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SCIENTIFIC AND PUBLISHING CENTER "PROGRESS"
MODERN RESEARCH AND EDUCATION
PROCEEDINGS OF THE INTERNATIONAL SCIENTIFIC AND PRACTICAL CONFERENCE
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THE EFFECT OF N-STEAROYLETHANOLAMINE ON RESOLVIN E1 CONCENTRATION AND PROINFLAMMATORY CYTOKINES IN A RAT MODEL OF IMIQUIMOD-INDUCED PSORIASIS-LIKE INFLAMMATION

Abstract. The article presents the results of experimental research concerning the effect of N-stearoylethanolamine (NSE) on the levels of pro-inflammatory mediators (IL-23 and IL-17) and resolvin E1 (RvE1) in a rat model of imiquimod-induced psoriasis-like inflammation. It was observed that NSE facilitates the

transition of the inflammatory response to the resolution phase by reducing the content of pro-inflammatory cytokines and normalizing RvE1 levels, which, in turn, creates the necessary conditions for recovery processes.

Keywords: n-acylethanolamine, endocannabinoids, inflammation, psoriasis, skin diseases

Introduction: Today, skin diseases represent one of the central problems in the medico-social sphere, affecting a large portion of the global population and significantly impacting the quality of life for most people. The epidemiological profile of skin diseases encompasses a wide range of clinical signs, etiologies, disease courses, and prevalence rates. However, a common feature for most of them is the active development of an inflammatory response, which serves as the foundation in the pathogenesis of very common dermatological conditions, including, in particular, psoriasis.

Psoriasis is a chronic inflammatory dermatosis induced by the excessive activity of sensitized T-lymphocytes, particularly CD4+ (Th17 and Th1) cells. A characteristic feature of psoriasis progression is the excessive infiltration of migrating Th17 and Th1 cells into the dermal and epidermal layers of the skin. In response to IL-23 produced by myeloid dendritic cells, these T-cells begin to synthesize a wide range of pro-inflammatory cytokines (IL-21, IL-22, IL-26, TNF-α, and INF-γ), specifically IL-17. These cytokines serve as the initiating factor for keratinocyte hyperproliferation, forming the specific morphological hallmarks of psoriatic inflammation known as the psoriatic triad: the stearic spot sign, the psoriatic film (terminal film), and the Auspitz's sign [1, 2, 3].

Psoriatic inflammation is characterized by a chronic course, specifically the inability to enter the resolution stage, which is essential for the transition from an acute inflammatory response to the recovery phase [4]. One of the key components involved in the resolution stage are lipid-derived mediators - resolvins, particularly RvE1, derived from ω-3 fatty acids (eicosapentaenoic and docosahexaenoic acids). Recent studies have established that RvE1 exerts a pronounced suppressive effect on

the inflammatory process in psoriasis by inhibiting the synthesis of IL-23 by dendritic cells [5].

Today, it is known that the endocannabinoid system, which includes N-acylethanolamines as its components, acts as a regulatory system in the processes of skin cell proliferation and differentiation, ensuring the maintenance of homeostasis, particularly the protective function of the dermal-epidermal barrier. It has been established that dysfunction of the endocannabinoid system is associated with various dermatological diseases based on the progression of an inflammatory response [6]. Previous studies have demonstrated a pronounced anti-inflammatory effect of NSE, specifically through the reduction of pro-inflammatory cytokine levels [7]. This, in turn, leads to a decrease in the manifestations of the inflammatory response and its transition to the resolution phase.

Aim: The aim of the study was to determine the effect of NSE on the development of the inflammatory response in a rat model of imiquimod-induced psoriasis-like skin inflammation.

Methods: The psoriasis-like inflammation model was established in male Wistar rats (n=30). The rats were divided into the following 6 groups: control, IMQ-7, IMQ-11, IMQ-15, NSE-11, and NSE-15). For 6 consecutive days, 125 mg of the immunomodulator imiquimod was applied to a pre-shaved area of the rats' skin. On the 7th day of the model induction, an aqueous suspension of NSE (at a dose of 1 mg/0.3 ml) was applied to the affected skin areas in the NSE-11 and NSE-15 groups. The animals were euthanized under CO2 anesthesia on the days corresponding to the group designations, and blood plasma was collected for further analysis. The concentrations of pro-inflammatory cytokines were determined using enzyme-linked immunosorbent assay (ELISA): IL-17 was measured using the Rat Interleukin 17 (IL17) ELISA Kit (MyBioSource, USA), and IL-23 was measured using the Rat Interleukin 23 (IL23) ELISA Kit (MyBioSource, USA). The blood levels of RvE1 were determined using the Rat Resolvin E1 (RvE1) ELISA Kit (MyBioSource, USA). Statistical analysis of the data obtained was performed using Student's t-test ($P < 0,05$).

Results: In the imiquimod-induced psoriasis-like inflammation model, a significant increase in the levels of pro-inflammatory cytokines IL-17 (on days 7 and 11) (Fig. 1) and IL-23 (on days 11 and 15) (Fig. 2) was observed.

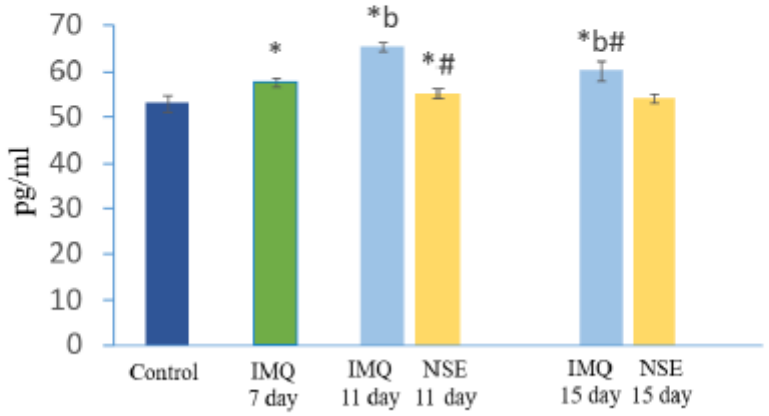

Fig. 1. The IL-17 content in the blood plasma of rats

* - P < 0,05 compared to control; b - P < 0,05 compared to IMQ-7; # - P < 0,05 compared to IMQ-11.

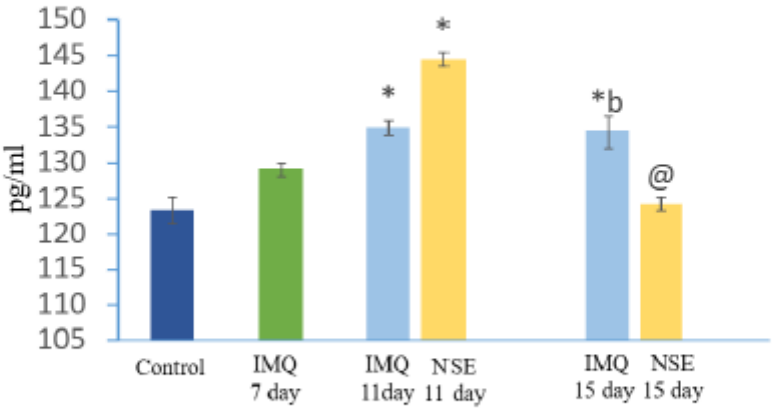

Fig. 2. The IL-23 content in the blood plasma of rats

* - P < 0,05 compared to control; b - P < 0,05 compared to IMQ-7; # - P < 0,05 compared to IMQ-11; @ - P < 0,05 compared to IMQ-15

Topical application of NSE to rats with induced psoriasis-like inflammation led to a reduction in IL-17 and IL-23 levels starting from the 11th day of application, returning to normal values by the 15th day of the experiment.

Under the influence of imiquimod, the concentration of resolvin remained at control levels on days 7 and 11, followed by a significant decrease on the 15th day of imiquimod exposure, indicating the transition of the inflammation into a chronic form. The administration of NSE led to a significant increase in RvE1 levels in the blood plasma of rats with imiquimod-induced psoriasis-like skin inflammation starting from the 11th day of the experiment, which indicates the initiation of the resolution stage of the inflammatory response (Fig. 3).

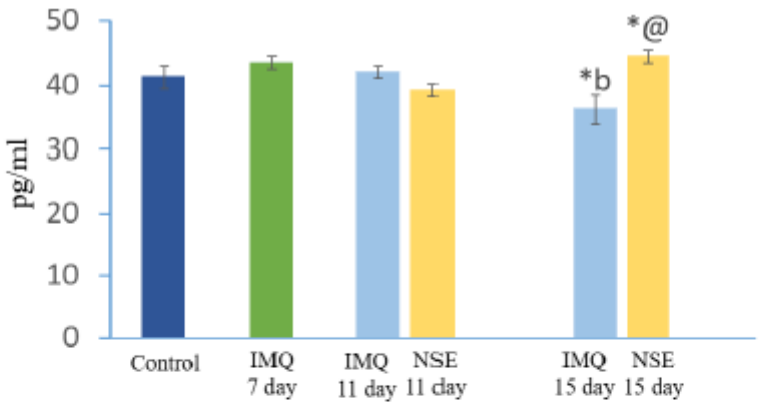

Fig. 3. The resolvin E1 content in the blood plasma of rats

* - P < 0,05 compared to control; b - P < 0,05 compared to IMQ-7; @ - P < 0,05 compared to IMQ-15

Discussion: Our results indicate that in a rat model of imiquimod-induced psoriasis-like inflammation, NSE exerts a pronounced anti-inflammatory effect by

reducing the concentrations of IL-17 and IL-23 and mediating an increase in RvE1, a key mediator of the resolution stage of the inflammatory response.

It is well known that the production of IL-17 by Th17 cells leads to keratinocyte hyperproliferation and the maintenance of chronic inflammation in the skin. In turn, IL-23 acts as a crucial regulatory factor in the process of Th17 cell differentiation, and its elevated levels are directly associated with the dynamic progression of psoriasis. The cooperation between IL-17 and IL-23 forms the central biochemical link in the pathogenetic profile of psoriasis and represents an attractive therapeutic target in modern medical practice [8]. The administration of NSE to rats with imiquimod-induced psoriatic inflammation promoted an effective reduction of the canonical cytokines IL-17 and IL-23, with complete normalization of their levels by the 15th day of the experiment. The results obtained indicate an inhibitory effect of NSE on the development of the inflammatory response in a rat model of imiquimod-induced psoriasis-like inflammation.

Current data indicate a pivotal role for RvE1 in suppressing the development of imiquimod-induced psoriasis in mice. It has been shown that RvE1 promoted a decrease in IL-23 mRNA expression in the skin, inhibited IL-23 production by dendritic cells, and suppressed the migration of dendritic and T-cell populations in mouse models. The observed suppressive effect of RvE1 is linked to its antagonistic interaction with the leukotriene B4 receptor, BLT1 [5]. These results suggest the existence of a biochemical mechanism that could potentially be considered a therapeutic target for the treatment of psoriasis. As a result of our studies, it was shown that the administration of NSE promoted an increase in RvE1 levels, thereby mediating the activation of the resolution stage of the inflammatory response.

Conclusion: The analysis of our data indicates that the topical application of an aqueous NSE suspension to rats with imiquimod-induced psoriatic inflammation leads to a decrease in the levels of IL-17 and IL-23, the primary marker cytokines of psoriasis, by the 11th day of exposure, reaching normal values by the final 15th day. Furthermore, it was established that NSE treatment results in a significant increase in RvE1 concentration in the blood plasma of the model rats on the 11th day of the

experiment, providing evidence of the activation of the resolution stage of the inflammatory response.

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