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MICROORGANISM-DEPENDENT PHAGOCYTIC RESPONSES IN MURINE ALVEOLAR MACROPHAGES

Background. ***Macrophages are a highly heterogeneous immune cell subset that play essential roles in innate and adaptive immunity. Alveolar macrophages (AMs) represent the major resident immune cell population in the distal airways and constitute the first line of cellular defense against inhaled microorganisms. Phagocytosis is a fundamental function of AMs, supporting both pathogen clearance and lung homeostasis. However, the efficiency with which AMs internalize different classes of microorganisms remains incompletely explored, despite its relevance for early host–pathogen interactions in the lung. This study aimed to quantitatively compare the phagocytic activity of murine alveolar macrophages toward Gram-negative bacteria (Escherichia coli), Gram-positive bacteria (Staphylococcus aureus), and the yeast Candida albicans under non-opsonized, non-inflammatory in vitro conditions using flow cytometry.***

Methods. ***Murine AMs were isolated from BALB/c mice by bronchoalveolar lavage. Phagocytic activity was assessed using FITC-labeled, inactivated microorganisms, including heat-killed Staphylococcus aureus (Gram-positive), heat-killed Escherichia coli (Gram-negative), and ethanol-fixed Candida albicans. AMs were incubated with microbial targets for 30 minutes at 37 °C. Phagocytosis was quantified by flow cytometry as the percentage of FITC-positive cells and the phagocytic index, expressed as mean fluorescence intensity.***

Results. ***Analysis of AM phagocytic activity revealed pronounced target-dependent differences. Uptake was lowest for Escherichia coli, intermediate for Staphylococcus aureus, and highest for Candida albicans. This pattern was consistent for both the percentage of phagocytosing cells and the phagocytic index.***

Conclusions. ***Taken together, murine alveolar macrophages exhibit higher phagocytic activity toward Candida albicans than toward Gram-positive or Gram-negative bacteria under non-opsonized, non-inflammatory conditions. These results highlight pathogen-dependent differences in alveolar macrophage phagocytosis relevant to early innate immune defense in the lung.***

Keywords: ***alveolar macrophages, phagocytic activity, Staphylococcus aureus, Escherichia coli, Candida albicans.***

Background

Tissue-resident macrophages are essential immune sentinels and guardians of tissue health, performing key functions like immune surveillance, clearing debris, maintaining tissue homeostasis, orchestrating wound healing, promoting tissue development / remodeling, and initiating/resolving inflammation through cytokine secretion and antigen presentation, all while adapting to their specific organ niche (Davies et al., 2013). Each of these functions relies fundamentally on the phagocytic activity of tissue macrophages (Hirayama, Iida, & Nakase, 2017). The nature of the target strongly shapes a macrophage's phagocytic response. Features such as size, shape, stiffness, and surface ligands determine receptor engagement, cytoskeletal remodeling, downstream signaling, and the efficiency of phagosome formation, ultimately producing target-specific immune responses. Distinct classes of particles – such as bacteria, apoptotic cells, or parasites – activate different receptor sets and elicit characteristic cytoskeletal architectures, highlighting the dynamic interplay between macrophages and the objects they engulf.

Lower respiratory tract infections (LRTIs), including pneumonia, acute bronchitis, and bronchiolitis, remain a major cause of morbidity and mortality worldwide, accounting for millions of deaths annually (GBD 2021 Lower Respiratory Infections and Antimicrobial Resistance Collaborators, 2024; Li, Liu, M., & Liu, J., 2025). While bacterial pathogens such as *Streptococcus pneumoniae*, *Staphylococcus aureus*, and *Klebsiella pneumoniae* are the primary contributors to LRTI-related mortality, fungal infections, though less frequent, demonstrate disproportionately high fatality rates, particularly in immunocompromised individuals (Denning, 2024). Given that the lungs are continuously exposed to airborne microorganisms, effective innate immune surveillance in the distal airways is critical for preventing infection. Alveolar macrophages (AMs) are the most abundant resident immune cells in the alveoli and constitute the first line of cellular defense against inhaled pathogens. AMs originate from fetal precursors and can self-renew, requiring little contribution from circulating monocytes under healthy conditions; however, postnatal AMs originate from bone marrow hematopoiesis (Arafa et al., 2022). Phagocytosis

by these cells represents a fundamental component of the lung's defense, particularly against pathogens transmitted by airborne droplets. As AMs continuously survey the inhaled air, they are the earliest cells to detect, internalize, and destroy viruses, bacteria, and other particulate matter reaching the distal airways; this rapid response helps prevent the establishment of infection and substantially reduces pathogen burden before epithelial cells become compromised (Panahipoor Javaherdehi et al., 2024; Schneider et al., 2014). Beyond this defensive function, AMs play vital roles in maintaining tissue homeostasis by clearing surfactant and cellular debris, recognizing pathogens, and regulating both the onset and resolution of lung inflammation to support tissue repair. Under normal conditions, they maintain a balanced environment by generating low levels of inflammatory cytokines while exhibiting strong phagocytic capacity to restrain both inflammation and adaptive immune activation (Hu, & Christman, 2019; Mass et al., 2023). These cells possess a broad repertoire of phagocytic receptors, enabling them to recognize and internalize a diverse range of pathogenic microorganisms effectively. AMs utilize specific receptors (e.g., Scavenger Receptors like MARCO, TLR4 for lipopolysaccharide (LPS) of Gram-negative bacteria, and TLR2 for lipoteichoic acid (LTA) of Gram-positive bacteria) to recognize these, resulting in varied uptake rates and inflammatory signals (Hirayama, Iida, & Nakase, 2017). Our current understanding of the efficiency with which alveolar macrophages phagocytose various microorganisms remains limited, despite its crucial role in elucidating lung defense mechanisms, host susceptibility to disease, and the progression of infections into pathological conditions. This study aimed to quantitatively compare the phagocytic activity of murine alveolar macrophages toward Gram-negative bacteria (*Escherichia coli*), Gram-positive bacteria (*Staphylococcus aureus*), and the yeast *Candida albicans* under non-opsonized, non-inflammatory in vitro conditions using flow cytometry.

Methods

AM isolation. Alveolar macrophages were isolated from five male Balb/c mice aged 18–22 weeks. For each experimental group, three biological replicates were used, each derived from a different animal. Before experimentation, the animals were housed in the vivarium of the Educational and Scientific Center "Institute of Biology and Medicine" at Taras Shevchenko National University of Kyiv. They were maintained under standard conditions: 20 ± 2 °C, a 12-hour light/dark cycle, and unrestricted access to food and water. All procedures were conducted in accordance with approved ethical guidelines. The study protocol was reviewed and authorized by the University Bioethics Committee (protocol No. 4, 10.10.2022) in compliance with the Animal Protection Act. Experimental work adhered to the requirements of the Law of Ukraine No. 3447-IV "On the Protection of Animals from Cruelty," as well as the principles outlined in the Council of Europe's "European Convention for the Protection of Vertebrate Animals Used for Experimental and Other Scientific Purposes" (1997), the ethical standards established at the First National Congress on Bioethics of Ukraine (September 2001), and other relevant national and international regulations.

AM were collected using a bronchoalveolar lavage technique performed via orotracheal intubation, according to the protocol by Bruno Luckow and Michael H. Lehmann with minor adjustments (Luckow, & Lehmann, 2021). Before lavage, mice were humanely euthanized through an intraperitoneal overdose of anesthetics (ketamine 300–360 mg/kg and xylazine 30–40 mg/kg). For the intubation procedure, a straight, blunt-ended cannula equipped with an olive-shaped tip and a Luer lock connection (ACUFIRM Ernst Kratz GmbH, Berlin, Germany) was used. Bronchoalveolar lavage was performed with pre-warmed PBS containing 2 mM EDTA, maintained at 37 °C. To access the trachea, the ventral neck region was disinfected with ethanol, and a small incision was made. The salivary glands were gently displaced with forceps to expose the tracheal surface. A 1-mL syringe filled with lavage buffer was connected to the cannula, which was then carefully advanced through the oral cavity into the trachea. Once correctly positioned, the cannula was secured with a ligature placed behind the olive tip. A total of 0.6 mL of buffer was slowly infused into the lungs and then removed, and the recovered lavage fluid was collected in a sterile tube. This washing cycle was repeated three times. Following collection, cells were centrifuged and rinsed three times with PBS. Viable cell counts were determined using the trypan blue exclusion assay, and the viability consistently exceeded 95 %.

Microorganisms. Staphylococcus aureus (UCM B-918) and *Escherichia coli* (UCM B-926) were cultured in meat peptone agar at 37 °C for 24 h. Culture of *Candida albicans* (UCM Y-2681) was performed in Sabouraud broth at 35 °C and 220 rpm for 24 h. Suspensions of *S. aureus* and *E. coli* were obtained in PBS, heat-inactivated at 95 °C for 30 min, precipitated by centrifugation at 1000 g for 25 minutes, washed, and resuspended in carbonate–bicarbonate buffer (pH 9.5) at a concentration of 1×10^8 cells/mL. *C. albicans* cells were collected, precipitated by centrifugation at 450 g for 10 minutes washed in PBS, fixed in 70 % ethanol for 1 h, and similarly adjusted to 1×10^8 cells/mL in carbonate–bicarbonate buffer. Microbial suspensions were incubated with FITC, dissolved in DMSO (0.05 mg per 1×10^8 bacteria; 0.01 mg per 1×10^8 yeast cells) for 1 h at room temperature in the dark. After staining, microorganisms were washed three times in PBS, standardized to 2×10^8/mL (bacteria) or 8×10^7/mL (yeast) and stored at 4 °C.

Flow cytometry. Phagocytic activity of AM was evaluated using FITC-labeled microorganisms (*S. aureus, E. coli*, and *C. albicans*) as targets for endocytic uptake. For the phagocytosis reaction, AM (1×10^5) were mixed with FITC-labeled microbes and incubated for 30 min at 37 °C and 5 % CO_2. After incubation, cells were washed with PBS, fixed with 0.04 % paraformaldehyde, and analyzed on a DxFlex flow cytometer (Beckman Coulter, USA). Phagocytic activity was evaluated by determining the percentage of FITC-positive cells and the phagocytic index (mean fluorescence intensity). Data was collected on the DxFlex flow cytometer and processed with Kaluza C Analysis Software (Beckman Coulter, Inc., USA). The overall study design is illustrated in Fig. 1.

Statistical analysis. Data normality was evaluated using the Shapiro – Wilk test to ensure that the distribution of the measured variables met the assumptions required for parametric analysis (Mishra et al., 2019). All results are presented as the mean ± standard error of the mean (SEM). Statistical comparisons between experimental groups were carried out using a one-way ANOVA (Ntumi, 2021). When interpreting the outcomes, values of $p < 0.05$ were considered statistically significant.

Results

Analysis of phagocytic activity revealed clear differences in the AMs' ability to internalize the three tested microorganisms. The phagocytosis percentage was lowest for *Escherichia*

coli, higher for *Staphylococcus aureus*, and the highest for *Candida albicans* (Fig. 2).

A similar pattern was observed for the phagocytic index (MFI). AM incubated with both *E. coli* and *S. aureus* exhibited low MFI values. In contrast, in *C. albicans*, significantly higher fluorescence intensity was observed, indicating a substantially greater amount of internalized fungus per cell (Fig. 3).

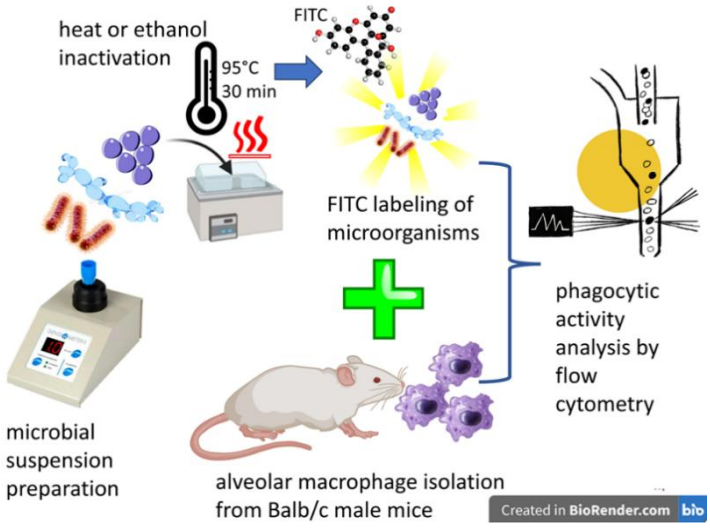

Fig. 1. Study design

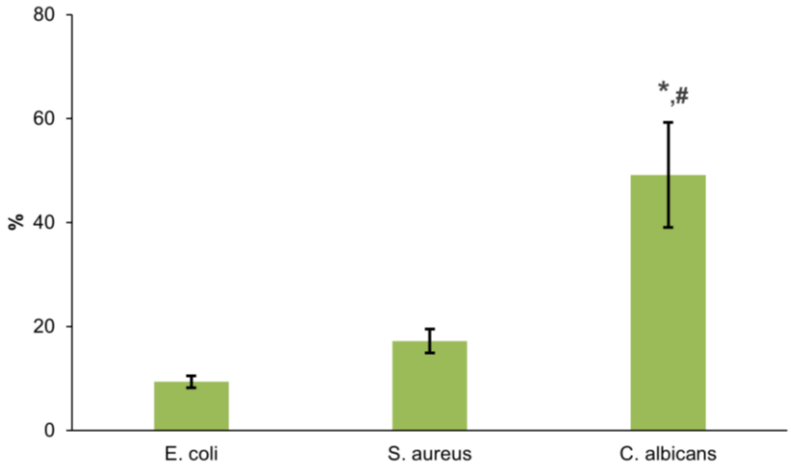

Fig. 2. Phagocytosis percentage of murine alveolar macrophages incubated with FITC-labeled *E. coli, S. aureus*, and *C. albicans*
(n = 3; **p* < 0.01 compared with *Escherichia coli*; #*p* < 0.05 compared to *Staphylococcus aureus*)

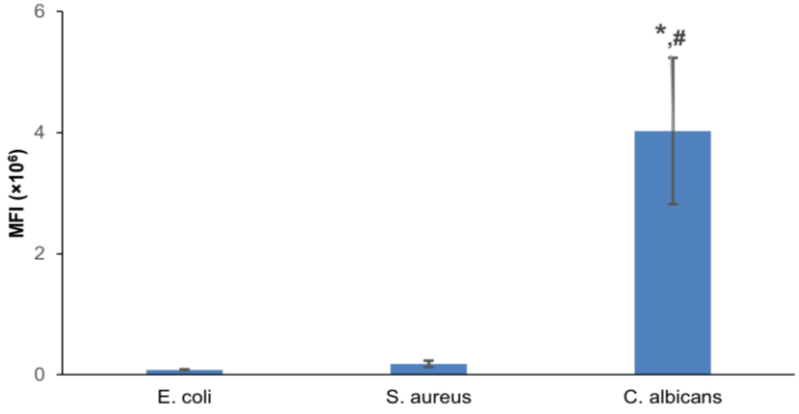

Fig. 3. Phagocytic index (MFI) of murine alveolar macrophages incubated with FITC-labeled *E. coli, S. aureus*, and *C. albicans*. MFI – mean fluorescence intensity
(n = 3; **p* < 0.05 compared to *Escherichia coli*; #*p* < 0.05 compared to *Staphylococcus aureus*)

Discussion and conclusions

Bacteria employ multiple immune-evasion strategies that limit phagocytic clearance, including masking of pathogen-associated molecular patterns and opsonins by capsules, as well as interference with phagosome maturation and phagolysosome formation (Gao et al., 2024; Lee et al., 2020; Uribe-Querol, & Rosales, 2017). By using inactivated microorganisms, the present study specifically assessed the intrinsic phagocytic capacity of alveolar macrophages, independent of active microbial evasion mechanisms.

Phagocytosis is initiated through the recognition of pathogen-associated molecular patterns by pattern recognition receptors expressed on macrophages, and differences in uptake efficiency likely reflect variations in receptor engagement (Mukhopadhyay, Plüddemann, & Gordon, 2009). Among these receptors, C-type lectin receptors (CLRs) play a central role in antifungal immunity. Macrophages express several CLRs, including Dectin-1, Dectin-2, the Mannose Receptor (CD206), and DC-SIGN, which efficiently recognize fungal cell wall components such as β-glucans and mannans (Patin, Thompson, & Orr, 2019). Signaling through Dectin-1 promotes phagocytosis, reactive oxygen species generation, cytokine release, inflammasome activation, and Th1/Th17 polarization (Geijtenbeek, & Gringhuis, 2009; 2016).

However, pathogen recognition by macrophages is not limited to CLRs. Toll-like receptors (TLRs), another major class of PRRs, detect a wide range of PAMPs and contribute substantially to microbial sensing. Gram-negative bacteria activate TLR4 via lipopolysaccharide, whereas Gram-positive bacteria and fungi primarily signal through TLR2 (Paul-Clark et al., 2006). Although TLR4 is constitutively expressed, TLR2 expression in murine alveolar macrophages is strongly inducible and upregulated only in response to inflammatory stimuli such as LPS, TNF-α, or IL-1β through p38 kinase and NF-κB signaling pathways (Oshikawa, & Sugiyama, 2003). Consequently, under the non-inflammatory conditions employed in this study, limited TLR2 expression likely constrained bacterial phagocytosis, particularly of *Staphylococcus aureus*.

Pulmonary surfactant components further modulate phagocytic signaling. Surfactant Protein A (SP-A) enhances the expression of TLR2 and the Mannose Receptor, thereby promoting the clearance of fungi and Gram-positive bacteria, while simultaneously facilitating the degradation of TLR4 (Henning et al., 2008; Freundt et al., 2022). This regulatory pattern is consistent with our findings, which demonstrate the lowest phagocytic efficiency toward Gram-negative bacteria compared with microorganisms recognized predominantly via TLR2 and CLRs.

Our results differ from those of studies employing live bacteria, which often report enhanced uptake of Gram-negative species. For example, *Dictyostelium discoideum*, a commonly used model for studying phagocytosis, preferentially migrates toward Gram-negative bacteria (Rashidi, & Ostrowski, 2019), and Gram-negative pathogens induce dendrite-like pseudopod formation in macrophages via LPS-dependent mechanisms (Fan et al., 2025). However, such responses require prolonged stimulation (1–2 h) and are more pronounced in less differentiated monocytic cell models, such as THP-1 cells, than in resident tissue macrophages. In the present study, fully differentiated murine alveolar macrophages were exposed to microorganisms for only 30 minutes, which likely limited the contribution of these mechanisms.

The high efficiency of *Candida albicans* uptake observed here may contribute to the relatively low incidence of invasive pulmonary candidiasis in immunocompetent hosts (Arana et al., 2025). Nevertheless, extrapolation to humans should be made with caution, as mice exhibit low susceptibility to *C. albicans* colonization, whereas *Candida* spp. commonly colonize the human respiratory tract without causing disease (Ader et al., 2011; Pendleton, Huffnagle, & Dickson, 2017).

Taken together, our results show that murine alveolar macrophages exhibit significantly higher phagocytic activity toward *Candida albicans* than toward Gram-positive or Gram-negative bacteria under non-opsonized, non-inflammatory conditions. This pattern is consistent with preferential engagement of fungal recognition pathways, particularly C-type lectin receptors, in resting alveolar macrophages. The comparatively low bacterial uptake in this model suggests that efficient antibacterial phagocytosis requires additional signals, such as inflammatory priming or opsonization. Overall, this study delineates pathogen-specific differences in phagocytic efficiency relevant to early innate pulmonary defense.

Authors' contribution: Sofia Suslova: investigation, writing – original draft; Nataliia Steniakina: investigation, writing – original draft; Roman Dovhyi: writing – review & editing; Tetiana Babich: formal analysis; Yuliia Yumyna: conceptualization.

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МІКРООРГАНІЗМ-ЗАЛЕЖНА ФАГОЦИТАРНА АКТИВНІСТЬ АЛЬВЕОЛЯРНИХ МАКРОФАГІВ МИШЕЙ

В с т у п . ***Макрофаги – це фенотипово та функціонально гетерогенні клітини, що відіграють ключову роль у реакціях вродженого й адаптивного імунітету. Альвеолярні макрофаги (АМ) є основною популяцією резидентних імунних клітин у дистальних відділах дихальних шляхів і становлять першу лінію клітинного захисту від мікроорганізмів, що потрапляють у дихальні шляхи при вдиханні. Фагоцитоз є фундаментальною функцією АМ, що забезпечує як видалення патогенів, так і підтримання гомеостазу легень. Проте ефективність, з якою АМ поглинають різні класи мікроорганізмів, досі вивчена недостатньо, незважаючи на її значення для раннього розпізнавання патогенів у легенях. Метою цього дослідження було за допомогою проточної цитофлуориметрії кількісно порівняти фагоцитарну активність альвеолярних макрофагів мишей щодо грамнегативних бактерій (Escherichia coli), грампозитивних бактерій (Staphylococcus aureus) і дріжджоподібного гриба Candida albicans в умовах in vitro без опсонізації та запальної стимуляції.***

М е т о д и . ***АМ мишей лінії BALB/c були виділені шляхом отримання бронхоальвеолярного лаважу. Фагоцитарну активність оцінювали, використовуючи мічені FITC інактивовані мікроорганізми, зокрема інактивований нагріванням Staphylococcus aureus (грампозитивна бактерія), інактивовану нагріванням Escherichia coli (грамнегативна бактерія) та інактивовану спиртом Candida albicans. АМ інкубували з мікроорганізмами 30 хв при 37 °C. Фагоцитоз кількісно оцінювали методом проточної цитометрії за відсотком FITC-позитивних клітин і фагоцитарним індексом, вираженим як середня інтенсивність флуоресценції.***

Р е з у л ь т а т и . ***Аналіз фагоцитарної активності АМ показав виражену залежність від типу мікроорганізму. Найнижчий рівень поглинання спостерігався в Escherichia coli, проміжний – у Staphylococcus aureus, а найвищий – у Candida albicans. Така закономірність спостерігалася як для відсотка фагоцитуючих клітин, так і для фагоцитарного індексу.***

В и с н о в к и . ***В умовах in vitro без опсонізації та запальної стимуляції альвеолярні макрофаги мишей ефективніше фагоцитують Candida albicans, ніж грампозитивні або грамнегативні бактерії. Ці результати підкреслюють відмінності у фагоцитарній активності альвеолярних макрофагів, що залежать від виду патогену та мають значення для раннього вродженого імунного захисту легень.***

К л ю ч о в і с л о в а : ***альвеолярні макрофаги, фагоцитарна активність, Staphylococcus aureus, Escherichia coli, Candida albicans.***

Автори заявляють про відсутність конфлікту інтересів. Спонсори не брали участі в розробленні дослідження; у зборі, аналізі чи інтерпретації даних; у написанні рукопису; в рішенні про публікацію результатів.

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