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ОРИГИНАЛЬНОЕ ИССЛЕДОВАНИЕ
ORIGINAL RESEARCH

Therapeutic activity of proinflammatory macrophages in endometriosis is driven by their antiproliferative and proapoptotic action

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Abstract: *Relevance.* Despite longstanding recognition of endometriosis, there's an absence of effective treatment. Existing treatments carry significant risk, so research into cell therapy is gaining popularity. Macrophages are a promising agent. A previous study using an animal model demonstrated that introducing proinflammatory macrophages led to a significant reduction in endometriosis lesions. *The aim of the study* is to analyze the effect of macrophages with a proinflammatory phenotype introduced into an animal model on the proliferation and survival of cells in endometriosis lesions. *Materials and Methods.* A syngeneic model of endometriosis was obtained in female C57Bl/6 mice by intraperitoneal transplantation of uterine fragments from a donor mouse. RAW264.7 mouse macrophages were utilized as the cellular agent. The animals were then divided into groups: "Control" comprised mice that did not receive macrophage therapy; "Control of therapy" comprised mice that received unpolarized RAW264.7 macrophages; and "Therapy" comprised mice with endometriosis that were injected with RAW264.7 macrophages with a proinflammatory phenotype. *Results and Discussion.* Consequently, the administration of macrophages with a proinflammatory phenotype resulted in a significant decrease in the production of the proliferation marker protein Ki-67 and a significant increase in the production of effector caspase 3 in the cells of endometriosis lesions compared to the "Control" group. Concurrently, the level of production of the tumor suppressor protein p53, which is involved in the initiation of apoptosis in cells, was comparable to that in the "Control" group. This is in contrast to the group of animals that received unpolarized macrophages. *Conclusion.* We found that the anti-endometriosis activity of macrophages with a proinflammatory phenotype is associated with the fact that their introduction suppressed the proliferation of endometriosis cells and enhanced their apoptotic death through the activation of p53 and caspase 3.

Keywords: endometriosis, mouse model, cell therapy, macrophages

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Introduction

Endometriosis, a serious disease affecting 10% of women of reproductive age, is characterized by pelvic pain and infertility, which can have a significant impact on the quality of life of patients [1, 2]. The gold standard in the treatment of endometriosis remains laparoscopic removal of lesions, but this surgical procedure is associated with risks of damage to healthy tissues and organs [3]. Furthermore, research has demonstrated that the frequency of repeat operations following surgical removal of lesions ranges from 27% to 58%, as evidenced by various data sources [4, 5]. In addition to laparoscopic removal of lesions, the administration of pharmaceuticals is also employed to alleviate pain symptoms [1]. Moreover, there have been documented cases in which the administration of drug therapy within six days following surgical excision of lesions has been shown to reduce the risk of recurrence [4]. However, the efficacy of pharmaceutical interventions in managing the disease’s symptoms is questionable, as is their potential to induce adverse effects. These include, but are not limited to, irregular bleeding, nausea, cardiovascular complications, weight gain, and the potential for contraceptive effects [1]. In this regard, a relevant area of research is the development of minimally invasive therapies using cellular agents.

In a preceding study, an investigation was conducted on a syngeneic mouse model of endometriosis, in which it was demonstrated that the introduction of allogeneic macrophages with a proinflammatory phenotype into the animal model resulted in a significant reduction in the size and number of lesions [3].

The objective of the present study was to ascertain the mechanisms underlying this effect, specifically to evaluate the impact of introducing macrophages with

a proinflammatory phenotype on the survival of cells in endometriosis lesions and their proliferation.

Materials and methods

Syngeneic model of endometriosis

Thirty female mice of the C57Bl/6 strain, aged 4–8 weeks and weighing 18–20 g, were obtained from the Andreevka Branch of the National Center for Biomedical Technologies (NCBMT) of the Federal Medical Biological Agency (FMBA) of Russia. The animals were maintained under standard conditions, with unrestricted access to water and food for a total of 12 hours per day/night. Two weeks after acclimatization, the animals were included in the experiment. A syngeneic model of endometriosis in mice was obtained by intraperitoneal transplantation of uterine fragments using a method like that described in our previous work [3].

Therapy of animals with proinflammatory macrophages

The RAW264.7 mouse macrophage cell line (Cat. No. 186, BioCollection, Moscow, Russia) was cultured in complete RPMI-1640 medium (PanEco, Russia) with the addition of 10% fetal bovine serum (FBS) (Biowest, Brazil), L-glutamine (PanEco, Russia), and penicillin-streptomycin (PanEco, Russia) at +37 °C and 5% CO2 in 175 cm2 culture flasks (Corning Inc., USA) until a 100% confluent monolayer was achieved.

Then, to polarize macrophages toward a proinflammatory phenotype, according to the protocol presented in our previous study [3], the cells were incubated with lipopolysaccharide (LPS) isolated from Escherichia coli O111: B4 (Sigma-Aldrich, USA) (100 ng/ml) for 24 hours. Unpolarized macrophages were used as a control for the therapy. Three weeks after transplantation of uterine fragments, the animals were divided into three groups: "Control" — animals with endometriosis that did not receive macrophage therapy; "Therapy control" — animals with endometriosis that received unpolarized macrophages; "Therapy" — animals with endometriosis that were injected with polarized macrophages with a proinflammatory phenotype. After the cells were injected, the animals were removed from the experiment by overdosing them with Zoletil 100 (Virbac, France) and Xylanit (Nita-Farm, Russia) two weeks later. The study was approved by the local ethics committee of the Petrovsky National Research Center for Surgery, protocol No. 3 of March 23, 2023.

Western blot analysis

Following the removal of the animals from the experiment, the endometriosis lesions were extracted and lysed in Protein Solubilization Buffer (PSB) (Bio-Rad Laboratories, USA), which was supplemented with Complete™ Protease Inhibitor Cocktail (Roche, Switzerland), with the objective of obtaining a protein fraction. The protocol for the western blot analysis was executed in accordance with the method outlined in a previously published study [6]. The following primary antibodies were used in the staining procedure: mouse anti-GAPDH (cat. no. 5G4, clone 4G5, HyTest, Russia) (1/5000), rabbit anti-Ki-67 (ab16667, Abcam, UK) (1/1000), and mouse anti-p53 (ab154036, Abcam, UK) (1/1000). Secondary antibodies were used for detection: goat anti-rabbit IgG H&L (HRP) (ab6721, Abcam, UK) (1/5000), goat anti-mouse IgG H&L (HRP) (ab6789, Abcam, UK) (1/2000). The membranes were developed using the NovexTM ECL HRP Chemiluminescent Substrate Reagent Kit (Invitrogen, USA). The signal was detected on a Fusion FX system (Vilber Lourmat, France) using Fusion Software. The relative protein content of the sample was assessed by normalization to the signal of the control protein GAPDH.

Immunohistochemical (IHC) analysis

The extracted endometriosis lesions were preserved in liquid nitrogen, then cryosections were prepared and transferred to Superfrost Plus Microscope Slides (Thermo Fisher Scientific, USA). Prior to staining, the sections were washed with phosphate-buffered saline (PBS) (Panecho, Russia). The sections were then subjected to an overnight incubation with antibodies directed against caspase 3 (AF6311, Affinity Biosciences, China) at a concentration of 1:100. Sub-

sequently, the sections were subjected to a thorough washing process with phosphate-buffered saline (PBS). Thereafter, they were placed into an incubator with secondary antibodies that were rabbit-conjugated with Alexa Fluor 555 (AS058, ABclonal, Inc., China) at a concentration of 1/200 for a duration of one hour at ambient temperature. Subsequently, the nuclei were stained with DAPI (0.1 μg/ml). A quantitative analysis of the content of positively stained cells was performed using QuPath software, developed in the United Kingdom [7].

Statistical analysis

The statistical data processing was performed using GraphPad Prism 8 software (GraphPad Software, USA). The normality of distribution was assessed using the Shapiro-Wilk test. In the case of normal distribution, multiple comparisons of samples were performed using one-way ANOVA with Tukey's post-hoc test. In instances where the distribution was deemed to be non-normal, the non-parametric Kruskal-Wallis test was employed, followed by the Dunn's post-hoc test to identify significant differences. The analysis was conducted using two-tailed tests and a 95% confidence interval. Statistical significance was defined as $p < 0.05$.

Results and discussion

Proinflammatory macrophages induced a decrease in cell proliferation in endometriosis lesions and supported p53 protein production in them

In established endometriosis lesions following therapy with proinflammatory macrophages, a significant decrease ($p < 0.05$) in the production of the Ki-67 proliferation marker protein was observed compared to control lesions extracted from animals not exposed to therapy (Figure 1 A, B). A parallel decline in protein production was observed in nonpolarized macrophages upon administration.

Furthermore, an assessment was conducted to determine the level of production of the stress-activated protein p53. The investigation revealed that the introduction of macrophages with a proinflammatory

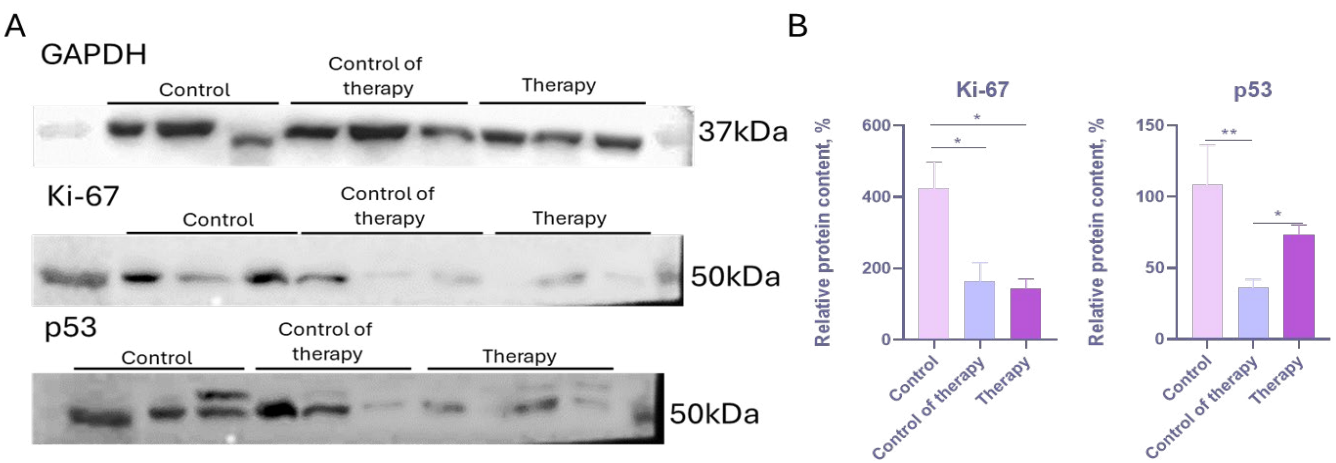

Fig. 1. A. — Western blot analysis of Ki-67, p53 proteins in endometriosis lesions in the "Control", "Control of Therapy", and "Therapy" groups. The reference protein GAPDH was used as a loading control. B — Relative levels of Ki-67, p53 proteins. Statistical analysis was performed using the non-parametric Kraskell-Wallis test with Dunn's post-hoc test (mean with SEM)

Note: * — significance of differences at $p < 0.05$; ** — significance of differences at $p < 0,005$.

phenotype did not suppress the production of p53 protein in endometriosis lesions when compared to the introduction of unpolarized macrophages as a control therapy (Figure 1 A, B).

Proinflammatory macrophages induced apoptosis in endometriosis lesions

As a consequence of the IHC analysis, it was determined that the introduction of macrophages exhibiting both a proinflammatory phenotype and an unpolarized state into animal models resulted in the activation of effector caspase-3 production in endometriosis lesions cells (Figure 2).

A quantitative analysis of positively stained cells revealed a significant increase ($p < 0.05$ and $p < 0.005$) in the content of cells actively producing caspase 3 following the introduction of macrophages (Figure 3). Concurrently, comparable levels of caspase 3-producing cells were observed subsequent to the introduction of proinflammatory and unpolarized macrophages.

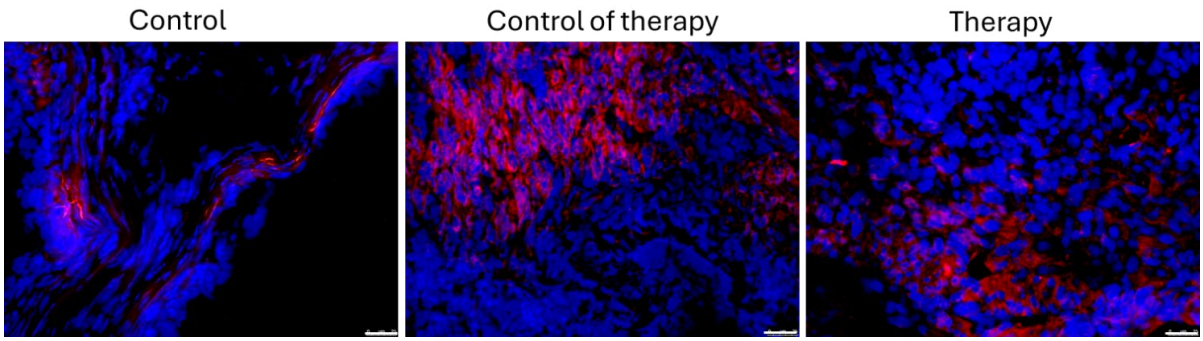

Fig. 2. A — IHC staining of endometriosis lesions against caspase 3 (red) in the "Control", "Control of Therapy", and "Therapy" groups. Nuclei are stained with DAPI (blue). Scale bar 25 μm

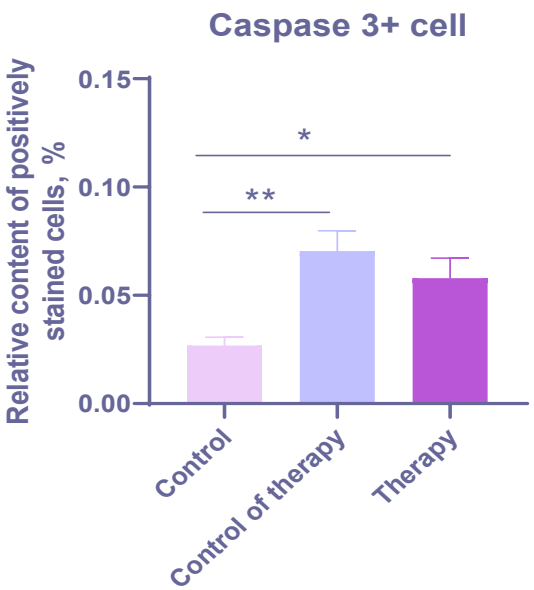

Fig. 3. Relative content of cells positively stained for caspase 3. Statistical analysis was performed using the non-parametric Kraskell-Wallis test with Dunn's post-hoc test (mean with SEM)

Note: *- significance of differences at $p < 0.05$; ** — significance of differences at $p < 0,005$.

Consequently, endometriosis manifests as a highly proliferative disease, exhibiting characteristics reminiscent of malignant neoplasms in its progression [8]. It has been demonstrated in previous studies that macrophages with a proinflammatory phenotype exhibit antitumor activity [9].

A similar therapeutic effect of proinflammatory macrophages in endometriosis has been previously studied [3]. The introduction of macrophages with a proinflammatory phenotype resulted in their migration to the lesions of endometriosis, thereby contributing to a reduction in the number and size of the lesions. Furthermore, the population of peritoneal macrophages was restored to a level corresponding to that observed in animals without endometriosis.

In this study, we sought to investigate the mechanisms by which macrophages with a proinflammatory phenotype exert an anti-endometriosis effect. Thus, we demonstrated that macrophages with a proinflammatory phenotype suppressed cell proliferation and stimulated the production of effector caspase 3, which triggers apoptosis in the cells of the lesions. Previous studies have shown that macrophages with a proinflammatory phenotype suppress proliferation and induce apoptosis in tumor cells [10].

Concurrently, our data suggest that the introduction of unpolarized macrophages into an animal model of endometriosis also led to a significant decrease in Ki-67 protein production compared to the control group, which corresponds to a decrease in cell proliferation in endometriosis lesions. Furthermore, unpolarized macrophages augmented the activation of caspase 3 production in the cells of the lesions in comparison to the control group. Consequently, the transplantation of macrophages that were not polarized towards proinflammatory activation also exerted an inhibitory effect on the growth of endometriosis lesions by suppressing cell proliferation and activating apoptosis. Consequently, unpolarized macrophages may also function as effective agents in the treatment of endometriosis.

Furthermore, it was demonstrated that the introduction of macrophages with a proinflammatory phenotype, in contrast to nonpolarized macrophages, does not result in the suppression of the production of the stress-induced tumor suppressor protein p53. A previous study indicated that reduced levels of p53 protein production in lesions in patients are associated with a recurrent form of deep endometriosis [11]. Furthermore, a decline in the proapoptotic activity of lesions cells has been observed to be associated with a recurrent form of endometriosis.

Conclusion

In a syngeneic mouse model of endometriosis, we demonstrated that the introduction of polarized macrophages toward a proinflammatory phenotype led to the inhibition of cell proliferation in endometriosis lesions, as well as their apoptosis due to increased production of p53 and caspase 3 proteins. Moreover, unpolarized macrophages have been shown to suppress cellular proliferation in endometriosis lesions and to activate apoptosis.

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Антипролиферативное и проапоптотическое действие — терапевтическая активность провоспалительных макрофагов при эндометриозе

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Аннотация: *Актуальность.* Эндометриоз, несмотря на длительное изучение, остается заболеванием с недостаточно эффективными и безопасными методами терапии, которые могли бы предотвратить его прогрессирование и улучшить качество жизни пациентов. Разработка новых методов лечения эндометриоза — это актуальная задача современной биомедицины. В связи с этим растет интерес к клеточной терапии, в частности к применению макрофагов. *Цель исследования* — проанализировать влияние введенных в животную модель макрофагов с провоспалительным фенотипом на пролиферацию и выживание клеток в очагах эндометриоза. *Материалы и методы.* На самках мыши линии C57Bl/6 получали сингенную модель эндометриоза путем внутрибрюшинной трансплантации фрагментов матки от мыши-донора. В качестве клеточного агента использовали мышиные макрофаги линии RAW264.7. Затем животных разделяли на группы: «Контроль» — мыши, которым не проводили терапию макрофагами; «Контроль терапии» — мыши, которым вводили неполяризованные макрофаги линии RAW264.7; «Терапия» — мыши с эндометриозом, которым вводили макрофаги линии RAW264.7 с провоспалительным фенотипом. *Результаты и обсуждение.* В результате введения макрофагов с провоспалительным фенотипом мы выявили значимое снижение продукции белка-маркера пролиферации Ki-67, а значимое увеличение продукции эффекторной каспазы 3 в клетках очагов эндометриоза по сравнению с «Контролем». При этом уровень продукции белка-супрессора опухоли p53, участвующих в запуске апоптоза в клетках, был сопоставим с таковым у группы «Контроль», в отличие от группы животных, которым вводились неполяризованные макрофаги. *Выводы.* Мы выявили, что анти-эндометриозная активность макрофагов с прововоспалительным фенотипом связана с тем, что их введение в мышиную модель подавляло пролиферацию клеток эндометриоза и усиливало их апоптотическую гибель благодаря активации белков p53 и каспазы 3.

Ключевые слова: эндометриоз, мышиная модель, клеточная терапия, макрофаги

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