АКТИВНОСТЬ ЯДЕРНОГО ФАКТОРА ТРАНСКРИПЦИИ вB В ПОПУЛЯЦИЯХ ЛИМФОЦИТОВ У ДЕТЕЙ С ПСОРИАЗОМ

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Изменения в путях передачи внутриклеточных сигналов, влияющих на активацию иммунных клеток, пролиферацию и дифференцировку кератиноцитов при псориазе, могут объяснить сложный патогенез заболевания. Одним из путей передачи внутриклеточных сигналов является NF-кB, участвующий в регуляции большого количества провоспалительных генов и влияющий на продукцию провоспалительных цитокинов, непосредственно участвующих в развитии псориаза. Целью нашего исследования было оценить число клеток с транслокацией NF-кB в популяциях лимфоцитов у детей с псориазом в зависимости от тяжести заболевания и проводимой терапии. Обследовано 130 детей с вульгарным псориазом. В группу сравнения вошли 30 здоровых детей. Исследование проводили методом проточной цитометрии с визуализацией Amnis ImageStreamX. Показано, что число клеток, в которых происходит транслокация NF-кB, достоверно различается в популяциях лимфоцитов у детей с псориазом, так и в группе сравнения. У детей с псориазом в 2-4 раза повышается число клеток с транслокацией NF-кB в популяции Т-хелперов, Трег и Th17 по сравнению со здоровыми детьми (p < 0,05). Число клеток с транслокацией NF-кB у детей с псориазом соотносится с тяжестью состояния по PASI (R = 0,32), BSA (R = 0,31) и длительностью заболевания (p < 0,05). Определение NF-кB может быть рассмотрено как дополнительный критерий оценки тяжести состояния у детей с псориазом. Показано, что значительно снижается число клеток с транслокацией NF-кB через сутки после введения биологической терапии (adalimumab, etanercept, ustekinumab) и наблюдается усиление эффекта через 2 нед.

Ключевые слова: дети, вульгарный псориаз, PASI, BSA, лимфоциты, NF-кB, биологические препараты


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Psoriasis is a multifactorial immune-mediated inflammatory skin disorder with a complex pathogenesis [1–3], caused by interactions of immune system, psoriasis susceptibility loci, psoriasis autoantigens, and numerous environmental factors [4]. Psoriasis has various cutaneous clinical manifestations, based on which a number of disease forms is distinguished. Psoriasis vulgaris, being the most common form, is diagnosed in 90% of cases [5, 6]. Psoriasis is characterized by hyperproliferation of keratinocytes, blood vessel dilation and leukocyte cell infiltration in skin dermis, and skin plaques [7].

It is believed that synthesis of pro-inflammatory cytokines, being the pathogenic cascade triggers, by activated T cells plays a key role in the skin inflammation associated with psoriasis [8]. Disease begins with T cell activation by unknown antigen or gene product. After the T cell activation, a number of pro-inflammatory cytokines and chemokines are secreted [9]. Cytokine action contributes to proliferation of keratinocytes, epidermal hyperplasia, neutrophil migration, enhanced T helper type 1 (Th1) cell response [10]. The increase in the number of T helper 17 cells (Th17) in blood and skin of patients with psoriasis with the progression of skin lesions was reported [11–13]. It was also found that elevated blood counts of regulatory T cells (Treg) in patients with psoriasis did not suppress inflammatory response due to the cells’ functional failure [14, 15].

Complex pathogenesis of psoriasis could be explained by alterations in the intracellular signaling pathways. Impaired regulation of the signal transduction pathways affects activation, proliferation and differentiation of keratinocytes, associated with psoriasis [16, 17]. The nuclear factor NFKB (NF-κB) pathway, which has been first discovered in mature B cells by R. Sen and D. Baltimore, is one of the intracellular signaling pathways [18]. NF-κB is capable of binding with the gene promoter encoding the immunoglobulin — light chain. NF-κB is a family of structurally similar transcription factors, such as RelA (p65), NF-κB1 (p50/p105), NF-κB2 (p52/p100), c-Rel, and RelB. The NF-κB family members function as various hetero- and homodimers (p50–p55; RelB-p100), except for RelB. The main active form of NF-κB is a complex formed by subunit p65 and subunit p50 or p52 bound to the I-κB inhibitor protein [19]. The advanced Amnis ImageStreamX imaging flow cytometry system makes it possible to assess the percentage of cells with NF-κB translocation (% of activated cells with nuclear localization of NF-κB) in various populations [20, 21].

Under physiological conditions, the NF-κB/I-κB complex is a self-regulating system [22]. NF-κB activation occurs in response to the wide range of stimuli [23]. Stimulus activates the NF-κB signaling pathway, which leads to the NF-κB release from the inhibitor complex and NF-κB translocation from cytoplasm into cell nucleus. NF-κB is involved in regulation of numerous pro-inflammatory genes and affects synthesis of pro-inflammatory cytokines, such as IL-1β, IL-6, IL-8, TNFα, directly involved in the development of psoriasis [24–26]. Multiple studies confirmed impaired regulation of the NF-κB signaling pathways associated with psoriasis in vitro models, animal models, and skin biopsy samples, obtained from patients with psoriasis [10, 25, 26]. Furthermore, anti-TNF therapy and glucocorticoid therapy reduce the levels of active NF-κB and related elements of the signaling pathway [27, 30]. Currently, the effects of other targeted biologics on the variation in the number of cells with NF-κB translocation are under study, however, chronic NF-κB pathway inhibition may reduce therapeutic efficacy in patients with immune-mediated diseases [28]. Thus, quantification of cells with NF-κB translocation could be a promising tool for evaluation of the disease severity and therapeutic efficacy in patients with immune-mediated diseases. The study was aimed to quantify cells with NF-κB translocation in the lymphocyte populations of children with psoriasis depending on the disease severity and therapy.

METHODS

Clinical characteristics of patients

A total of 130 children and adolescents with psoriasis vulgaris were enrolled in the study, of them 42 patients were followed-up. The comparison group included 30 healthy children. The surveyed children’s age was 1–18 years: psoriasis — Me 12.5 [8.3; 15.5], comparison group — Me 12.4 [7.4; 16.1]. There were 62 girls and 68 boys in the group with psoriasis, and 18 girls and 12 boys in the comparison group. All children were of European origin. Inclusion criteria: proven case of psoriasis vulgaris. Exclusion criteria: other forms of psoriasis, 18 years of age or more, inability to obtain blood sample.

Severity of psoriasis was assessed in all patients using the PASI (Psoriasis Area and Severity Index) and BSA (Body Surface Area, %) indices. Children with psoriasis received different pathogenetic therapy in accordance with the guidelines and psoriasis severity. Children were prescribed topical corticosteroids and emollients with keratolytic effects (n = 41), systemic methotrexate therapy (n = 28), genetically-engineered biological drugs (GEBD; n = 61). The effects of GEBD were assessed in 42 patients with psoriasis before and 24 hours after administration of the following drugs: adaлимумаб (n = 24), etanercept (n = 8), устекинумаб (n = 10).

Isolation of peripheral blood mononuclear cells

To isolate the peripheral blood mononuclear cells (PBMCs), the 5 mL fasting whole blood samples were used, collected in the morning into EDTA tubes. Blood samples were processed on the same day. Whole blood was diluted with warm glutamine-free RPMI 1640 medium (PanEco; Russia) in a ratio of 1:3, gently layered over 2 mL of the Histopaque-1.077 g/cm3 medium (Sigma-Aldrich; USA), then centrifuged at 2000 RPM for 20 min at room temperature. Buffy coat was collected into 15 mm Falcon tubes (BD; USA) and washed with RPMI medium for 8 min under the same conditions. After that supernatant was drained, and the cells were diluted with the RPMI-1640 medium to the desired volume.

Lymphocyte immunophenotyping and evaluation of NF-κB translocation from cytoplasm into nucleus

Cell populations were isolated by step-wise gating using monoclonal antibodies (mAbs) labeled with fluorochromes (Beckman Coulter; USA): CD19-PE, CD4-PE, CD8-PE, CD16-PE, CD19-PE, CD127-PE, CD25-PE, CD29-PE, CD45RA-PE, CD4-PE, CD4-PE, CD45RA-PE, CD3-PE, CD25 PE-Cy7. Cells with NF-κB translocation were quantified in lymphocyte populations with the imaging flow cytometry Amnis ImageStreamX Mk II platform (Luminex; USA) using the Amnis NF-κB Translocation Kit (Luminex; USA), which included antibody Anti-Hu NF-κB (p50) conjugated to Alexa Fluor 488 for detection of NF-κB and the 7-AAD nuclear dye. The tubes, containing 100 μL of PBMCs, were added 10 μL of mAb and incubated for 20 min in a dark place, then NF-κB translocation was assessed in accordance with the manufacturer’s instructions. The following cell populations were assessed: CD3+ (T cells); CD3+CD4+ (T helper cells); CD3+CD8+ (cytotoxic T cells); CD3+CD19+ (B cells); CD3+CD16+/CD56+ (natural killer cells).
Fig. 1. Stages of analysis when quantifying cells with NF-κB translocation. A. Gallery of recorded cell images. B. Isolation of cells in good focus based on Gradient RMS. C. Isolation of single events. D. Isolation of the studied lymphocyte population, particularly CD3+CD4–CD8– (immature T cells); CD4+CD25+CD127high (activated T helper cells, T_{act}); CD4+CD25+CD127low (T_{reg}); CD4+CD16+CD3+ (Th17). E. Isolation of double-positive cells: NF-κB+/7-AAD+ (NF-κB_FITC — channel 2; 7-AAD — channel 5). F. Isolation of the proportion of cells with NF-κB translocation based on Similarity>1 regardless of morphology (Fig. 1F). Similarity was calculated based on the Pearson correlation coefficient as described earlier [20, 21].

**Table 1.** Number of cells with NF-κB translocation in children with psoriasis and comparison group

<table>
<thead>
<tr>
<th>Population</th>
<th>Patients with psoriasis (n = 130)</th>
<th>Healthy children (n = 30)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>B cells</td>
<td>48.3 [38;64]</td>
<td>58.4 [40;72]</td>
<td>0.322</td>
</tr>
<tr>
<td>T cells</td>
<td>19.9 [17;26]</td>
<td>18.0 [16;21]</td>
<td>0.113</td>
</tr>
<tr>
<td>T helper cells</td>
<td>20.1 [17;26]</td>
<td>18.4 [14;20]</td>
<td>0.001</td>
</tr>
<tr>
<td>Cytotoxic T cells</td>
<td>18.0 [15;24]</td>
<td>17.2 [15;23]</td>
<td>0.402</td>
</tr>
<tr>
<td>Immature T cells</td>
<td>24.4 [18;36]</td>
<td>23.7 [19;33]</td>
<td>0.473</td>
</tr>
<tr>
<td>NK cells</td>
<td>28.0 [19;39]</td>
<td>29.9 [18;42]</td>
<td>0.829</td>
</tr>
<tr>
<td>NKT cells</td>
<td>22.3 [16;34]</td>
<td>20.1 [17;25]</td>
<td>0.315</td>
</tr>
<tr>
<td>Activated T helper cells</td>
<td>18.7 [15;24]</td>
<td>15.2 [14;18]</td>
<td>0.012</td>
</tr>
<tr>
<td>Regulatory T cells</td>
<td>23.0 [19;32]</td>
<td>20.3 [16;26]</td>
<td>0.032</td>
</tr>
<tr>
<td>T helper 17 cells</td>
<td>20.7 [17;27]</td>
<td>19.6 [17;23]</td>
<td>0.034</td>
</tr>
</tbody>
</table>

Note: p — significance of differences between groups, Mann–Whitney U test.
Fig. 2. Number of major (A) and minor (B) populations of lymphocytes and NK cells with NF-κB translocation in children with psoriasis and comparison group. *p* — significance of differences between populations, Wilcoxon signed rank test for dependent variables.

Analysis revealed differences in the percentage of cells with NF-κB translocation in the studied cell populations of surveyed children. The number of cells with NF-κB translocation was significantly (*p* = 0.000) higher in B cell population compared to NK cells, T helper cells, cytotoxic T cells, and in the NK cell population it was significantly (*p* = 0.000) higher compared to the populations of T helper cells and cytotoxic T cells in both children with psoriasis and comparison group; moreover, it was 2.5 times higher compared to other cell populations (Table 1; Fig. 2A).

### Table 2. Correlations of the number of cells with NF-κB translocation in lymphocyte populations with the patients’ age and the duration of psoriasis

<table>
<thead>
<tr>
<th>Population</th>
<th>Psoriasis (n = 130)</th>
<th>Comparison group (n = 30)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Age</td>
<td>r</td>
</tr>
<tr>
<td>B cells</td>
<td>0.09</td>
<td>0.299</td>
</tr>
<tr>
<td>T cells</td>
<td>0.21</td>
<td>0.016</td>
</tr>
<tr>
<td>T helper cells</td>
<td>0.22</td>
<td>0.011</td>
</tr>
<tr>
<td>Cytotoxic T cells</td>
<td>0.19</td>
<td>0.031</td>
</tr>
<tr>
<td>Immature T cells</td>
<td>0.2</td>
<td>0.028</td>
</tr>
<tr>
<td>NK cells</td>
<td>0.24</td>
<td>0.006</td>
</tr>
<tr>
<td>NKT cells</td>
<td>0.16</td>
<td>0.071</td>
</tr>
<tr>
<td>Activated T helper cells</td>
<td>0.21</td>
<td>0.023</td>
</tr>
<tr>
<td>Regulatory T cells</td>
<td>0.2</td>
<td>0.031</td>
</tr>
<tr>
<td>T helper 17 cells</td>
<td>0.23</td>
<td>0.015</td>
</tr>
</tbody>
</table>

Note: *r* — Pearson correlation coefficient, *p* — probability of non-zero regression coefficients.
of healthy children, the number of cells with NF-κB translocation in the populations of T helper cells and cytotoxic T cells was the same \((p = 0.101)\). In contrast to healthy children, the increased percentage of cells with NF-κB translocation in the population of T helper cells compared to cytotoxic T cells was revealed in patients with psoriasis \((p = 0.000; \text{Fig. 2A})\).

In the group of healthy children, it was found that the average number of cells with NF-κB translocation in T cell populations, including T helper, Th17, and T cell, was 15\%\%20\%. The largest proportion of cells with NF-κB translocation was found in the T helper population (20\% of cells), and the lowest proportion was observed in the T cell population (15\% of cells; \(p = 0.011\)), the difference was considered significant \((p = 0.002; \text{Fig. 2B})\).

In children with psoriasis, the number of cells with NF-κB translocation in the T cell, Th17, and T cell populations was significantly higher compared to the comparison group (Table 1). In healthy children, the highest percentage of cells with NF-κB translocation was also found in the T cell population (23\% of cells), and was significantly higher than in the populations of T helper cells, Th17, and T cell \((p = 0.000; \text{Fig. 2B})\). The number of T cell with NF-κB translocation was significantly lower compared to the T helper cell population, just like in the group of healthy children \((p = 0.000; \text{Fig. 2B})\). In contrast to healthy children, the number of Th17 with NF-κB translocation was significantly higher compared to the T cell population \((p = 0.001)\).

<table>
<thead>
<tr>
<th>Population</th>
<th>Number of cells with NF-κB translocation before</th>
<th>Number of cells with NF-κB translocation after</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adalimumab ((n = 24))</td>
<td>before</td>
<td>after</td>
</tr>
<tr>
<td>T cells</td>
<td>40.3 [30; 54]</td>
<td>32.0 [25; 37]</td>
</tr>
<tr>
<td>T helper cells</td>
<td>20.0 [17; 25]</td>
<td>19.5 [15; 21]</td>
</tr>
<tr>
<td>Cytotoxic T cells</td>
<td>20.2 [18; 24]</td>
<td>18.3 [16; 21]</td>
</tr>
<tr>
<td>Immature T cells</td>
<td>22.5 [16; 31]</td>
<td>21.2 [20; 38]</td>
</tr>
<tr>
<td>NK cells</td>
<td>27.7 [18; 32]</td>
<td>26.2 [19; 29]</td>
</tr>
<tr>
<td>NKT cells</td>
<td>21.6 [15; 27]</td>
<td>19.7 [15; 24]</td>
</tr>
<tr>
<td>Activated T helper cells</td>
<td>19.1 [16; 21]</td>
<td>17.1 [14; 20]</td>
</tr>
<tr>
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</tr>
<tr>
<td>T helper 17 cells</td>
<td>22.4 [18; 23]</td>
<td>18.5 [16; 21]</td>
</tr>
</tbody>
</table>

Note: \(p\) — significance of differences in the numbers of cells with NF-κB translocation obtained before and 24 h after administration of GEBD, Mann–Whitney U test.
Correlations of the number of cells with NF-κB translocation with the patients’ age and the duration of psoriasis

Analysis of the relationship between the number of cells with NF-κB translocation and the healthy children’s age revealed no correlations in all cell populations, except for T helper 17 cells, the number of which increased with age (Table 2). In children with psoriasis, the proportion of cells with NF-κB translocation increased with age in all populations, except for B cells and NKT cells. With increased disease duration, the number of cells with NF-κB translocation significantly increased in the populations of T helper cells, cytotoxic T cells, immature T cells, NK cells and Th17 (Table 2). In children with psoriasis, correlation between the patient’s age and the disease duration was revealed (p = 0.000): the older the child, the longer he was ill.

Relationship between the content of cells with NF-κB translocation and psoriasis severity in children

PASI and BSA indices were evaluated in the surveyed children with psoriasis. The psoriasis severity PASI scores in children varied between 0–70% (Me 13.3 [5; 22]), and BSA scores varied between 0–100% (Me 20 [4; 40]).

Multiple correlation analysis of the relationship between the psoriasis severity and the number of cells with NF-κB translocation showed the increase in the PASI score with the decrease in the number of Treg and NK cells with NF-κB translocation and the increase in the proportion of immature T cells with translocation (Rreg = 0.32; Fig. 3A). The increased BSA scores are associated with the decreased number of cells with NF-κB translocation in the Treg population and increased number in B cell population (Rint = 0.31; Fig. 3B).

Assessing the number of cells with NF-κB translocation in children with psoriasis receiving various types of therapy

Comparison of the proportion of cells with NF-κB translocation in lymphocyte populations of children with psoriasis receiving various types of therapy revealed no significant differences between groups of children receiving different therapy. However, maximum deviation from healthy children was observed in the group of children, who received biological therapy. The number of cells with NF-κB translocation in the T helper cell populations of children, who received topical therapy, increased by 6%, in the group of children, who received methotrexate, it increased by 10%, and in the group of children, who received biologics, by 11.5% compared to healthy children. The number of cells with NF-κB translocation in B cell population decreased by 17% in patients, who received topical therapy, by 4% in patients, who received methotrexate, and by 29% in children, who received biologics, compared to healthy children.

To assess the effects of GEBD on the number of cells with NF-κB translocation, we examined 42 children with psoriasis before and 24 h after administration of adalimumab, etanercept, ustekinumab. A significantly decreased number of cells with NF-κB translocation in the populations of B cells, T helper cells, cytotoxic T cells, Tact, and Th17 was observed 24 h after administration of adalimumab (Table 3).

The significantly decreased number of cells with NF-κB translocation was revealed in the populations of B cells, T helper cells, Tact and Th17 after administration of etanercept. The significantly decreased number of NF-κB cells was observed in the populations of NK cells, immature T cells, Tact, Treg and Th17 after administration of ustekinumab (Table 3).

It is interesting to note that biologics, adalimumab and etanercept, significantly decreased the number of B cells with NF-κB translocation after 24 h: by 20.4 and 31%, respectively. However, administration of ustekinumab resulted in the insignificantly decreased number (Table 3). The same downward trend was observed in the T helper cell population after administration of adalimumab (by 10.4%) and etanercept (by 8.6%). Significant alterations in the number of cytotoxic lymphocytes with NF-κB translocation were observed after administration of adalimumab (Table 3).

The number of Tact with NF-κB translocation significantly decreased 24 h after administration of etanercept (by 37.8%) and ustekinumab (by 48.7%) (Table 3). Only the administration of ustekinumab resulted in the decrease in the number of Treg with NF-κB translocation by 62%, however, administration of adalimumab resulted in the significant decrease in their number by 6%. Administration of all biologics resulted in the decreased number of Th17 with NF-κB translocation: the maximum decrease was observed after administration of ustekinumab (by 42.9%), and administration of adalimumab and etanercept resulted in the decrease by 21.1 and 8%, respectively (Table 3).

Furthermore, only administration of ustekinumab decreased the number of cells with NF-κB translocation in the population of NK cells by 49.8% and in the population of immature T cells by 69%.

DISCUSSION

Alterations in signaling pathways, such as NF-κB, JAK-STAT, Janus kinase (JAK)/signal transducer, Akt and Wnt signaling pathways, are observed in individuals with psoriasis [16, 17]. The number of cells with NF-κB translocation in children with psoriasis was assessed using the ImageStreamX advanced technique. Our findings about the number of cells with NF-κB translocation are in line with the data reported by other researchers: the maximum number of cells with NF-κB translocation has been revealed in B cell population [18].

Our study has shown that in children with psoriasis, the number of cells with NF-κB translocation in the populations of T helper cells, Treg, Tact and Th17 is increased compared to healthy children. That could explain the effects of the NF-κB signaling pathway activation in these cells, which are responsible for production of pro-inflammatory cytokines directly involved in pathogenesis of psoriasis [24–26].

There was a direct correlation between the number of cells with NF-κB translocation and the children’s age in children with psoriasis; the proportion of such cells in healthy children did not change with age. Probably, our findings about the relationship between the number of cells with NF-κB translocation and age in children with psoriasis are attributable to the correlation between the duration of psoriasis and the surveyed children’s age.

The number of Treg with NF-κB translocation decrease with the increase in the psoriasis severity in children according to PASI and BSA, which is in line with the other researchers’ report of the decreased Treg functional activity in psoriasis [27]. This fact confirms the importance of assessing both Treg number and functional activity [14, 15]. As one of the factors involved in the cell functional activity regulation, NF-κB determination could be considered an additional criterion for the disease severity evaluation in patients with psoriasis.

Our findings are in line with the data on the NF-κB activation reduction by anti-TNF therapy (adalimumab) in adults with psoriasis [28–30]. The maximum decrease in the number of cells with NF-κB translocation in the populations of NK cells, immature T cells, Tact, Treg and Th17 of children with
psoriasis was detected when using ustekinumab. The use of TNFα and IL12/23 blockers reduces the number of cells with NF-κB translocation to varying degrees, depending on the cell population and therapy applied. Taking into account the findings and the minimally invasive method of assessing the number of cells with NF-κB translocation in the whole blood of children with psoriasis, we consider this line of research to be perspective for diagnosis of the disease severity and predicting therapeutic efficacy.

CONCLUSIONS

Significant differences between lymphocyte populations by the number of cells with NF-κB translocation were revealed in both children with psoriasis and comparison group. The increased number of cells with NF-κB translocation in the populations of T helper cells, T_{cyt}, T_{reg}, and Th17 in children with psoriasis compared to healthy children was observed. The number of cells with NF-κB translocation correlates with the disease severity and duration. NF-κB determination could be considered an additional criterion for evaluation of the disease severity in children with psoriasis. The number of cells with NF-κB translocation in lymphocyte populations decreases 24 h after administration of GEBD and depends on the antipsoriatic therapy targets. Studying NF-κB activation in cell populations offers the prospect of understanding pathogenetic mechanisms of inflammation and developing new treatment methods for psoriasis.

References


