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Original Article

Phthalates exposures and atopic dermatitis

Dermatology

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ABSTRACT

Background: Atopic dermatitis (AD) is one of the most common cutaneous diseases all over the world. It has a complex multifactorial etiology. It is a chronic, non-communicable disease with variable clinical features according to the age. The clinical picture of atopic dermatitis is usually presented as persistent itching, ketosis, and scaling. It is also associated with atopic march (atopic rhinitis, conjunctivitis, and bronchial asthma). AD has been divided into infantile, childhood, and adulthood phases based on some characteristic clinical features. Infantile AD appears at 2 to 6 months of age as papules and papulovesicles that may form large plaques that ooze and crust, mainly on the face, hands, and extensors, but the scalp, neck, and trunk may also be involved. The diaper area is usually spared; however, diaper dermatitis is very common in atopic children. Childhood AD is presented at the age of 2 years to puberty with dry, lichenified papules and plaques and involves the face, antecubital and popliteal areas, hands, and feet, which are more commonly affected. Environmental chemicals, such as phthalates, may cause immunological disorders and aggravate allergic diseases**.** Phthalates are now everywhere in the environment; they are commonly used as stabilizers and plasticizers in plastics, toys, medical equipment, and food packing, and they are among the most frequently encountered indoor pollutants as building materials.

Objective: This overview describes the potential detrimental effects of phthalates on children's AD to provide feasible strategies to reduce this.

Conclusion: Phthalate exposure is a risk factor for the development of AD in children. Careful observation of the child's environment to make sure of minimum exposure to phthalate and modify the risk of development of AD.

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INTRODUCTION

Atopic dermatitis (AD) (atopic eczema) is a complex multifactorial dermatologic disorder. It is a heterogeneous non-communicable disease with chronic relapsing and remitting course [1]. The clinical picture of lesions varies according to the age. In infants, lesions usually appear as tiny erythematous cheeks, but older children and adults usually appear as rashes on the elbows or knees (usually in the flexure aspect of joints), on the scalp and on the extensor aspect of the hands [2]. AD may be associated with secondary infection, failure to thrive, sleep deprivation, and impaired psychosocial well-being[3]. Diagnostic approach of AD is dependent mainly on clinical picture, no specific laboratory test or definite histopathological finding [4] . This review describes the potential detrimental effects of Phthalates on children's AD to provide feasible strategies to reduce this exposure.

Epidemiology

Atopic dermatitis (AD) is one of the most prevalent cutaneous disorders all over the world. AD ranked as the third most prevalent dermatologic condition[5].

During the last few years, there has been increase in the prevalence of AD up to 8%-30% in pediatrics with great variation throughout the world especially in industrialized countries [6].

Etiology and pathogenesis

The pathogenesis of atopic dermatitis is multifactorial, including genetic, immunologic, and environmental factors that disturb epidermal barrier [7].

- **Epidermal barrier dysfunction:** formation of epidermal barriers requires the integrity of the following: stratum corneum (SC) or corneocytes, cornified envelopes, lipids such as ceramide and cholesterol, junctional proteins, proteases, protease inhibitors, and antimicrobial peptides [8]. Any defect in filaggrin protein affects the epidermal barrier function through affection of corneocyte cohesion, promotion of water loss, different lipid content, more susceptibility to skin infection, and finally, a rise in skin pH, which negatively affects the natural moisturizing factor (NMF) of skin[9]. Epidermal barrier dysfunction may be acquired due to mechanical disruption and chemical irritants [10].
- **Genetics:** Homozygous filaggrin mutations are associated with an elevated risk of development of severe AD with earlier onset, longer persistence, and skin infections[11]. Epidermal barrier dysfunction triggers inflammation through the release of thymic stromal lymphopoietin (TSLP) and other cytokines by damaged keratinocytes [12].
- **Immunological dysfunction:** AD is a complex immune-mediated disease with activation of Th2 cells and immunologic cascade, which leads to the AD phenotype, which is considered the first manifestation of the allergic march. Pruritus in AD is associated with skin-homing T cells and increased IL-31 levels [13]. Innate immune response: Decreasing the antimicrobial peptides (AMP) may increase liability to multiple infections in AD patients due to colonization with Staphylococcus aureus bacteria that exacerbates cutaneous lesions [14]. IL-17 regulates AMP in keratinocytes, and its downregulation may increase susceptibility to skin infections in AD patients [15]. Adaptive immune response: In the initial and acute phase of AD, a Th2-dependent immune response (IL-13, IL-4, TSLP, and eosinophils) is predominant, while in chronic AD cutaneous lesions, a Th1/Th0-dominant immune response with an increase of the following cytokines (GM-CSF, IFN-γ, IL-12, and IL-5) [16].

Determinants of atopic dermatitis

The development and course of AD are significantly influenced by a wide range of risk factors. They fall into two categories: modifiable and non-modifiable. Factors that are difficult to alter through routine activities are referred to as non-modifiable factors. They are helpful in determining who is at a high risk of developing AD, but not in developing measures to prevent AD [17]. Medical treatments, indoor and outdoor environmental factors, and individual lifestyle factors are among the modifiable elements that enable intervention to lower the prevalence of disease [18]. The fundamental feature of AD is the breakdown of the epidermal barrier, which can be caused by both inherited and environmental factors. This increases the skin's sensitivity to allergens, irritants, microbes, toxins, and pollutants, which can cause sensitization, inflammation, and colonization [19].

Industrialization and urbanization have increased people's exposure to indoor and outdoor environmental toxins, posing a significant health risk [20]. Phthalate acid esters (PAEs) are a chemical compound with a basic structure of phthalic acid and alkyl groups' alcohol via an ester bond. They are well-known for their endocrine disruptive properties. High-molecular-weight phthalates (HMWPs) and low-molecular-weight phthalates (LMWPs) are derivatives of phthalate acid esters. Their environmental properties are influenced by their general lipophilia [21].

Human exposure to phthalates

Phthalates released from various household products contaminate dust or food and are absorbed into the human body through inhalation, ingestion, or dermal contact and parenteral routes. They were also found to cross the placenta-blood barrier, which is the major exposure route of the fetus [22].

1. **Inhalation:** Indoor air and dust can be sources of phthalates, which are substances that leach from various materials and products. These include building products, household furnishings, upholstered furniture, floor tiles, clothing, and accessories, such as children's PVC backpacks. Additionally, phthalates may also be present in the interiors of automobiles due to plasticized components. Proximity to phthalate manufacturing industries further contributes to this exposure [23].

2. **Ingestion:** Phthalates can come from various sources, including medications, nutritional supplements, enteral nutritional formulas, and sucking toys for kids. Processing, handling, shipping, packing, and storage can all lead to food contamination. High levels of phthalates are seen in bread, meat, oils, fish, seafood, dairy products, and beverages [24].

3. **Dermal contact:** Lower molecular weight phthalates, which are included in a variety of goods, including toys, cosmetics, and clothing, are mostly exposed through skin absorption. Dimethyl phthalate (DMP) and dibutyl phthalate (DBP), two important phthalates that are known to be potentially harmful, may be found in pesticides and expose people to them through their skin or lungs [25].

4. **Parenteral route& medical devices:** A range of medical devices manufactured from PVC plasticized with DEHP (di(2-ethylhexyl) phthalate) are deployed in healthcare settings to provide essential medical care. These devices include endotracheal tubes, intravenous (IV) fluid bags and tubing, as well as equipment for extracorporeal membrane oxygenation and dialysis, and umbilical vessel catheters [26]. The use of these devices can lead to the leaching of DEHP, resulting in acute

exposures that may surpass the established tolerable daily intake, particularly after medical procedures [27] . Research has indicated a correlation between the intensity of medical care and the frequency of DEHP-containing device usage with elevated levels of urinary metabolites of DEHP, suggesting that increased exposure occurs with more intensive or frequent medical interventions [28] .

Phthalate metabolite exposure and child health

Phthalates are frequently present in a wide range of consumer goods, and prolonged exposure to their metabolites in humans raises questions about possible health implications, especially for young people [29] . Recent studies indicate that children's acceptable phthalate consumption has frequently been surpassed, suggesting heightened vulnerability to problems with the skin barrier, as well as the possibility of immunological disorders and the escalation of allergic diseases. Nevertheless, information regarding the negative health effects of phthalate exposure is still scarce and even contradictory[30]

The relationship between AD and phthalate exposure

Early childhood eczema risk may be impacted by phthalate exposure during the final trimester of pregnancy. Early-life exposure to phthalates raises immunoglobulin E (IgE) levels, which causes atopic dermatitis (AD) in kids. AD in children ages 3 to 7 was substantially linked to higher levels of phthalate esters in household dust [31] . According to urine phthalate metabolites, children have higher daily phthalate intakes than adults, and they also demonstrated a greater susceptibility to AD when exposed to phthalate in household dust. Compared to adults, children are much more vulnerable and sensitive to indoor environmental hazards like phthalate exposure, mainly during early growth. Because they breathe more air per kilogram of body weight than adults and spend a longer time at indoor facilities such as school or day care centers[32]. These results indicate that children exposed to phthalates may be more susceptible to fragile skin barrier function. Phthalates can cause immunological disorders and aggravate allergic diseases[31] .

The effects of early life phthalate exposure on IgE levels can potentially cause AD in children in a 10-year cohort**.** Another study has reported that higher levels of phthalate esters in house dust were significantly associated with atopic dermatitis among children aged between 3 and 7 years. Children showed a clearer association of AD with phthalate than adults [33].

The connection between phthalate exposure and the onset or worsening of allergic diseases remains uncertain. Investigating this link presents numerous challenges. For instance, evaluating human exposure to phthalates is quite difficult. Currently, the methods employed for assessing exposure involve measuring the levels of urinary phthalate metabolites, which indicate the overall exposure amount, or analyzing the quantity of phthalates present in indoor dust [33].

However, urinary levels fluctuate based on the host's metabolic rate, and the exposure route may have a more significant impact relative to the target organ. Additionally, factors such as the chemical structure and duration of exposure could influence disease outcomes. Experimental research has proposed several mechanisms, including receptor interactions, oxidative stress, and transcriptional and epigenetic pathways, but the precise molecular mechanisms remain not fully understood [33].

Given that phthalates are among the most prevalent environmental pollutants and are commonly encountered by humans, even minimal evidence should not be disregarded. Additional research is necessary to identify environmental risk factors, such as phthalates, to enable the effective prevention and treatment of allergic diseases [33].

Preventive strategies

The main exposure route for the general public to phthalates and BPA is through diet. Interventions that use non-plastic containers, like stainless steel, have been effective in reducing BPA exposure. Many interventions were short-term, leading to biomarker levels returning to baseline afterward. Access to fresh and organic foods is limited for some families due to income and availability, and even carefully chosen food items can still result in exposure to chemicals [34].

Data on ingredient composition in consumer products is often hard to obtain due to a lack of legal reporting requirements in the US. For instance, phthalates, commonly used in fragrances, do not need to be labeled as it is considered a "trade secret" by the FDA. Consequently, consumer-level strategies to avoid such chemicals may not effectively reduce exposure, highlighting the need for policies aimed at minimizing population-level exposure [34].

The use of phthalate-free medical devices and IV bags can reduce human exposure to harmful substances. Despite this, the US FDA has made minimal progress in removing phthalates and other endocrine-disrupting chemicals from medical equipment over the past 20 years, even after recommending alternatives to phthalate ester-containing devices. DEHP can make up to 40% of the weight in IV bags that contain it. In 2021, US Congress members urged the FDA to address this issue, highlighting the need for research into the barriers preventing healthcare providers from adopting phthalate ester-free medical equipment [34].

The use of various derivatives of phthalates was prohibited in toys and childcare products in 2007, specifically in toys designed for mouthing. After 2015, various derivatives of phthalate, categorized as a reproductive toxic category 1B, could not be used for any purpose within the EU without authorization. Using these strategies will yield optimal results for patients

with AD [34]. One of the important screening tests for measuring exposure to phthalates is the measurement of urinary MEHP (mono-2-ethylhexyl phthalate) levels, which may be more important and more relevant to studying the association between exposure to DEHP (di-2-ethylhexyl phthalate) and adverse health outcomes [34].

CONCLUSION

Variable patterns of exposure to phthalates are a risk factor for the development of AD in children. Careful observation of the child's environment to make sure of minimum exposure to phthalates. With an understanding of phthalates and their implications in children, the physicians need to be aware of the possible harmful effects of these chemicals deliberately added to emollients or that unintentionally are released from the plastic containers into the products.

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الملخص العربى

التعرض للفثالات والتهاب الجلد التأتبي

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ملخص البحث:

الخلفية: يُعد التهاب الجلد التأتبي (AD) أحد أكثر الأمراض الجلدية شيوعًا في جميع أنحاء العالم و له مسببات معقدة متعددة العوامل .وهو مرض مزمن غير معدي، وتختلف أعراضه السريرية باختلاف العمر .عادةً ما تظهر الصورة السريرية لالتهاب الجلد التأتبي على شكل حكة مستمرة، واحمرار بالجلد وتقشر .كما يرتبط أيضًا بأعراض التهاب الجلد التأتبي (التهاب الأنف التأتبي، والتهاب الملتحمة، والربو القصبي) .قُسّم التهاب الجلد التأتبي إلى مراحل رضعية، وطفولة، وبلوغ، بناءً على بعض الأعراض السريرية المميزة .يظهر التهاب الجلد التأتبي لدى الرضع في عمر شهرين إلى ستة أشهر على شكل حطاطات وحويصلات قد تُشكل لويحات كبيرة تتسرب وتتقشر، خاصةً على الوجه واليدين والعضلات الباسطة وهي المناطق الأكثر تأثرًا، ولكن قد تُصيب فروة الرأس والرقبة والجذع أيضًا .عادةً ما تُجنّب منطقة الحفاضات الإصابة؛ ومع ذلك، يُعد التهاب الجلد التأتبي شائعًا جدًا لدى الأطفال من عمر سنتين وحتى سن البلوغ .قد تسبب المواد الكيميائية البيئية، مثل الفثالات، اضطرابات مناعية وتؤدي إلى تفاقم أمراض الحساسية .تنتشر الفثالات الآن في كل مكان في البيئة؛ فهي تُستخدم عادةً كمثبتات وملدنات في البلاستيك، والألعاب، والمعدات الطبية، وتغليف المواد الغذائية، كما أنها من بين أكثر الملوثات الداخلية شيوعًا كمواد بناء .الهدف: تصف هذه النظرة العامة الآثار الضارة المحتملة للفثالات على مرض التهاب الجلد التأتبي لدى الأطفال، وذلك لتقديم استراتيجيات عملية للحد منه .

الاستنتاجات: يُعد التعرض للفثالات عامل خطر للإصابة بمرض التهاب الجلد التأتبي لدى الأطفال .لذا، فإن المراقبة الدقيقة لبيئة الطفل للتأكد من الحد الأدنى من التعرض للفثالات.

الكلمات المفتاحية: الأتوبية؛ التهاب الجلد التأتبي؛ التعرض للفثالات.

الباحث الرئيسي:
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