, as a natural synbiotic, influences the development of beneficial bacteria in the gut microbiome and contributes to reducing the risk of AD occurrence later in life.

Another protective factor is exposure to environmental pollutants, tobacco smoke, and PFASs avoiding. It is recommended to check air pollution levels before leaving home and avoid visiting polluted environments. It would also be advisable for family members to stop smoking, as both active and passive exposure to tobacco smoke during pregnancy is a risk factor for AD developing in offspring. In the household, it is recommended to use only PFAS-free products. Everyday items containing PFASs are being gradually phased out, and new substitutes for these substances are appearing on the market, but their potential toxicity and long-term impact on the health of pregnant women and fetuses require further research [20].

It is also crucial to limit a pregnant woman's contact with infected people and prevent her from receiving antibiotic therapy. If there are clinical indications for antibiotic use, a physician needs to consider the time of

administration and dosage of the drug to minimize the exposure of the pregnant woman and the developing fetus. Furthermore, probiotic therapy has been shown to have a protective effect. When initiated prenatally and continued for the first six months after birth, it can reduce the risk of AD in infants and older-age children. The protective factors have been taken together and presented in graphical form (Figure 1).

Figure 1: Protective factors for AD development in children



Conclusion

The data collected in this study clearly indicate the influence of prenatal and perinatal risk factors on the occurrence of AD in offspring. These factors are divided into modifiable factors, whose elimination or reduction through the implementation of appropriate preventive measures can significantly reduce the risk of AD in offspring, and non-modifiable factors, which cannot be influenced. Another classification divides these factors into the following categories: biological, clinical, environmental, and behavioral. The final classification, considered in the above study, is based on time and distinguishes prenatal and perinatal factors.

The data presented in this study demonstrate that the AD development in children is multifactorial and results from the interaction of genetic factors, as well as environmental, behavioral, and clinical prenatal and perinatal determinants. Knowledge of modifiable factors is crucial, as their elimination or reduction early in a child's life can significantly reduce the risk of AD occurrence in offspring. Therefore, educating parents and caregivers on how to avoid risk factors and implement appropriate preventive measures is crucial. Further research and analyses are also necessary to identify other prenatal and perinatal risk factors in order to deepen knowledge in

this area and increase parental awareness, which will consequently contribute to reducing the AD incidence in offspring.

Disclosure

Author's contribution

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