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ЕЖЕМЕСЯЧНЫЙ НАУЧНЫЙ ЖУРНАЛ

Медицинские новости Грузии

საქართველოს სამედიცინო სიახლენი

GEORGIAN MEDICAL NEWS

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GMN: Georgian Medical News is peer-reviewed, published monthly journal committed to promoting the science and art of medicine and the betterment of public health, published by the GMN Editorial Board since 1994. GMN carries original scientific articles on medicine, biology and pharmacy, which are of experimental, theoretical and practical character; publishes original research, reviews, commentaries, editorials, essays, medical news, and correspondence in English and Russian.

GMN is indexed in MEDLINE, SCOPUS, PubMed and VINITI Russian Academy of Sciences. The full text content is available through EBSCO databases.

GMN: Медицинские новости Грузии - ежемесячный рецензируемый научный журнал, издаётся Редакционной коллегией с 1994 года на русском и английском языках в целях поддержки медицинской науки и улучшения здравоохранения. В журнале публикуются оригинальные научные статьи в области медицины, биологии и фармации, статьи обзорного характера, научные сообщения, новости медицины и здравоохранения. Журнал индексируется в MEDLINE, отражён в базе данных SCOPUS, PubMed и ВИНИТИ РАН. Полнотекстовые статьи журнала доступны через БД EBSCO.

GMN: Georgian Medical News – საქართველოს სამედიცინო სიახლენი – არის ყოველთვიური სამეცნიერო სამედიცინო რეცენზირებადი ჟურნალი, გამოიცემა 1994 წლიდან, წარმოადგენს სარედაქციო კოლეგიისა და აშშ-ის მეცნიერების, განათლების, ინდუსტრიის, ხელოვნებისა და ბუნებისმეტყველების საერთაშორისო აკადემიის ერთობლივ გამოცემას. GMN-ში რუსულ და ინგლისურ ენებზე ქვეყნდება ექსპერიმენტული, თეორიული და პრაქტიკული ხასიათის ორიგინალური სამეცნიერო სტატიები მედიცინის, ბიოლოგიისა და ფარმაციის სფეროში, მიმოხილვითი ხასიათის სტატიები.

ჟურნალი ინდექსირებულია MEDLINE-ის საერთაშორისო სისტემაში, ასახულია SCOPUS-ის, PubMed-ის და ВИНИТИ РАН-ის მონაცემთა ბაზებში. სტატიების სრული ტექსტი ხელმისაწვდომია EBSCO-ს მონაცემთა ბაზებიდან.

WEBSITE

www.geomednews.com

К СВЕДЕНИЮ АВТОРОВ!

При направлении статьи в редакцию необходимо соблюдать следующие правила:

1. Статья должна быть представлена в двух экземплярах, на русском или английском языках, напечатанная через **полтора интервала на одной стороне стандартного листа с шириной левого поля в три сантиметра**. Используемый компьютерный шрифт для текста на русском и английском языках - **Times New Roman** (**Кириллица**), для текста на грузинском языке следует использовать **AcadNusx.** Размер шрифта - **12**. К рукописи, напечатанной на компьютере, должен быть приложен CD со статьей.

2. Размер статьи должен быть не менее десяти и не более двадцати страниц машинописи, включая указатель литературы и резюме на английском, русском и грузинском языках.

3. В статье должны быть освещены актуальность данного материала, методы и результаты исследования и их обсуждение.

При представлении в печать научных экспериментальных работ авторы должны указывать вид и количество экспериментальных животных, применявшиеся методы обезболивания и усыпления (в ходе острых опытов).

4. К статье должны быть приложены краткое (на полстраницы) резюме на английском, русском и грузинском языках (включающее следующие разделы: цель исследования, материал и методы, результаты и заключение) и список ключевых слов (key words).

5. Таблицы необходимо представлять в печатной форме. Фотокопии не принимаются. **Все цифровые, итоговые и процентные данные в таблицах должны соответствовать таковым в тексте статьи**. Таблицы и графики должны быть озаглавлены.

6. Фотографии должны быть контрастными, фотокопии с рентгенограмм - в позитивном изображении. Рисунки, чертежи и диаграммы следует озаглавить, пронумеровать и вставить в соответствующее место текста **в tiff формате**.

В подписях к микрофотографиям следует указывать степень увеличения через окуляр или объектив и метод окраски или импрегнации срезов.

7. Фамилии отечественных авторов приводятся в оригинальной транскрипции.

8. При оформлении и направлении статей в журнал МНГ просим авторов соблюдать правила, изложенные в «Единых требованиях к рукописям, представляемым в биомедицинские журналы», принятых Международным комитетом редакторов медицинских журналов - http://www.spinesurgery.ru/files/publish.pdf и http://www.nlm.nih.gov/bsd/uniform_requirements.html
В конце каждой оригинальной статьи приводится библиографический список. В список литературы включаются все материалы, на которые имеются ссылки в тексте. Список составляется в алфавитном порядке и нумеруется. Литературный источник приводится на языке оригинала. В списке литературы сначала приводятся работы, написанные знаками грузинского алфавита, затем кириллицей и латиницей. Ссылки на цитируемые работы в тексте статьи даются в квадратных скобках в виде номера, соответствующего номеру данной работы в списке литературы. Большинство цитированных источников должны быть за последние 5-7 лет.

9. Для получения права на публикацию статья должна иметь от руководителя работы или учреждения визу и сопроводительное отношение, написанные или напечатанные на бланке и заверенные подписью и печатью.

10. В конце статьи должны быть подписи всех авторов, полностью приведены их фамилии, имена и отчества, указаны служебный и домашний номера телефонов и адреса или иные координаты. Количество авторов (соавторов) не должно превышать пяти человек.

11. Редакция оставляет за собой право сокращать и исправлять статьи. Корректура авторам не высылается, вся работа и сверка проводится по авторскому оригиналу.

12. Недопустимо направление в редакцию работ, представленных к печати в иных издательствах или опубликованных в других изданиях.

При нарушении указанных правил статьи не рассматриваются.

REQUIREMENTS

Please note, materials submitted to the Editorial Office Staff are supposed to meet the following requirements:

1. Articles must be provided with a double copy, in English or Russian languages and typed or compu-ter-printed on a single side of standard typing paper, with the left margin of **3** centimeters width, and **1.5** spacing between the lines, typeface - **Times New Roman** (**Cyrillic**), print size - **12** (referring to Georgian and Russian materials). With computer-printed texts please enclose a CD carrying the same file titled with Latin symbols.

2. Size of the article, including index and resume in English, Russian and Georgian languages must be at least 10 pages and not exceed the limit of 20 pages of typed or computer-printed text.

3. Submitted material must include a coverage of a topical subject, research methods, results, and review.

Authors of the scientific-research works must indicate the number of experimental biological species drawn in, list the employed methods of anesthetization and soporific means used during acute tests.

4. Articles must have a short (half page) abstract in English, Russian and Georgian (including the following sections: aim of study, material and methods, results and conclusions) and a list of key words.

5. Tables must be presented in an original typed or computer-printed form, instead of a photocopied version. **Numbers, totals, percentile data on the tables must coincide with those in the texts of the articles.** Tables and graphs must be headed.

6. Photographs are required to be contrasted and must be submitted with doubles. Please number each photograph with a pencil on its back, indicate author's name, title of the article (short version), and mark out its top and bottom parts. Drawings must be accurate, drafts and diagrams drawn in Indian ink (or black ink). Photocopies of the X-ray photographs must be presented in a positive image in **tiff format**.

Accurately numbered subtitles for each illustration must be listed on a separate sheet of paper. In the subtitles for the microphotographs please indicate the ocular and objective lens magnification power, method of coloring or impregnation of the microscopic sections (preparations).

7. Please indicate last names, first and middle initials of the native authors, present names and initials of the foreign authors in the transcription of the original language, enclose in parenthesis corresponding number under which the author is listed in the reference materials.

8. Please follow guidance offered to authors by The International Committee of Medical Journal Editors guidance in its Uniform Requirements for Manuscripts Submitted to Biomedical Journals publication available online at: http://www.nlm.nih.gov/bsd/uniform_requirements.html
http://www.icmje.org/urm_full.pdf
In GMN style for each work cited in the text, a bibliographic reference is given, and this is located at the end of the article under the title "References". All references cited in the text must be listed. The list of references should be arranged alphabetically and then numbered. References are numbered in the text [numbers in square brackets] and in the reference list and numbers are repeated throughout the text as needed. The bibliographic description is given in the language of publication (citations in Georgian script are followed by Cyrillic and Latin).

9. To obtain the rights of publication articles must be accompanied by a visa from the project instructor or the establishment, where the work has been performed, and a reference letter, both written or typed on a special signed form, certified by a stamp or a seal.

10. Articles must be signed by all of the authors at the end, and they must be provided with a list of full names, office and home phone numbers and addresses or other non-office locations where the authors could be reached. The number of the authors (co-authors) must not exceed the limit of 5 people.

11. Editorial Staff reserves the rights to cut down in size and correct the articles. Proof-sheets are not sent out to the authors. The entire editorial and collation work is performed according to the author's original text.

12. Sending in the works that have already been assigned to the press by other Editorial Staffs or have been printed by other publishers is not permissible.

Articles that Fail to Meet the Aforementioned Requirements are not Assigned to be Reviewed.

ავტორთა საყურადღებოდ!

რედაქციაში სტატიის წარმოდგენისას საჭიროა დავიცვათ შემდეგი წესები:

1. სტატია უნდა წარმოადგინოთ 2 ცალად, რუსულ ან ინგლისურ ენებზე,დაბეჭდილი სტანდარტული ფურცლის 1 გვერდზე, 3 სმ სიგანის მარცხენა ველისა და სტრიქონებს შორის 1,5 ინტერვალის დაცვით. გამოყენებული კომპიუტერული შრიფტი რუსულ და ინგლისურენოვან ტექსტებში - **Times New Roman (Кириллица)**, ხოლო ქართულენოვან ტექსტში საჭიროა გამოვიყენოთ **AcadNusx.** შრიფტის ზომა – 12. სტატიას თან უნდა ახლდეს CD სტატიით.

2. სტატიის მოცულობა არ უნდა შეადგენდეს 10 გვერდზე ნაკლებს და 20 გვერდზე მეტს ლიტერატურის სიის და რეზიუმეების (ინგლისურ, რუსულ და ქართულ ენებზე) ჩათვლით.

3. სტატიაში საჭიროა გაშუქდეს: საკითხის აქტუალობა; კვლევის მიზანი; საკვლევი მასალა და გამოყენებული მეთოდები; მიღებული შედეგები და მათი განსჯა. ექსპერიმენტული ხასიათის სტატიების წარმოდგენისას ავტორებმა უნდა მიუთითონ საექსპერიმენტო ცხოველების სახეობა და რაოდენობა; გაუტკივარებისა და დაძინების მეთოდები (მწვავე ცდების პირობებში).

4. სტატიას თან უნდა ახლდეს რეზიუმე ინგლისურ, რუსულ და ქართულ ენებზე არანაკლებ ნახევარი გვერდის მოცულობისა (სათაურის, ავტორების, დაწესებულების მითითებით და უნდა შეიცავდეს შემდეგ განყოფილებებს: მიზანი, მასალა და მეთოდები, შედეგები და დასკვნები; ტექსტუალური ნაწილი არ უნდა იყოს 15 სტრიქონზე ნაკლები) და საკვანძო სიტყვების ჩამონათვალი (key words).

5. ცხრილები საჭიროა წარმოადგინოთ ნაბეჭდი სახით. ყველა ციფრული, შემაჯამებელი და პროცენტული მონაცემები უნდა შეესაბამებოდეს ტექსტში მოყვანილს.

6. ფოტოსურათები უნდა იყოს კონტრასტული; სურათები, ნახაზები, დიაგრამები - დასათაურებული, დანომრილი და სათანადო ადგილას ჩასმული. რენტგენოგრამების ფოტოასლები წარმოადგინეთ პოზიტიური გამოსახულებით **tiff** ფორმატში. მიკროფოტოსურათების წარწერებში საჭიროა მიუთითოთ ოკულარის ან ობიექტივის საშუალებით გადიდების ხარისხი, ანათალების შეღებვის ან იმპრეგნაციის მეთოდი და აღნიშნოთ სურათის ზედა და ქვედა ნაწილები.

7. სამამულო ავტორების გვარები სტატიაში აღინიშნება ინიციალების თანდართვით, უცხოურისა – უცხოური ტრანსკრიპციით.

8. სტატიას თან უნდა ახლდეს ავტორის მიერ გამოყენებული სამამულო და უცხოური შრომების ბიბლიოგრაფიული სია (ბოლო 5-8 წლის სიღრმით). ანბანური წყობით წარმოდგენილ ბიბლიოგრაფიულ სიაში მიუთითეთ ჯერ სამამულო, შემდეგ უცხოელი ავტორები (გვარი, ინიციალები, სტატიის სათაური, ჟურნალის დასახელება, გამოცემის ადგილი, წელი, ჟურნალის №, პირველი და ბოლო გვერდები). მონოგრაფიის შემთხვევაში მიუთითეთ გამოცემის წელი, ადგილი და გვერდების საერთო რაოდენობა. ტექსტში კვადრატულ ფჩხილებში უნდა მიუთითოთ ავტორის შესაბამისი N ლიტერატურის სიის მიხედვით. მიზანშეწონილია, რომ ციტირებული წყაროების უმეტესი ნაწილი იყოს 5-6 წლის სიღრმის.

9. სტატიას თან უნდა ახლდეს: ა) დაწესებულების ან სამეცნიერო ხელმძღვანელის წარდგინება, დამოწმებული ხელმოწერითა და ბეჭდით; ბ) დარგის სპეციალისტის დამოწმებული რეცენზია, რომელშიც მითითებული იქნება საკითხის აქტუალობა, მასალის საკმაობა, მეთოდის სანდოობა, შედეგების სამეცნიერო-პრაქტიკული მნიშვნელობა.

10. სტატიის ბოლოს საჭიროა ყველა ავტორის ხელმოწერა, რომელთა რაოდენობა არ უნდა აღემატებოდეს 5-ს.

11. რედაქცია იტოვებს უფლებას შეასწოროს სტატია. ტექსტზე მუშაობა და შეჯერება ხდება საავტორო ორიგინალის მიხედვით.

12. დაუშვებელია რედაქციაში ისეთი სტატიის წარდგენა, რომელიც დასაბეჭდად წარდგენილი იყო სხვა რედაქციაში ან გამოქვეყნებული იყო სხვა გამოცემებში.

აღნიშნული წესების დარღვევის შემთხვევაში სტატიები არ განიხილება.

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EXPERIMENTAL EVALUATION OF TISSUE RESPONSE TO IMPLANT MATERIALS UNDER *ESCHERICHIA COLI* CONTAMINATION

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Abstract.

Aim of the study: To compare tissue responses to xenoperitoneum-derived extracellular matrix (ECM), UltraPro mesh, and preserved dura mater in a rat model of implant-associated *Escherichia coli* infection.

Methods: Abdominal wall repair was performed in 42 rats using ECM, UltraPro mesh, or preserved dura mater with intraoperative *E. coli* contamination. Histological and morphometric evaluations were conducted on postoperative days 10 and 20, assessing inflammation, abscess formation, necrosis, neovascularization, and tissue integration.

Results: On day 10, all groups demonstrated acute inflammation. Abscess and necrosis were significantly lower with UltraPro than with ECM and preserved dura mater ($p<0.05$). By day 20, UltraPro maintained minimal tissue damage and showed the highest neovascularization and tissue integration, while preserved dura mater exhibited persistent inflammation. ECM demonstrated reduced necrosis and signs of tissue remodeling at later stages.

Conclusion: UltraPro mesh provides superior early resistance to infection-related tissue damage, whereas potential advantages of ECM appear context-dependent and are more evident at later stages.

Key words. Xenoperitoneum-derived extracellular matrix, UltraPro mesh, dura mater, *Escherichia coli*, implant-associated infection, tissue response, angiogenesis, abdominal wall reconstruction.

Introduction.

Reconstruction of anterior abdominal wall defects is among the most commonly performed procedures in abdominal surgery. Despite the widespread use of synthetic mesh implants, which have significantly reduced hernia recurrence rates, the success of abdominal wall repair largely depends on the condition of the surgical field and the severity of the local inflammatory response [1-3]. Even minimal bacterial contamination may impair implant integration, intensify acute inflammation, and lead to mesh-related complications [1,4].

Gram-negative microorganisms, particularly *Escherichia coli*, are of major clinical relevance, as they represent one of the most frequent causative agents of early postoperative infections following abdominal surgery [4]. Implant-associated *E. coli* infection is characterized by a pronounced neutrophilic response, endotoxin-mediated tissue injury, and destabilization of the extracellular matrix [5,6]. These factors create unfavorable conditions for tissue remodeling and increase the risk of reconstructive failure.

The ability of *E. coli* to form stable biofilms on the surface of various implant materials is a key determinant of its pathogenicity. Bacterial adhesion and subsequent biofilm formation significantly reduce the effectiveness of antibacterial therapy and promote chronic inflammation in the peri-implant zone [2,7]. As a result, the risk of abscess formation, fistula development, and implant explantation increases. Therefore, the selection of implant material for hernia repair in the context of potential gram-negative contamination is of critical importance. Recent experimental studies conducted in Kazakhstan have highlighted the relevance of interactions between biomaterials and microorganisms, demonstrating antibacterial and antifungal activity of novel biocomposite materials and underscoring the importance of material properties in infected surgical environments [6].

The development of biological implants based on decellularized extracellular matrix (ECM) has substantially expanded the possibilities of reconstructive surgery. Owing to the preserved collagen architecture and biologically active stromal components, these materials provide a more physiological pattern of integration, induce a less pronounced foreign-body reaction, and promote neovascularization [8]. Experimental studies have demonstrated that biological scaffolds can elicit a regulated immune response and support tissue remodeling even under contaminated conditions [9]. However, data on the morphological interaction between ECM-based materials and host tissues in the setting of *E. coli* infection remain limited.

In the Republic of Kazakhstan, a novel biological implant based on xenoperitoneum-derived extracellular matrix has been developed. Its manufacturing technology, structural and biomechanical properties, as well as experimental outcomes under aseptic conditions, have been reported in several studies, including a national patent and a doctoral dissertation [10-17]. Nevertheless, the impact of gram-negative contamination on the morphological integration of this material has not been previously described, highlighting the relevance of studies aimed at evaluating tissue responses to different implant materials under *E. coli* infection.

The aim of this study was to perform a comparative histological and morphometric assessment of inflammatory response, angiogenesis, and early tissue remodeling around xenoperitoneum-derived extracellular matrix, synthetic UltraPro mesh, and preserved dura mater in an experimental *E. coli* infection model.

Materials and Methods.

Study design:

A controlled experimental study was conducted to evaluate morphological changes in the implantation zone of different materials following targeted *E. coli* contamination. The study was performed in accordance with the ARRIVE guidelines and the ICMJE recommendations regarding reproducibility and transparency of experimental data.

Randomization and blinding:

Animals were randomly allocated to experimental groups using a random number generator prior to surgery. Histological and morphometric evaluations were performed by an investigator blinded to group allocation to minimize observer bias and ensure objectivity of the assessment.

Experimental Animals and Housing Conditions:

The study included 42 adult outbred white rats of both sexes, weighing 180–220 g. Animals were housed in the vivarium of Karaganda Medical University under standard conditions, including controlled temperature, free access to food and water, and a fixed light–dark cycle. Animals were selected based on clinical well-being, absence of external signs of disease, and body weight compliance (±10%). Each animal was assigned an individual identification number. Housing conditions complied with the *Guide for the Care and Use of Laboratory Animals* (NIH, 2011). General health status was monitored daily.

Experimental Groups:

The contaminated experimental model included 36 rats distributed according to three types of implanted materials and two observation periods—postoperative days 10 and 20. Each subgroup consisted of six animals.

The following experimental groups were formed:

- **Xenoperitoneum-derived extracellular matrix (ECM):**
  – *E. coli*, day 10: n = 6
  – *E. coli*, day 20: n = 6
- **UltraPro mesh (Ethicon, USA):**
  – *E. coli*, day 10: n = 6
  – *E. coli*, day 20: n = 6
- **Preserved dura mater:**
  – *E. coli*, day 10: n = 6
  – *E. coli*, day 20: n = 6

The bacterial contamination was performed using a clinically relevant *E. coli* strain isolated from a postoperative surgical site infection. The strain belongs to the Enterobacteriaceae family and represents a Gram-negative, facultative anaerobic bacillus. It is characterized by high adhesive capacity, the ability to form stable biofilms on implant surfaces, and pronounced endotoxin-mediated pathogenicity associated with lipopolysaccharide (LPS) expression. These properties make this strain suitable for modeling implant-associated infections and early postoperative inflammatory complications.

UltraPro mesh is a partially absorbable composite surgical implant consisting of non-absorbable polypropylene monofilaments combined with absorbable polyglycolide fibers. This lightweight mesh design is intended to reduce foreign-body reaction while maintaining sufficient mechanical strength and is widely used in clinical hernia repair, including in potentially contaminated surgical fields.

Non-infected control group:

A non-infected control group (total n = 6) underwent the same surgical procedure with implantation of the corresponding material without bacterial inoculation. To ensure equal handling, animals received 0.5 mL of sterile 0.9% sodium chloride solution applied to the implant surface and surrounding tissues (sterile vehicle control). The control group included n = 2 animals per material (ECM, UltraPro, and preserved dura mater).

This group was used for qualitative assessment of baseline foreign-body tissue response under aseptic conditions and for validation of the model. Quantitative morphometric comparisons and statistical testing were pre-specified for contaminated groups only, which constituted the primary objective of the study.

Implant Materials:

The following materials were used:

1. **Xenoperitoneum-derived extracellular matrix (ECM)** — a biological implant obtained through a multistep detergent–enzymatic decellularization process followed by gamma irradiation sterilization;
2. **UltraPro mesh** (Ethicon) — a polypropylene–polyglycolide composite mesh commonly used in hernia surgery;
3. **Preserved dura mater** — a biological material applied in reconstructive procedures.

All implant samples were standardized to a size of 1 × 1 cm.

Bacterial Agent:

To model an infected surgical field, a clinical strain of *E. coli* at a concentration of 10^9 CFU/mL was used. Prior to application, the strain was verified using standard microbiological methods. Contamination was performed intraoperatively by evenly distributing 0.5 mL of the bacterial suspension over the implant surface and surrounding tissues. Animals in the control group received an equivalent volume of sterile physiological saline.

Surgical Procedure:

All procedures were performed under inhalation ether anesthesia. A midline incision approximately 2 cm in length was made on the anterior abdominal wall. A standardized aponeurotic defect measuring 1 × 1 cm was created, and one of the investigated implants was fixed using interrupted PDS 5/0 sutures. Muscle layers and skin were closed in layers. Postoperative care included daily clinical observation and assessment of general condition. Animals were euthanized on postoperative days 10 and 20.

Histological Sample Collection:

After euthanasia, a tissue block including the implant, adjacent muscle and connective tissue, and areas of granulation tissue was excised. The resection margin was at least 5 mm from the implant edge. Specimens were fixed in 4% neutral buffered formalin, processed using standard histological techniques,

embedded in paraffin, sectioned at 5 μm, and stained with hematoxylin and eosin.

Morphological and Morphometric Evaluation.

Qualitative Assessment:

The analysis included evaluation of the inflammatory infiltrate pattern, predominant cell populations, presence of microabscesses and necrosis, degree of granulation tissue maturation, signs of fibrotic remodeling, and involvement of multinucleated giant cells.

Cellular Morphometry:

Cell counting was performed at ×400 magnification in the implant–tissue interface zone, avoiding areas containing suture material. At least 300 cells per specimen were analyzed, including granulocytes, lymphocytes, macrophages, plasma cells, and fibroblasts/fibrocytes.

Connective Tissue Maturation:

A semi-quantitative scale (0–3 points) was used to assess the degree of fibrotic remodeling:

- 0 — absence of mature connective tissue;
- 1 — focal collagen fiber formation;
- 2 — pronounced areas of connective tissue formation;
- 3 — diffuse mature fibrosis.

Scoring Parameters:

Mean scores ($\bar{X} \pm SD$) were calculated for the following parameters:

- abscess formation (0–4 points);
- necrosis (0–3 points);
- depth of inflammation (0–4 points);
- neovascularization (0–4 points);
- cellular infiltration and tissue integration (0–4 points).

Detailed scoring criteria are provided in the Results section.

Statistical Analysis:

Data processing was performed using IBM SPSS Statistics 26.0 and Microsoft Excel 2019. Given the small sample size and ordinal nature of several variables, nonparametric statistical methods were applied. Comparisons among three materials at the same time point were conducted using the Kruskal–Wallis test (H, df, p). Comparisons between independent groups at postoperative days 10 and 20 were performed using the Mann–Whitney U test (U, p). Data are presented as mean ± SD for clarity, while statistical evaluation was conducted using nonparametric tests. Differences were considered statistically significant at $p \leq 0.05$.

Ethical Approval:

The experimental protocol was approved by the Local Ethics Committee of Karaganda Medical University (Protocol No. 3, dated 27 February 2024). All procedures complied with the European Convention for the Protection of Vertebrate Animals (ETS No. 123), the Guide for the Care and Use of Laboratory Animals (NIH, 2011), and the regulatory requirements of the Republic of Kazakhstan.

Results.

In the non-infected control animals, implantation of all materials resulted in a mild foreign-body tissue reaction without abscess formation or necrosis, supporting the adequacy of the aseptic surgical model. Therefore, the pronounced abscess formation and necrotic changes observed in the experimental groups should be interpreted as being primarily driven by bacterial contamination rather than implantation alone.

Cellular Composition of the Inflammatory Infiltrate:

Quantitative assessment of the cellular inflammatory infiltrate on postoperative days 10 and 20 following implantation and targeted *E. coli* contamination is presented in Table 1.

Day 10. In all study groups, a pronounced inflammatory response was observed, characterized by a predominance of granulocytes and lymphocytes. In the ECM group, the number of granulocytes and lymphocytes was 177.7 ± 7.8 and 55.3 ± 9.1, respectively. In the UltraPro group, these values were 172.2 ± 12.8 and 47.8 ± 4.4, while in the preserved dura mater group they reached 194.0 ± 25.8 and 47.3 ± 9.4, respectively.

The macrophage response was most pronounced in the UltraPro group (40.2 ± 12.4), whereas lower values were recorded in the ECM and dura mater groups (28.7 ± 2.6 and 21.3 ± 12.6, respectively). The number of fibroblasts/fibrocytes on day 10 remained low in all groups: 15.7 ± 4.0 in the ECM group, 32.2 ± 10.5 in the UltraPro group, and 20.7 ± 6.4 in the dura mater group.

Morphologically, the ECM group demonstrated the formation of granulation tissue with areas of granulocytic and lymphomacrophage infiltration, as well as localized microabscesses and small necrotic foci (Figure 1).

In the UltraPro group, focal lymphocytic infiltration and an attenuating perivascular granulocytic reaction were observed. Small and medium-sized abscesses were identified around the mesh elements (Figure 2).

In the dura mater group, diffuse leukocytic infiltration with the presence of microabscesses around the implant was observed (Figure 3).

Day 20: By day 20, a divergent inflammatory response was observed among the study groups. The ECM and UltraPro groups demonstrated a significant decrease in the number of granulocytes compared with postoperative day 10, to 146.0 ± 16.7 and 106.2 ± 6.0, respectively ($p < 0.05$). Concurrently, an increase in lymphocyte counts was noted in these groups, reaching 85.8 ± 9.9 in the ECM group and 80.2 ± 11.7 in the UltraPro group ($p < 0.05$ compared with day 10), indicating a shift toward a more chronic inflammatory phase.

In contrast, no significant changes were observed in the dura mater group compared with day 10, with granulocyte and lymphocyte counts of 192.8 ± 31.0 and 43.0 ± 11.3, respectively ($p > 0.05$). Morphologically, the ECM group exhibited the formation of loose connective tissue; however, pronounced granulocytic and lymphomacrophage infiltration persisted (Figure 4).

In the UltraPro group, more mature connective tissue was formed, accompanied by scattered lymphohistiocytic infiltration and a persistent perivascular inflammatory reaction (Figure 5).

In the dura mater group, microabscesses and focal lymphocytic infiltration persisted (Figure 6).

Abscesses and necrosis: The assessment of abscess formation and necrotic changes in the implantation zone is presented in Table 2.

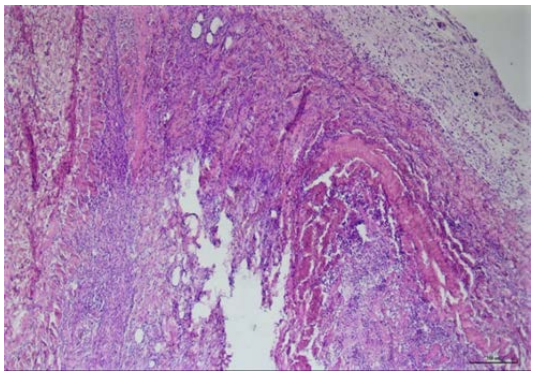

Figure 1. *ECM, E. coli, day 10. Fragments of the implant with granulocytic and lymphomacrophage infiltration and areas of forming connective tissue. Well-demarcated necrotic foci and microabscesses. Hematoxylin and eosin staining, ×100.*

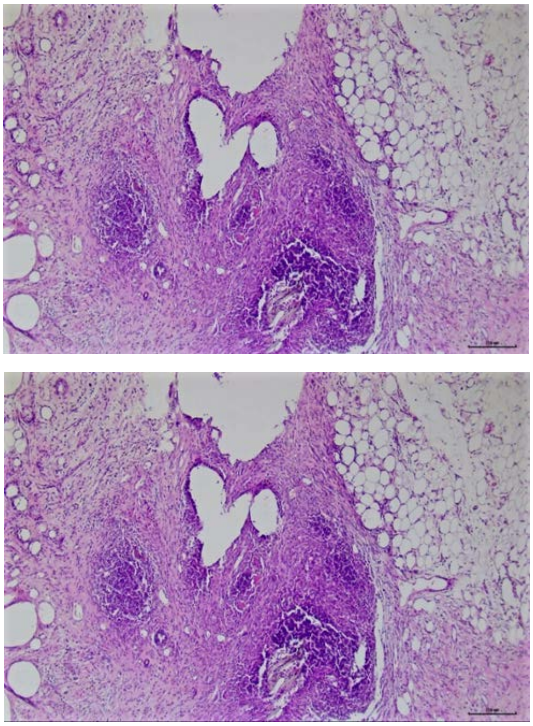

Figure 2. *UltraPro mesh, E. coli, day 10. Areas of forming connective tissue around the mesh elements; small to medium-sized abscesses within the implantation zone. Hematoxylin and eosin staining, ×100.*

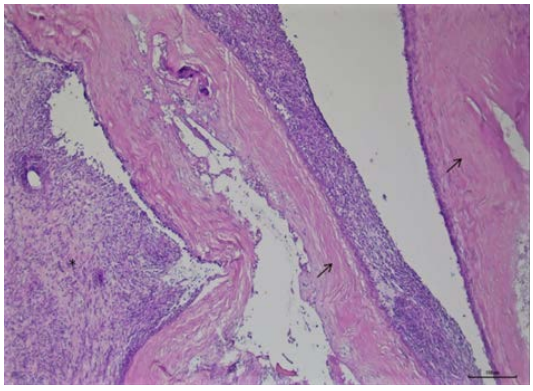

Figure 3. *Preserved dura mater, E. coli, day 10. Dura mater (black arrow) with adjacent connective tissue (black asterisk) showing diffuse leukocytic infiltration. Hematoxylin and eosin staining, ×200.*

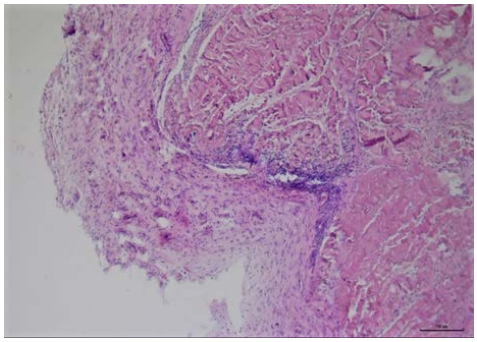

Figure 4. *ECM, E. coli, day 20. Fragments of connective tissue with granulocytic and lymphomacrophage infiltration. Hematoxylin and eosin staining, ×100.*

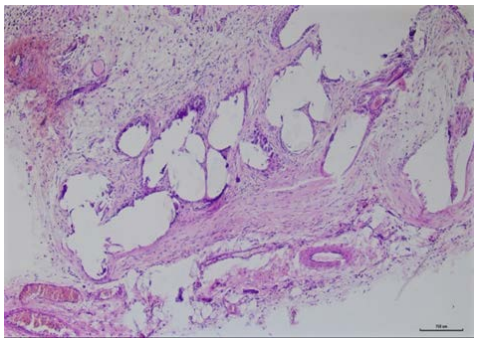

Figure 5. *UltraPro mesh, E. coli, day 20. Mature connective tissue formed around the mesh filaments with scattered lymphohistiocytic infiltration and persistent perivascular infiltration by polymorphonuclear leukocytes. Hematoxylin and eosin staining, ×100.*

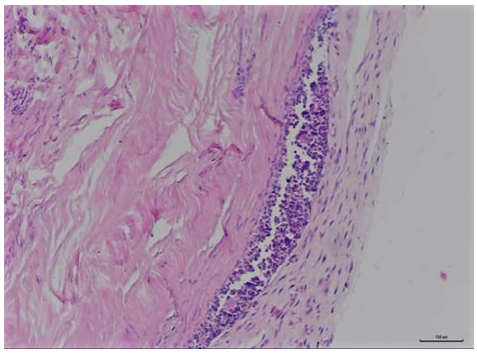

Figure 6. *Dura mater, E. coli, day 20. Fragment of the implant with microabscesses. Hematoxylin and eosin staining, ×200.*

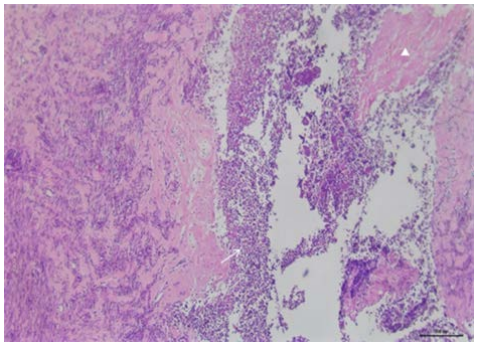

Figure 7. *Preserved dura mater, E. coli, day 10. Fragment of the implant demonstrating material degradation with abscess formation and necrotic changes. White arrow indicates areas of abscess formation; white triangle denotes necrotic tissue. Hematoxylin and eosin staining, ×100.*

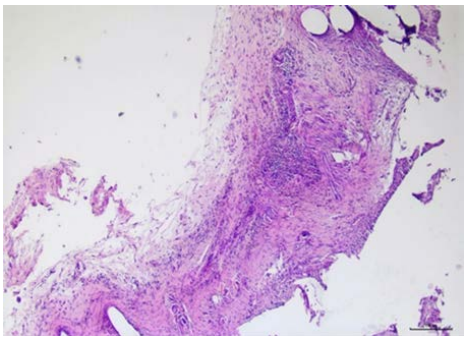

Figure 8. *UltraPro mesh, E. coli, day 10. Formation of connective tissue around the mesh with occasional small microabscesses. Predominance of fibroblasts and fibrocytes within the cellular infiltrate and persistent perivascular infiltration by polymorphonuclear leukocytes. Hematoxylin and eosin staining, ×100.*

Table 1. *Quantitative assessment of the cellular infiltrate in the implantation zone after E. coli contamination, mean ± SD.*

Cellular infiltrate	ECM		UltraPro		Preserved dura mater	
	10 days	20 days	10 days	20 days	10 days	20 days
Granulocytes	177.7±7.8	146±16.7	172.2±12.8	106.2±6.0	194±25.8	192.8±31
Lymphocytes	55.3±9.1	85.8±9.9	47.8±4.4	80.2±11.7	47.3±9.4	43.0±11.3
Plasma cells	22.7±4.1	21.2±7.8	7.7±3.3	13.8±3.6	16.7±5.3	14.0±5.9
Macrophages	28.7±2.6	20.2±12.5	40.2±12.4	43.2±9.9	21.3±12.6	21.3±10.2
Fibroblasts / fibrocytes	15.7±4.0	26.8±8.8	32.2±10.5	56.7±9.2	20.7±6.4	25.5±15.8

Table 2. *Abscess formation and necrotic changes in the implantation zone of the studied materials after E. coli contamination, $\bar{X} \pm SD$.*

Parameter	ECM		UltraPro		Preserved dura mater	
	10 days	20 days	10 days	20 days	10 days	20 days
Abscesses *	2.1±0.6	1.1±0.5	0.6±0.4	0.1±0.5	2.6±0.5	1.2±0.5
Necrosis**	1.3±0.6	0.7±0.5	0.5±0.5	0.1±0.4	1.6±0.5	0.5±0.5

Notes:
* – Histological abscess scoring scale: 0 - no abscesses; 0,5 - one small abscess; 1- several small abscesses; 2 - one medium-sized abscess; 3 - one or several medium-sized abscesses; 4 - one very large or multiple large abscesses;
** – Histological necrosis scoring scale: 0 - no necrosis; 1 - total necrotic area ≤ 1/3 of tissue area, 2 - necrotic area between 1/3 and 2/3 of tissue area; 3 - necrotic area > 2/3 of tissue area.

Table 3. *Depth of inflammation, neovascularization, cellular infiltration and tissue integration following E. coli contamination, mean ± SD.*

Inflammatory parameters	ECM		UltraPro		Preserved dura mater	
	10 days	20 days	10 days	20 days	10 days	20 days
Depth of inflammatory response *	1.8±0.5	0.9±0.5	1.5±0.5	0.6±0.5	2.1±0.5	1.9±0.5
Neovascularization **	0.9±0.4	1.4±0.5	1.7±0.5	2.8±0.4	0.8±0.5	1.3±0.6
Cellular infiltration and tissue integration***	0.5±0.4	1.5±0.5	1.5±0.4	3.4±0.5	0.5±0.5	1.4±0.5

Notes:
* – Depth of inflammatory response scoring scale: absence of inflammatory cells– 0, inflammatory cells present within one third of the tissue matrix – 1, inflammatory cells present within two thirds of the tissue matrix – 2, inflammatory cells present throughout the entire thickness of the tissue matrix – 3, pronounced diffuse inflammatory infiltration– 4;
** – Neovascularization scoring scale: absence of capillaries – 0, rare capillaries – 1, few capillaries (<5 capillaries per high-power field, HPF) – 2, moderate number of capillaries (5–10 capillaries per HPF) – 3, abundant capillary formation (granulation tissue) – 4;
*** – Cellular infiltration and tissue integration scoring scale: absence of fibroblast nuclei or cellular infiltration – 0; fibroblast nuclei or cellular infiltration present within one third of the implant or matrix – 1; within two thirds – 2; throughout the entire thickness – 3; pronounced or diffuse tissue integration / matrix remodeling – 4.

Day 10. Abscess formation was most pronounced in the xenoperitoneum extracellular matrix and dura mater groups, with mean scores of 2.1 ± 0.6 and 2.6 ± 0.5, respectively, whereas significantly lower values were observed in the UltraPro group (0.6 ± 0.4) ($p < 0.05$).

Necrotic changes were also more prominent in biologically derived implants, with scores of 1.3 ± 0.6 for the xenoperitoneum extracellular matrix and 1.6 ± 0.5 for the dura mater, compared with 0.5 ± 0.5 in the UltraPro group. These findings were supported by morphological observations revealing extensive areas of tissue destruction in the biological materials and more limited changes in the UltraPro group (Figures 7 and 8).

Day 20: In the ECM and preserved dura mater groups, a statistically significant reduction in necrosis scores was observed compared with postoperative day 10: from 1.3 ± 0.6 to 0.7 ± 0.5 in the ECM group ($p < 0.05$) and from 1.6 ± 0.5 to 0.5 ± 0.5 in the dura mater group ($p < 0.05$). Abscess scores also decreased in these groups (ECM: 2.1 ± 0.6 to 1.1 ± 0.5; dura mater: 2.6 ± 0.5 to 1.2 ± 0.5). In the UltraPro group, both abscess and necrosis scores remained low across the observation period (abscesses: 0.6 ± 0.4 to 0.1 ± 0.5; necrosis: 0.5 ± 0.5 to 0.1 ± 0.4).

Depth of inflammation, neovascularization, cellular infiltration and tissue integration.

In the present study, this parameter reflects biologically distinct processes depending on implant type: cellular infiltration and tissue integration for synthetic meshes, and matrix remodeling with gradual replacement by host tissue for biological extracellular matrix implants.

The results of the semi-quantitative assessment are presented in Table 3.

Day 10: The depth of the inflammatory response was more pronounced in the ECM and preserved dura mater groups (1.8 ± 0.5 and 2.1 ± 0.5, respectively), whereas a lower score was observed in the UltraPro group (1.5 ± 0.5). At this time point, the neovascular response was markedly higher in the UltraPro group (1.7 ± 0.5) compared with the biological materials (0.9 ± 0.4 for ECM and 0.8 ± 0.5 for PDM). Cellular infiltration and tissue integration at day 10 remained minimal in all groups, particularly in the biological implants (0.5 ± 0.4 for ECM and 0.5 ± 0.5 for PDM), whereas UltraPro demonstrated more pronounced cellular repopulation (1.5 ± 0.4).

Day 20: Both the ECM and UltraPro groups demonstrated a significant reduction in the depth of the inflammatory response to 0.9 ± 0.5 and 0.6 ± 0.5, respectively ($p < 0.05$). In contrast, the level of inflammation in the preserved dura mater group remained high (1.9 ± 0.5; $p > 0.05$). Neovascularization increased in all groups, with the most pronounced response observed in the UltraPro group (2.8 ± 0.4). Cellular infiltration and tissue integration also intensified across all implants; however, the highest score was recorded in the UltraPro group (3.4 ± 0.5), which was statistically higher than that of the biological materials ($p < 0.05$).

Discussion.

The obtained results demonstrate pronounced differences in tissue responses to implants of various types under *E. coli* infection. According to the current literature, Gram-negative pathogens induce intense neutrophilic infiltration, cause extracellular matrix damage mediated by lipopolysaccharides, and exert an adverse effect on the early stages of implant integration [9,18]. Our findings are consistent with these mechanisms: on postoperative day 10, all groups exhibited a predominance of granulocytes and lymphocytes, along with the formation of microabscesses and foci of necrosis.

Recent systematic reviews and narrative analyses published over the last decade have emphasized that the choice between biologic and synthetic meshes in contaminated or infected settings remains highly context-dependent. Contemporary data indicate that synthetic meshes often demonstrate superior early resistance to infection and structural stability, whereas biological matrices may provide advantages in terms of tissue remodeling and immunomodulation, particularly under controlled contamination or antibiotic-protected conditions. These findings highlight the absence of a universally optimal implant material and underscore the importance of tailoring material selection to the severity of contamination, host factors, and timing of reconstruction [6,7,19,20].

Acute inflammatory response: differences between implant types:

Biological materials (xenoperitoneum ECM and dura mater) exhibited a more pronounced acute inflammatory response and a higher incidence of abscess formation and necrosis on postoperative day 10. Similar findings have been reported in studies showing that biomaterials exposed to bacterial load may retain bacterial debris for a prolonged period, thereby sustaining the acute phase of inflammation [22]. In contrast, synthetic materials, including polypropylene-based composite meshes, generally induce less severe necrotic changes at early time points [23], which is consistent with the results obtained for UltraPro in the present study.

According to the quantitative data presented in Table 2, the synthetic UltraPro mesh demonstrated significantly lower abscess formation and necrotic changes on postoperative day 10 compared with the xenoperitoneum-derived extracellular matrix. These findings indicate superior early resistance of the synthetic material to infection-related tissue damage under conditions of severe bacterial challenge.

In contrast, the biological ECM exhibited a higher susceptibility to early abscess formation and necrosis. Given the high bacterial load used in this model (10^9 CFU/mL), this response may be explained by the biological nature of the matrix, which under overwhelming infection conditions can function as a provisional scaffold facilitating bacterial persistence and abscess development. Thus, the early disadvantage of ECM observed in this study appears to be closely linked to the severity of the infection model rather than to an inherent inferiority of the material.

It is well established that *E. coli* is capable of forming stable biofilms on both biological and synthetic implants [24]. Biofilm formation is associated with chronic inflammation and reduced phagocytic efficiency, which in our study was reflected by the persistence of lymphomacrophage infiltration at 20 days, particularly in the dura mater group. The more pronounced macrophage response observed around UltraPro is in line with reports describing sustained M1 macrophage activation as a characteristic feature of synthetic implants [25].

Cellular infiltration and tissue integration, and angiogenesis:

It should be emphasized that the term "tissue integration / matrix remodeling" encompasses different biological processes depending on implant type. In synthetic meshes such as UltraPro, this parameter reflects cellular infiltration and fibrous tissue integration around mesh filaments. In contrast, for biological ECM, it represents progressive scaffold remodeling and replacement by host-derived connective tissue.

Despite the pronounced acute inflammatory response, the xenoperitoneum ECM demonstrated signs of earlier tissue reconstruction, including enhanced tissue integration, formation of loose connective tissue, and increased capillary growth by day 20. These findings are consistent with numerous experimental studies showing that ECM-based materials promote constructive remodeling even under contaminated conditions [4,26,27]. Preservation of the collagen scaffold architecture and the presence of biologically active components create favorable conditions for fibroblast and endothelial cell migration. In contrast, the dura mater group exhibited delayed vascular network formation and a lower degree of tissue integration, which may be related to the preservation method and a reduction in the biological activity of the material.

UltraPro was characterized by minimal necrotic tissue damage and pronounced neovascularization; however, this was accompanied by a persistent macrophage-dominated inflammatory response. Such an immune profile is typical of polypropylene-based synthetic meshes and reflects prolonged foreign-body interaction rather than acute infectious tissue destruction [25,28].

It should be emphasized that systemic antibiotic therapy was deliberately excluded from the experimental protocol. This design choice was made to isolate and compare intrinsic tissue–material interactions under standardized bacterial exposure, minimizing pharmacological confounding factors.

We acknowledge that this approach does not replicate routine clinical practice, in which perioperative antibiotic prophylaxis is mandatory during contaminated surgery. Moreover, the bacterial inoculum used in this study (10^9 CFU/mL) exceeds the conventional threshold of surgical contamination and should be interpreted as a severe infection/abscess challenge model rather than a model of routine clinical contamination.

Consequently, the present experiment should be regarded as a comparative assessment of material behavior under extreme bacterial stress, aimed at revealing differential susceptibility of implant materials to early infection-related tissue damage. The translational relevance of the findings is therefore context-dependent and may be enhanced in future studies incorporating systemic antibiotic therapy and lower bacterial loads that more closely resemble clinical conditions.

Under conditions of severe experimental infection, the synthetic UltraPro mesh demonstrated superior early resistance to infection-associated tissue damage, as evidenced by significantly lower abscess and necrosis scores in the early postoperative period.

The xenoperitoneum-derived extracellular matrix showed increased susceptibility to early abscess formation and necrosis under high bacterial load, which may be related to its biological composition in the context of overwhelming infection. At later observation periods, ECM demonstrated signs of tissue remodeling and angiogenesis; however, its potential advantages appear to be context-dependent and may be more relevant under conditions of moderate contamination or in combination with systemic antibiotic therapy.

These findings highlight that the choice of implant material in contaminated or infected settings should consider both the severity of bacterial challenge and the specific phase of tissue response.

Limitations.

The non-infected control group was used for qualitative model validation rather than for quantitative morphometric comparison; therefore, statistical analyses were restricted to contaminated groups.

Study funding.

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Conflict of interest.

The authors declare no conflicts of interest to report.

Further research.

Overall, the obtained data indicate that the performance of xenoperitoneum-derived ECM under *E. coli* exposure is context-dependent: it is more susceptible to early abscess formation under severe bacterial challenge, yet demonstrates tissue remodeling and angiogenesis at later time points. Further studies using lower bacterial loads and systemic antibiotic regimens are warranted to better define potential clinical advantages of ECM-based implants.

Authors' contribution.

N. Urazbayev, R. Badyrov – concept and design of the study, editing.

R. Badyrov, N. Abatov – performance of surgical procedures, experimental modeling.

A. Lavrinenko – histological and morphometric analysis, collection and processing of material.

Y. Kamyshanskiy – pathological evaluation and interpretation of histological findings, editing.

I. Azizov – microbiological expertise, methodological consultation.

N. Urazbayev, R. Badyrov – writing the text, statistical processing.

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Аннотация.

Цель исследования: Сравнить тканевую реакцию на ксенобрюшинный внеклеточный матрикс (ECM), синтетическую сетку UltraPro и консервированную твёрдую мозговую оболочку в экспериментальной модели имплант-ассоциированной инфекции *Escherichia coli* у крыс.

Материалы и методы: У 42 крыс выполнена пластика дефекта передней брюшной стенки с использованием ECM, сетки UltraPro или твёрдой мозговой оболочки с интраоперационной контаминацией *E. coli*. Гистологическую и морфометрическую оценку проводили на 10-е и 20-е сутки после операции с анализом выраженности воспаления, абсцедирования, некроза, неоангиогенеза и тканевой интеграции.

Результаты: На 10-е сутки во всех группах отмечалось острое воспаление. Показатели абсцедирования и некроза были достоверно ниже при использовании сетки UltraPro по сравнению с ECM и твёрдой мозговой оболочкой ($p<0,05$). К 20-м суткам UltraPro сохраняла минимальные признаки тканевого повреждения и демонстрировала наиболее выраженные неоангиогенез и тканевую интеграцию, тогда как в группе твёрдой мозговой оболочки сохранялась воспалительная активность. В группе ECM отмечались снижение некроза и признаки тканевого ремоделирования.

Заключение: Синтетическая сетка UltraPro обладает большей устойчивостью к раннему инфекционно-обусловленному повреждению тканей, тогда как потенциальные преимущества ксенобрюшинного внеклеточного матрикса носят контекст-зависимый характер и проявляются на более поздних сроках.

Ключевые слова: внеклеточный матрикс ксенобрюшины; биологические имплантаты; синтетическая сетка; инфекция *Escherichia coli*; тканевая реакция; ангиогенез; экспериментальное исследование.

რეზიუმე.

მიზანი: შეადაროს ქსოვილოვანი რეაქცია კსენოპერიტონეუმისგანმიღებულექსტრაცელულარულ

მატრიქსზე (ECM), სინთეზურ ბადე UltraPro-ზე და კონსერვირებულ მყარ ტვინის გარსზე ვირთაგვებში იმპლანტთან ასოცირებული Escherichia coli ინფექციის ექსპერიმენტულ მოდელში.

მეთოდები: კვლევა ჩატარდა 42 ვირთაგვზე. წინა მუცლის კედლის დეფექტის პლასტიკა შესრულდა ECM-ის, UltraPro ბადის ან მყარი ტვინის გარსის გამოყენებით E. coli-ით ინტრაოპერაციული კონტამინაციის პირობებში. ჰისტოლოგიური და მორფომეტრიული შეფასება ჩატარდა ოპერაციის შემდეგ მე-10 და მე-20 დღეს, ანალიზით ანთების გამოხატულების, აბსცესის წარმოქმნის, ნეკროზის, ნეოანგიოგენეზისა და ქსოვილოვანი ინტეგრაციის მიხედვით.

შედეგები: მე-10 დღეს ყველა ჯგუფში აღინიშნებოდა მწვავე ანთებითი რეაქცია. აბსცესისა და ნეკროზის მაჩვენებლები სარწმუნოდ დაბალი იყო UltraPro ბადის გამოყენებისას ECM-სა და მყარ ტვინის გარსთან შედარებით ($p<0,05$). მე-20 დღისთვის UltraPro-ს ჯგუფში შენარჩუნებული იყო მინიმალური ქსოვილოვანი დაზიანება და გამოვლინდა ყველაზე გამოხატული ნეოანგიოგენეზი და ქსოვილოვანი ინტეგრაცია, მაშინ როდესაც მყარი ტვინის გარსის ჯგუფში ანთებითი აქტივობა კვლავ შენარჩუნდა. ECM-ის ჯგუფში აღინიშნებოდა ნეკროზის შემცირება და ქსოვილოვანი რემოდელირების ნიშნები.

დასკვნა: სინთეზური ბადე UltraPro გამოირჩევა უფრო მაღალი მდგრადობით ადრეული ინფექციით განპირობებული ქსოვილოვანი დაზიანების მიმართ, მაშინ როდესაც კსენოპერიტონეუმისგან მიღებული ექსტრაცელულარული მატრიქსის პოტენციური უპირატესობები კონტექსტზე დამოკიდებულია და უფრო გვიან პერიოდში ვლინდება.