TASHKENT MEDICAL ACADEMY

100 TMA ANNIVERSARY

Journal of Educational and Scientific Medicine

Issue 2 | 2025

OAK.uz

Google Scholar

ISSN: 2181-3175

Journal of Education & Scientific Medicine

ISSN 2181-3175

Tashkent Medical Academy

Review Article

Open © Access

Epidemiological and Demographic Issues of the Prevalence of Atopic Dermatitis

Fatima Irsaliyeva[1], Gulmira Razikova[2]

ABSTRACT

Over the past decade, significant progress has been made in understanding the pathogenetic mechanisms of the development and course of atopic dermatitis. To a certain extent, this knowledge was obtained thanks to a breakthrough in large-scale research on the epidemiology and treatment results of atopic dermatitis, as well as genetic studies, immunological and in-depth molecular biochemical studies for this disease.

***Keywords**: atopic dermatitis, epidemiology, demography*

One of the large-scale (global) studies of the epidemiology of atopic dermatitis was presented in the Global Burden of Disease resource, which has been updated regularly since 1990 [1].

The Global Burden of Disease Study is an annually updated resource for examining disease-related morbidity and mortality worldwide.

Throughout the entire period of research, it was found that atopic dermatitis occupies one of the leading places among diseases included in the category of non-fatal diseases. According to international DALY data, atopic dermatitis ranks 15th and has a high burden of disease among skin diseases, which is characterized by a progressive decrease in its quality and an increase in disability throughout the patient's life.

Statistics confirm the increase in the incidence of atopic dermatitis over the past 30 years. So, if at the end of the XX century the total number of atopic dermatitis was equal to 123 million, then after 30 years the increase

[1] DSc, Associate Professor, Professor of the Department of Allergology and Clinical Immunology, Tashkent Medical Academy, Tashkent, Uzbekistan, e-mail: irsalieva73@mail.ru, ORCID: 0009-0006-0114-2870.

[2] PhD, Republican Specialized Scientific and Practical Medical Center of Allergology and Clinical Immunology, Tashkent, Uzbekistan, e-mail: razikova055@icloud.com, ORCID: 0009-0006-9463-4099.

was noted by 28.6% and was already equal to 171 million.

In the 90s of the last century, Sweden (327 patients), Great Britain (284 patients) and Iceland (277 patients) were considered to be the countries leaders in the incidence of atopic dermatitis. Uzbekistan in this global list was declared 85 patients per 100,000 population on a par with Tajikistan and Armenia.

Countries in the Asia-Pacific region have already made a significant contribution to the increase in the number of cases of atopic dermatitis in the global system, in contrast to previous periods. So, according to Chinese health sources, there were 5,837,355 cases and 35,583,695 common cases of atopic dermatitis at this time. China is followed by India with 3,739,094 cases and 25,923,780 prevalent cases, Indonesia with 1,437,343 cases and 9,679,480 prevalent cases, and Pakistan with 782,596 cases and 4,972,106 prevalent cases.

In the United States, the prevalence of atopic dermatitis was noted in 888,977 cases and 8,610,796 common cases.

As mentioned above, the highest prevalence of atopic dermatitis was recorded in the high-income Asia-Pacific region at 4 876 per 100 000 population and in Central Asia at 4 678 per 100 000 population. In contrast, the lowest prevalence rates of atopic dermatitis were recorded in countries in Africa, including Central, Eastern, Southern and Western Sub-Saharan Africa, with 1,081, 1,082, 1,083 and 1,102 per 100,000 population, respectively.

In most regions, the prevalence of atopic dermatitis remained stable from 1990 to 2019; the sharpest increase was recorded in Kenya – by only 5.3%, and the largest decrease was noted in the Maldives – by the same 6.6%.

The distribution of atopic dermatitis by age groups shows that there are 2 peaks of growth in its development among patients of early childhood and among patients of middle and older age.

The global burden of disease revealed a moderate positive correlation between a country's gross domestic product and the burden of disease. Global burden of disease data support the high importance of atopic dermatitis, which has remained stable since 1990 but shows significant geographic variation. Lifestyle factors, partly related to affluence, are likely important factors in disease. However, the methodology for estimating the global burden of disease needs further refinement to include environmental risk factors, such as UV exposure, to better understand geographic and age differences in the burden of disease.

The Global Burden of Disease study estimated the overall prevalence of atopic dermatitis to be 15-20% among children and up to 10% among adults.

Although atopic dermatitis is traditionally thought to have a lower prevalence in Africa compared to Europe, more recent studies have shown that atopic dermatitis is more common in children, reaching 31% in Ghana, possibly due to increasing urbanization [2].

Population-based studies of atopic dermatitis in sub-Saharan Africa have also revealed a growing prevalence in this region, where more than 15% of children suffer from this disease [3].

Given the limited resources in these regions, there is a need for additional collaboration with pharmaceutical companies to improve governance in these areas.

In order to determine the reasons for the increase in the prevalence of atopic dermatitis in Africa and Latin America, studies have been carried out aimed at the importance of racial factors of patients along with exposure to pollutants and other environmental hazards, which significantly affects the restructuring of acquired immunity and may affect the genetic transformation of the innate immune system [4, 5].

Thanks to such studies, an assumption was made about the variability of the impact of factors for the development of atopic dermatitis with the change of generations, which are characterized by an increase in aberrant immune reactions. Such changes can naturally be based on epigenetic and other mechanisms.

On the other hand, the authors concluded that both social and public policies in general play an important role in the increase in the incidence of atopic dermatitis. To a certain extent, such transformations can affect different populations for a long time after their impact.

In studies conducted by J.M. Biagini et al. [6] The significance of endotypes of atopic dermatitis between Caucasian and African children has been clearly demonstrated. They have shown that black children show a higher risk of developing atopic dermatitis despite a more intact skin barrier, as evidenced by lower transepidermal water loss in lesions and without lesions and higher expression of filaggrin outside lesions.

In response to these questions, the American Academy of Allergy, Asthma, and Immunology Committee on Low-Income Populations presented a report highlighting the need for a tiered approach between patients, health care providers, government, nonprofits, and professional societies to more effectively address inequities in atopy [7]. This report highlights the lack of research on differences specifically within atopic dermatitis and suggests

that effective interventional trials in atopic dermatitis should improve reporting and consideration of race and ethnicity in their results.

Studies aimed at assessing the influence of the environment on the incidence of atopic dermatitis are more devoted to the study of problems associated with ecosystem disturbances. For example, the studies of S.K. Park et al. [8] Information has been obtained regarding the role of pollutants in the development of atopic dermatitis. They conducted a study in 209,168 people from the Republic of Korea between 2008 and 2013, which examined the long-term average concentration of air pollutants before being diagnosed with atopic dermatitis. A significant association has been shown between the incidence of atopic dermatitis and exposure to particulate matter, sulphur dioxide, nitrogen dioxide and carbon monoxide. These associations persisted after adjusting for age, sex, income, comorbidities, and meteorological variables.

In a study conducted in Tasmania by scientists from the University of Melbourne, led by D.J. Lopez et al. [9] It has been proven that an increase in the concentration of nitrogen dioxide is directly related to an increase in the incidence of atopic dermatitis. Moreover, male patients were a priority. Studies of this level complement the information on the role of environmental disorders in the development of atopic dermatitis.

Statistical studies in this area have confirmed that parents suffering from allergic bronchial asthma, allergic rhinitis, food allergies, etc., transmit to their offspring the likelihood of developing atopic dermatitis 1.5-2 times more often. Along with this, in the studies conducted by T. Torres et al. [10] It has been shown that in such families, in the presence of atopic dermatitis in 1 of the parents, the risk of developing this disease in children can increase by 3 times, and in the case of atopic dermatitis in both parents, the likelihood of developing atopic dermatitis in children increases by 5 times.

Back in 1993, the Danish scientist F.S. Larsen [11], based on the results of an analysis of a large archival material of a large dermatological clinic over the past 50 years, came to the conclusion that the risk of developing atopic dermatitis among monozygotic twins ranges from 72% to 86%, while among dizygotic twins this figure has decreased to 21%.

It is known that there are a large number of interrelated and interacting genes, including those involved in the development of atopic dermatitis. Unfortunately, their decoding has not always been successful in determining the topical significance of indicators. Genes themselves are also subject to various heredity phenomena under the influence of various factors, such as epigenetic changes, incomplete gene penetrance and genomic imprinting. one different chromosomal locus containing genes for predisposition to atopic dermatitis [12].

According to B. Nedoszytko et al. [13], several groups of genes are currently known, among which the most important are genes encoding structural and functional proteins of the epidermis, and genes encoding proteins regulating innate and acquired immune responses. Gene mutations in the first group lead to a violation of the barrier function of the epidermis. The most popular of this group is the filaggrin gene mutation, which is considered one of the main genes for atopic dermatitis.

The filaggrin gene is located in the epidermal differentiation gene complex on the long arm of chromosome 1q21. The epidermal differentiation gene complex contains 27 genes, 14 of which are expressed in the process of final differentiation of keratinocytes and are mainly proteins in the keratinized membrane.

The remaining 13 genes located in the epidermal differentiation gene complex are genes encoding proteins that, according to the assumptions of A.P. South et al. [14] will play the role of signal transducers in the differentiation of keratinocytes and other cells and tissues.

The filaggrin mutations 2282del4 and R501X are the main mutation variants in Europeans. Both are null alleles, which leads to the lack of production of the protein encoded by the genes. [15] Filaggrin mutations have been shown to be a high-risk factor for the development of atopic dermatitis and are associated with early onset and severe phenotype. It is worth emphasizing that the mutation can occur in asymptomatic patients and that the absence of the mutated gene does not protect against the disease.

On the other hand, there are suggestions about the potential effect of filaggrin mutations on increased IgE levels, on the provocation of the development of an atopic state, including the development of atopic dermatitis, bronchial asthma, and other allergic diseases [16].

The starting product of the filaggrin gene is profilaggrin, a highly phosphorylated molecule rich in histidine, which is the main component of keratohyalin granules. Filaggrin is formed from an insoluble and functionally inactive precursor molecule under the influence of the proteolytic activity of enzymes from the group of serine proteases (e.g., Caspase-14) [17].

The resulting filaggrin monomers aggregate keratin fibers due to the catalytic activity of the enzyme transg-

lutaminase-1, which leads to flattening of cells. The so-called corneocytes build the stratum corneum [18]. In addition to filaggrin, many other proteins build a keratinized shell, such as loricrin, involucrin, and small proteins rich in proline.

Corneocytes are the scaffold for the extracellular matrix of lipids. In general, corneocytes protect the skin from excessive water loss, maintain the appropriate skin pH, inhibit the growth of Staphylococcus aureus, and limit the penetration of antigens into the deeper layers. Further transformations and degradation of filaggrin lead to the formation of glutamine, histidine, alanine, and their derivatives, such as pyrrolidone carboxylic acid and urocanic acid, which are part of the natural moisturizing factor [19].

Mutations leading to impaired protein synthesis cause increased transepidermal water loss, excessive dryness of the skin, increased pH on the surface of the skin, and disruption of the proportions and amounts of free fatty acids, ceramides, and triglycerides. Claudins and Occludins) belong to a group of genes responsible for the integrity and proper function of the epidermal barrier. It is these transmembrane and intracellular proteins that form complexes that connect neighboring cells, called tight junctions [20, 21]. They regulate the passage of ions, water, and solutes. In the epidermis, they are mainly located in the granular layer and are responsible for differentiation and keratinization. Damage to these proteins leads to increased water loss, dry skin, and infiltration and presentation of antigens in Langerhans cells. In patients with atopic dermatitis, there was a decrease in the expression of tight junction proteins and an inverse correlation of claudin-1 Th2 biomarkers [22, 23].

Other genes involved in the pathogenesis of atopic dermatitis at the level of the epidermal barrier are genes encoding a serine protease inhibitor (SPINK-5/LEKT1, cystatin A), genes encoding epidermal proteases: mast cell chymase gene, epidermal chymotrypsin and trypsin genes, epidermal N-methyltransferase gene (responsible for histamine degradation) [24].

Thus, atopic dermatitis remains one of the diseases with a tendency to increase, the true cause of which is still far from certain, despite global studies.

Conflict of interest – the authors declare the absence of a conflict of interest

REFERENCES

1.Laughter MR, Maymone MBC, Mashayekhi S, Arents BWM, Karimkhani C, Langan SM, et al. The global burden of atopic dermatitis: lessons from the Global Burden of Disease Study 1990–2017. Br J Dermatol. 2021;184(2):304–9.

2.Rosenbaum BE, Klein R, Hagan PG, Seadey MY, Quarcoo NL, Hoffmann R, et al. Dermatology in Ghana: a retrospective review of skin disease at the Korle Bu Teaching Hospital Dermatology Clinic. Pan Afr Med J. 2017;26:125.

3.Skevaki C, Ngocho JS, Amour C, Schmid-Grendelmeier P, Mmbaga BT, Renz H. Epidemiology and management of asthma and atopic dermatitis in Sub-Saharan Africa. J Allergy Clin Immunol. 2021;148(6):1378–86.

4.Martinez A, de la Rosa R, Mujahid M, Thakur N. Structural racism and its pathways to asthma and atopic dermatitis. J Allergy Clin Immunol. 2021;148(5):1112–20.

5.Croce EA, Levy ML, Adamson AS, Matsui EC. Reframing racial and ethnic disparities in atopic dermatitis in Black and Latinx populations. J Allergy Clin Immunol. 2021;148(5):1104–11.

6.Biagini JM, Kroner JW, Baatyrbek Kyzy A, Gonzales A, He H, Stevens M, et al. Longitudinal atopic dermatitis endotypes: An atopic march paradigm that includes Black children. J Allergy Clin Immunol. 2022;149(5):1702–10.e4.

7.Davis CM, Apter AJ, Casillas A, Foggs MB, Louisias M, Morris EC, et al. Health disparities in allergic and immunologic conditions in racial and ethnic underserved populations: A Work Group Report of the AAAAI Committee on the Underserved. J Allergy Clin Immunol. 2021;147(5):1579–93.

8.Park S.K., Kim J.S., Seo H.M. Exposure to air pollution and incidence of atopic dermatitis in the general population: A national population-based retrospective cohort study. // J. Am. Acad. Dermatol. – 2021; 87(6):1321-1327.

9.Association between ambient air pollution and development and persistence of atopic and non-atopic eczema in a cohort of adults. / D.J. Lopez, C.J. Lodge, D.S. Bui, et al. // Allergy. - 2021; 76(8):2524-2534.

10.Update on Atopic Dermatitis. / T. Torres, E.O. Ferreira, M. Gonçalo, et al. // Acta Med. Port. - 2019; 32:606–613.

11.Larsen F.S. Atopic dermatitis: A genetic-epidemiologic study in a population-based twin sample. // J. Am. Acad. Dermatol. - 1993; 28: 719–723.

12.Multi-ancestry genome-wide association study of 21,000 cases and 95,000 controls identifies new risk loci for atopic dermatitis. / L. Paternoster, M. Standl, J. Waage, et al. // Nat. Genet. 2015; 47: 1449–1456.

13. Genetic and Epigenetic Aspects of Atopic Dermatitis. / B. Nedoszytko, E. Reszka, D. Gutowska-Owsiak, et al. // Int. J. Mol. Sci. 2020; 21: 6484.

14. Human epidermal differentiation complex in a single 2.5 Mbp long continuum of overlapping DNA cloned in bacteria integrating physical and transcript maps. / A.P. South, A. Cabral, J.H. Ives, et al. // J. Investig. Dermatol. 1999; 112: 910–918.

15. Loss-of-function variations within the filaggrin gene predispose for atopic dermatitis with allergic sensitizations. / S. Weidinger, T. Illig, H. Baurecht, et al. // J. Allergy Clin. Immunol. 2006; 118: 214–219.

16. Cepelak I., Dodig S., Pavic I. Filaggrin and atopic march. // Biochem. Med. 2019; 29: 020501.

17. Caspase-14 is required for filaggrin degradation to natural moisturizing factors in the skin. / E. Hoste, P. Kemperman, M. Devos, et al. // J. Investig. Dermatol. 2011;131:2233–2241.

18. Cell death by cornification. / L. Eckhart, S. Lippens, E. Tschachler, W. Declercq. // Biochim. Biophys. Acta. 2013;1833:3471–3480.

19. Skin barrier in atopic dermatitis: Beyond filaggrin. / M.C. Zaniboni, L.P. Samorano, R.L. Orfali, V. Aoki. // An. Bras. Dermatol. 2016;91:472–478.

20. Morita K., Miyachi Y., Furuse M. Tight junctions in epidermis: From barrier to keratinization. // Eur. J. Dermatol. 2011;21:12–17.

21. Tsukita S., Furuse M. Claudin-based barrier in simple and stratified cellular sheets. // Curr. Opin. Cell Biol. 2002;14:531–536.

22. Tight junction defects in patients with atopic dermatitis. / A. De Benedetto, N.M. Rafaels, L.Y. McGirt, et al. // J. Allergy Clin. Immunol. 2011;127:773–786.e7.

23. Claudin-based tight junctions are crucial for the mammalian epidermal barrier: A lesson from claudin-1-deficient mice. / M. Furuse, M. Hata, K. Furuse, et al. // J. Cell Biol. 2002;156:1099–1111.

24. Kiyohara C., Tanaka K., Miyake Y. Genetic susceptibility to atopic dermatitis. // Allergol. Int. 2008;57:39–56.

How to Cite: *Irsaliyeva F.Kh., Razikova G.R. Epidemiological and demographic issues of the prevalence of atopic dermatitis. // Journal of Educational & Scientific Medicine. 2025;Vol.1(2):36-41.*

ATOPIK DERMATIT TARQALISHIDA EPIDEMIOLOGIK VA DEMOGRAFIK MASALALAR

Fotima Irsaliyeva, Gulmira Razikova

Toshkent, O'zbekiston

XULOSA

So'nggi o'n yil ichida atopik dermatitning rivojlanishi va jarayonining patogenetik mexanizmlarini tushunishda sezilarli yutuqlarga erishildi. Bu ma'lum darajada atopik dermatitning epidemiologiyasi va davolash natijalari bo'yicha keng ko'lamli tadqiqotlar, shuningdek, genetik tadqiqotlar, ushbu kasallik uchun immunologik va chuqur molekulyar biokimyoviy tadqiqotlar tufayli erishildi.

Kalit so'zlar: atopik dermatit, epidemiologiya, demografiya

ЭПИДЕМИОЛОГИЧЕСКИЕ И ДЕМОГРАФИЧЕСКИЕ ВОПРОСЫ РАСПРОСТРАНЕННОСТИ АТОПИЧЕСКОГО ДЕРМАТИТА

Фатима Ирсалиева, Гульмира Разикова

Ташкент, Узбекистан

АБСТРАКТ

На протяжении последнего десятилетия достигнут значительный прогресс в понимании патогенетических механизмов развития и течения атопического дерматита. Эти знания в определенной степени были получены благодаря прорыву в масштабных исследованиях по вопросам эпидемиологии и результатов лечения атопического дерматита, а также проведенным генетическим, иммунологическим и глубоких молекулярно-биохимическим исследованиям при данном заболевании.

Ключевые слова: атопический дерматит, эпидемиология, демография