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**EVALUATION OF THE EFFICACY OF EXOGENOUS MELATONIN IN THE COMBINATION THERAPY OF PREMENSTRUAL DISORDERS**

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*The paper analyzes the effects of Melaxen® (Unipharm) at a dose of 1.5 mg taken orally once daily 30–40 minutes before bedtime for one month on the severity of manifestations of premenstrual symptoms and sleep disorders. It is shown that Melaxen® can be recommended to treat and correct premenstrual disorders.*

***Key words:*** *gynecology, melatonin, sleep disorders, menstrual cycle, premenstrual syndrome.*

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## from practical experience

**https://doi.org/10.29296/25877305-2019-03-06**

**DRY SKIN AS AN AUXILIARY DIAGNOSTIC CRITERION FOR ATOPIC DERMATITIS. EXPERIENCE IN EFFECTIVELY USING ECTOIN-CONTAINING EXTERNAL AGENT IN CHILDHOOD**

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Atopic dermatitis (AtD) is a chronic inflammatory skin disease, the basis for the pathogenesis of which is an incompetent skin barrier with an immune system imbalance. The skin forms mechanical and immune barriers that protect the body from aggressive environmental factors. Innate immunity imbalance leads to skin infection with opportunistic bacteria that maintain the local inflammatory process. Dry skin is now considered as an auxiliary criterion for AtD. Some experts are ready to include this factor for the progression of AtD in a set of its main criteria. The paper considers the experience with the new cream, the main component of which is the ectoin molecule at a concentration of 7%. There has been recently a noticeable increase in the number of works dealing with different therapeutic and prophylactic properties of ectoines, which are useful in medicine and cosmetology. Ectoin used as part of the agent to relieve inflammatory reactions in allergies and AtD has a certain positive effect.

Skin hydration control along with anti-inflammatory therapy can contribute to reducing the risk of more severe forms of the disease, by maintaining the achieved remission of AtD.

**Key words:** dermatology, atopic dermatitis, skin barrier, dry skin, skin moistening, ectoin.

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Atopic dermatitis (AD) is a chronic inflammatory skin disease, the development of which is mediated by both exogenous triggers comprising of various protein-based substances leading to body sensibilization, and endogenous factors, such as incompetent skin barrier with impaired protective properties due to innate immunity imbalance [1].

The endogenous factors predisposing to AD are heterogeneously inherited and realized under the influence of the environmental conditions. Presence of even several mutations meaningful for AD occurrence not always leads to a skin symptom [2]. The AD triggers may include both allergens and non-specific factors - ecological and physical ones; in recent years, the attention is increasingly called to potential participation of a psycho-emotional stress in the disease aggravation maintenance. It is well-known that AD is one of 8 psychosomatic illnesses [3].

Dysregulation of skin barrier formation mechanisms contributes to disruption of skin greasiness and water loss, the incompetence of the stratum corneum  protective properties - to transepidermal body sensibilization with respect to domestic and pollen allergens, and the inflammation focus generated - to colonization by potential pathogens. In 9 of 10 patients with AD, *Staphylococcus aureus* strains are isolated from the affected skin, which has a self-sufficient pathogenic meaning in this disease. It is well-known that staphylococcal enterotoxins being superantigens are capable of expanding the body sensibilization spectrum and maintain local inflammatory process [4]. Recent studies have found the direct relationship between the degree of skin colonization by *S. аureus* and AD progressing [5–7].

Healthy skin of a child can be considered as a multilevel system of interrelated physiological and immunological barriers, which retain moisture and prevent penetration of foreign substances and pathogenic microorganisms into the skin [7]. A damage of the stratum corneum can be a result of structural proteins deficiency, i.e. of filaggrin, involucrin, loricrin, and lipids. Reduced protective function of the skin associated with the imbalance of claudins, i.e. proteins closely adherent to each other, which are located on the reverse side of keratinocytes, exacerbates transepidermal water loss leading to expressed skin dryness. The proteins mentioned above form the second physiological barrier within the epidermis. The skin barrier incompetence due to structural proteins imbalance contributes to the immediate response of the innate and acquired immunity accompanied by antigen-presenting Toll-like receptors (TLR) expression on keratinocyte membranes. Stimulation of TLR with antimicrobial peptides release and an increase in protein contacts plays an important role as a factor preventing further penetration of foreign agents into the skin [8].

The skin barrier dysfunction causes Th2-inflammation [9]. Excessive surplus of Th2-cytokines in the skin suppresses the expression of the main antimicrobial peptide beta-defensin-3 (HβD-3) genes in keratinocytes [10].

Release of antimicrobial peptides by keratinocytes in AD appears to be insufficient to control infecting of the skin with *S. аureus* and viral replication [11]. A vicious circle occurs: the inflammatory process contributes to skin infecting, and *S. аureus* further disrupts the skin barrier functions exacerbating the AD course. Bacterial colonization of the skin in AD patients achieves high values: according to some authors, they are 55-90 versus 5 % in healthy people [12, 13]

*S. аureus* can worsen the skin barrier condition due to a release of large quantities of serine proteases. Bacterial colonization of the skin can increase pruritus; the IgE specific to staphylococcal toxins able to act like superantigens are found in peripheral blood of AD patients [14]. In the experiments on rats, staphylococcal enterotoxin B binding to TNF-receptor of monocytes inhibits the immunosuppressive activity of Treg-cells and stimulates proliferation of C-fibers with subsequent enhancement of neurogenic pruritus, and on healthy skin of healthy humans, it causes eczematous lesions [15].

Along with the hypothesis indicating that the skin barrier function impairment in presence of genetic mutations in its structural proteins is the main reason for the AD onset [16], there are convincing evidence that such pathologic process development includes the switch of the immune system response to the Th2-pathway, which is accompanied by suppressed differentiation and apoptosis of keratinocytes, leading to greater incompetence of the skin protective properties.

In line with the convincing evidence of the filaggrin gene mutation impact on the AD course, there are interesting facts about development of rather severe AD forms in absence of such mutation [17], and at the same time, revealing of the mutation in people without any skin diseases [18]. It is known that the majority of children having similar mutations "age out" of AD [19]. Additionally, unlike the pattern of the congenital genetic skin diseases, AD is associated with both affected and not affected skin areas. At the same time, a broad spectrum of various abnormalities of other structural proteins (loricrin, involucrin, claudins, etc.) is found on altered and not altered skin areas [20].

Also, it should be emphasized that the immune system activation lays behind the skin affection in AD. Chronic AD course is accompanied by enhanced (approximately 10 times greater than normal value) skin infiltration with T-lymphocytes and dendritic cells [21], increased production of cytokines and chemokines in the inflammation foci, epidermal hyperplasia with extremely high number of structural proteins in the terminal differentiation stage.

All these questions are open, and necessitate further search for the reasons for the AD onset and progressing.

AD is rather common condition [22]. Today, it attracts many investigators from different countries involved in great number of fundamental and clinical studies. Recently, new data on epigenetics and immunopathogenesis of the disease have appeared; simultaneously, the innovative treatment methods are being developed. The scientists from all over the world work on the problem of AD, and they summarize the accumulated knowledge and promote their ideas. Enrollment of any AD patient into the study aimed at investigation of the disease course peculiarities (should it be scientific development in genetics, pathogenesis, or contemporary treatment programmes) requires unambiguous diagnosing, which is the same for all patients worldwide.

Currently, no routine laboratory diagnostic procedure has been developed for this condition. Doctors and scientific researchers are guided by clinical criteria, which have been outlined with a purpose of unified diagnosing in various scientific and clinical centres. Most commonly (in 41.0 % of cases), the diagnostic criteria of Hanifin and Rajka are used; the English criteria are applied in 9.0 % of cases, the Japanese ones - in 4.2 % of cases, and the American ones - in 3.8 % of cases [23].

The criteria of Hanifin and Rajka can be divided into primary and auxiliary ones, and they should be taken into account while diagnosing AD [24]. Many investigators and doctors pay attention to the fact that some auxiliary criteria can be transferred to the primary ones, since their absence in the list of the primary criteria often leads to a mix-up and decreased quality of treatment for patients. The most debated aspects - assessment of the role of sensibilization in occurrence of AD and presence of expressed skin dryness.

In fact, relatively recent discoveries and studies in the sphere of immunology have clearly established a relationship between the skin damage typical for AD and the immune system imbalance accompanied by production of the allergen-specific IgE. For many years already, the scientists have been discussing the influence of sensibilization with respect to various allergens on the occurrence and progression of this disease [25]. At the same time, it has been found that presence of the expressed sensitivity to alimentary or domestic allergens in a patient more likely allows to diagnose atopic syndrome, and not AD. Currently, the statement "allergic inflammatory skin disease" has been deleted from the disease description, since such position leads to mandatory search for an exogenous trigger of inflammation in the patient resulting in prescription of non-justified restricting regimen [26].

According to the data of the recent studies, sensibilization in this condition is confirmed in less than 50 % cases, and only 30-40 % of patients report on alimentary allergy manifestations, commonly - in cases of moderately severe to severe AD with subsequent development of tolerance to many dietary proteins in older children [27]. Sometimes, in addition to 4 mandatory criteria of AD diagnosing (pruritus; typical morphology and localization of skin lesions; chronic continuously recurrent course, and hereditary burden), the criterion of totally dry skin in a patient is added [28]. In fact, the progressing AD course is accompanied by this symptom; the clinical recommendations in all countries include the instructions to the required use of moisturizing external agents able to prolong the remission within the baseline therapy.

As noted above, the criteria of Hanifin and Rajka are the most commonly used ones in clinical studies. During the period from 2007 till 2016, they were applied for pediatric and adult population in the majority of countries [23]. According to them, the AD diagnosis is confirmed if 3 out of 4 primary and 3 out of 19 auxiliary diagnostic criteria present [29].

Currently, there is no doubt that one of the auxiliary criteria of AD - skin dryness - should be controlled. Restoration of the skin barrier allows to reduce the risk of the next disease aggravation after successful topical anti-inflammatory treatment. Pharmaceutical market offers various cosmetic external agents recommended to control the skin condition in AD.

Currently, a new cream with 7 % content of ectoine molecule has appeared on the market - Perfectoin®. It may be combined with any medicinal product and used in combined treatment of AD, eczema, neurodermatitis, psoriasis, contact dermatitis, radiodermatitis, retinoid dermatitis, cheilitis, dry skin. It may be used in proactive therapy of AD due to its ability to stabilize and protect the skin barrier.

Numerous studies have confirmed the efficacy and safety of ectoine use in dermatoses. Additionally, the cream does not contain any potentially dangerous ingredients - preservatives, colourants, surface-active substances [30].

The main component of the cream is ectoine at 7 % concentration - a unique molecule of natural origin. Ectoine has been discovered by Swedish scientists, and is a unique substance produced by specific microorganisms for protection against detrimental environmental factors.

Ectoine generates strong bonds with water molecules forming a film on the skin surface - ectoine-hydro complex, which:

* + restores the skin barrier function;
  + retains moisture;

|  |  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- | --- |
| **Efficacy of the cream Perfectoin® use in AD children with cheilitis** | | | | | | | |
| **Disease** | **Age, years** | **Disease phase** | | **AD onset, years** | **Efficacy of external anti-inflammatory treatment, %** | **Efficacy of moisturizing with Perfectoin®, points** | **Manifestation of side effects, points** |
| **exacerbation** | **remission** |
| AD | 1.9±0.4 | n=14; 38% | n=21; 62% | 0.6±1.1 | 60 | 7.5±0.6 | 3.0±1.1 |
| AD + cheilitis | 7.6±2.7 | n=53; 3% | n=10; 67% | 0.9±0.3 | 73% | 7.2±0.8 | 2.8±1.5 |

* + prevents the effects of irritating factors and allergens;
  + reduces inflammation and pruritus.

Perfectoin® cream contains optimal lipids complex, which restores the optimal level of moisturizing by means of natural restoring of the water-lipid balance. The complex includes ceramides, squalane, natural oil lipids (olive and shea butter), Cardiospermum Halicacabum, triglycerides of capric and caprylic acids. Due to these components, the cream has a delicate texture, even a small amount is enough to apply over a large area of the skin surface.

We have analyzed the experience of Perfectoin® use as a cosmetic agent in children with AD to restore the skin barrier and control local inflammation around the lips. 50 children with mild and moderately severe AD have been examined with the SCORAD26 during the primary disease aggravation. In 35 children aged up to 3 years, the moisturizing agent was used to restore the skin barrier on the whole skin surface, in 15 older children with AD and cheilitis (see the table), Perfectoin® was applied on the skin around the lips. The treatment efficacy was evaluated after 2 weeks of the cream application by the patients' parents subjectively according to the online questionnaire using the following platform *https://docs.google.com/forms*.

|  |
| --- |
| 1st group 2nd group |
| Recommendations of the parents for the use of Perfectoin® cream;  1 – not recommended, 10 – recommended to all |

Perfectoin® cream was most commonly used during the AD remission period following the effective anti-inflammatory therapy. Secondary to acute AD aggravation, the moisturizing agent was applied around the lips in case of cheilitis aggravation and in combination topical therapy, which included the use of topical glucocorticosteroids (GCS) with low and medium biological activity (according to the European classification of potential activity of topical GCS, Miller & Munro). The moisturizing efficacy was evaluated based on a 10-scored system: 0 – no effect, 10 – good effect.

The parents of AD children, noted good tolerance of Perfectoin® cream with effective skin moisturizing effect (see the figure) both in case of isolated treatment (1st group) and in combination with cheilitis (2nd group) - 7.5 and 7.2 points, respectively. Such results confirm already existing data on the long-term skin moisturizing effects of ectoine [30]. When the cream was used secondary to the primary disease aggravation, sometimes discomfort was noted, such as burning and reddening. Primarily, the discomfort was observed only at the first application of the medicinal product, and subsequently, patients tolerated it well.

In 2 adolescents and 2 patients aged 2 years with cheilitis secondary to the AD aggravation, the use of Perfectoin® cream was discontinued due to the discomfort of the skin around the lips. In other children, the cream use was not accompanied by any complaints and was assessed as good both by the doctors and the patients (see the figure).

Thus, skin dryness associated with the skin barrier incompetence and transepidermal water loss occurring as a consequence, along with the skin greasiness reduction are the most common events accompanying the AD course and requiring constant monitoring with the help of moisturizing external agents. The use of Perfectoin® cream within the AD combination treatment allows to maintain normal physiological skin condition contributing to restoration of its protective properties and balanced microbiocenosis.

\* \* \*

*The authors declare no conflict of interest.*

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