



Cite as: ZIELNIK, Martyna, STRÓŻNA, Katarzyna, BŁĄŻOWSKA, Olga, JUREWICZ, Alicja, GUTOWSKA, Oliwia, NOWAK, Weronika, KOSTUCH, Wiktoria, KOWALEWSKI, Łukasz, STYCZYŃSKA, Maria and NAZIRI, Amir. The role of the gut-skin axis in the pathogenesis of rosacea. *Quality in Sport*. 2026;61:73044. <https://doi.org/10.12775/QS.2026.61.73044>

ARTICLE TIMELINE

Received: 04.06.2026. Revised: 03.07.2026. Accepted: 04.07.2026. Published: 07.07.2026.

The journal has been awarded 20 points in the parametric evaluation by the Polish Ministry of Higher Education and Science (Annex to the announcement of 05.01.2024, No. 32553). Unique Journal Identifier: 201398. Scientific disciplines assigned: Economics and finance (Field of social sciences); Management and Quality Sciences (Field of social sciences).

Punkty Ministerialne z 2019 – aktualny rok 20 punktów. Załącznik do komunikatu Ministra Szkolnictwa Wyższego i Nauki z dnia 05.01.2024 Lp. 32553. Posiada Unikatowy Identyfikator Czasopisma: 201398. Przypisane dyscypliny naukowe: Ekonomia i finanse (Dziedzina nauk społecznych); Nauki o zarządzaniu i jakości (Dziedzina nauk społecznych). © The Authors 2026.

OPEN ACCESS · CC BY-NC-SA 4.0 This article is published with open access under the License Open Journal Systems of Nicolaus Copernicus University in Toruń, Poland, and is distributed under the terms of the Creative Commons Attribution Non-commercial Share Alike License (<http://creativecommons.org/licenses/by-nc-sa/4.0/>), which permits unrestricted non-commercial use, distribution, and reproduction in any medium, provided the work is properly cited. The authors declare no conflict of interest regarding the publication of this paper.

The role of the gut-skin axis in the pathogenesis of rosacea

1. Martyna Zielnik <https://orcid.org/0009-0007-9787-0625>

The John Paul's II Western Hospital in Grodzisk Mazowiecki, Poland

ul. Daleka 11, 05-825 Grodzisk Mazowiecki

e-mail: martynazie@icloud.com

2. Katarzyna Stróżna <https://orcid.org/0009-0007-7614-1108>

Medical Center of Postgraduate Education, Professor Witold Orłowski Independent Public Clinical Hospital, Warszawa, Poland

Czerniakowska 231, 00-416 Warszawa

e-mail: katarzyna.stroznal@gmail.com

3. Olga Błażowska

<https://orcid.org/0009-0003-6989-3062>

Wolski Hospital, Warsaw, Poland

Marcina Kasprzaka 17, 01-211 Warszawa

e-mail: olga.blazowska@gmail.com

4. Alicja Jurewicz <https://orcid.org/0009-0002-4834-1499>

Independent Public Health Care Institution of the Ministry of Interior and Administration in Gdańsk, Poland Kartuska 4/6, 80-104 Gdańsk

e-mail: alajurewicz@gmail.com

5. Oliwia Barbara Gutowska
<https://orcid.org/0009-0000-7378-7383>
6th year medical student
Medical University of Gdańsk, Poland
ul. Marii Skłodowskiej-Curie 3a, 80-210 Gdańsk
e-mail: oliwia.gutowska.01@gmail.com

6. Weronika Nowak
<https://orcid.org/0009-0009-4325-4602>
6th year medical student
Medical University of Gdańsk, Poland
ul. Marii Skłodowskiej-Curie 3a, 80-210 Gdańsk
e-mail: weronikanowak@gumed.edu.pl

7. Wiktoria Magdalena Kostuch
<https://orcid.org/0009-0003-8084-0551>
6th year medical student
Medical University of Gdańsk, Poland
ul. Marii Skłodowskiej-Curie 3a, 80-210 Gdańsk
e-mail: wiktoriakostuch@gumed.edu.pl

8. Łukasz Kowalewski, ORCID <https://orcid.org/0009-0008-4005-2610>
University Clinical Centre, Gdańsk, Poland
ul. Dębinki 7, 80-952 Gdańsk
e-mail: lek.lukasz.kowalewski@gmail.com

9. Maria Aleksandra Styczyńska, ORCID <https://orcid.org/0009-0005-3583-1271>
University Clinical Centre, Gdańsk, Poland
ul. Dębinki 7, 80-952 Gdańsk
E-mail: mstyczynska00@gmail.com
F-

10. Amir Naziri, ORCID <https://orcid.org/0009-0009-4465-1685>
University Clinical Centre, Gdańsk, Poland
ul. Dębinki 7, 80-952 Gdańsk
E-mail: amirnaziri@o2.pl

Corresponding Author

Martyna Zielnik e-mail: martynazie@icloud.com

ABSTRACT

BACKGROUND AND AIM

Rosacea is a chronic inflammatory skin condition affecting the central facial region, characterized by erythema, papulopustules, telangiectasia, and flushing. The aetiology of this disease is multifactorial, involving immune dysregulation, neurovascular instability, and environmental triggers. The aim of this narrative review was to analyze the role of the gut-skin axis in the pathogenesis of rosacea, with particular emphasis on gut microbiota, immune mechanisms, and the potential of dietary and microbiome-targeted therapeutic interventions.

MATERIAL AND METHODS

A narrative review of current scientific literature was conducted using the MEDLINE (PubMed) database. Original studies, systematic reviews, meta-analyses, and randomized controlled trials published primarily between 2007 and 2025 were included, using search terms including "rosacea," "gut-skin axis," "gut microbiota," "dysbiosis," "SIBO," "Helicobacter pylori," "short-chain fatty acids," "diet," and "probiotics."

RESULTS

Dysbiosis, reduced SCFA production, and increased intestinal permeability promote systemic inflammation and cutaneous immune activation in rosacea. A significant association between rosacea and gastrointestinal disorders, particularly SIBO and *H. pylori* infection, was confirmed. SIBO eradication with rifaximin achieved lesion resolution in 78% of rosacea patients. Mediterranean dietary patterns demonstrated protective effects.

CONCLUSIONS

The gut-skin axis is a significant component of rosacea pathogenesis. Dietary modification, probiotic supplementation, and targeted eradication of SIBO and *H. pylori* represent promising adjunctive therapeutic strategies. Further randomized controlled trials are required.

KEY WORDS: Rosacea, gut-skin axis, gut microbiota, diet, dysbiosis, SIBO, *H. pylori*, short-chain fatty acids, probiotics

INTRODUCTION

DEFINITION OF ROSACEA

Rosacea is a chronic inflammatory skin condition affecting the central facial region. Rosacea can be difficult to recognize, and diagnosis may take several months due to its nonspecific symptoms. The first, often inconspicuous skin changes are recurrent facial erythema. Recurrent facial flushing may occur when consuming hot or spicy foods, when drinking alcohol, and when exposed to high ambient temperatures [1]. Between episodes of erythema, the skin appears normal.

Currently, there are four subtypes of rosacea: erythematous with telangiectasia (the most common subtype), papulopustular, hypertrophic, and ocular. Subtypes may occur simultaneously or change in severity depending on the progression of the disease [2]. In fact, rosacea is a multiphase disease that includes 4 phases: flushing, erythrosis, papulopustules, and phymata. Whereas flushing and erythrosis are common to most patients, the papulopustular and phyma phases occur, often on an erythrotic background, only in a minority of patients, suggesting that diverse etiopathogenetic factors, specific for each phase, are at work [3,4].

EPIDEMIOLOGY

The incidence rate is estimated at 2-10%, The exact prevalence is difficult to estimate due to diagnostic challenges. It favors adults between 30 and 50 years of age, affects females more than males, and is more commonly diagnosed in individuals with fair skin (phototypes I and II), affecting more than 10% of Whites [5]. Men are less likely to develop the condition, but when they do, it tends to be more severe, and they are more likely to have the hypertrophic form of rosacea [2].

PATHOPHYSIOLOGY OF ROSACEA

The causes of rosacea are multifactorial. Among other things, the causes of the lesions include hyperreactivity of the skin's blood vessels, immune system disorders, and environmental factors [2,6]. In this report, I will focus on discussing immunological and environmental factors.

IMMUNOLOGICAL MECHANISMS

Our immune system plays a very significant role in the pathogenesis of rosacea.

In the affected skin, inflammatory infiltrates consisting of type 1 and 17 helper T cells can be observed. Additionally, an increased number of cells such as macrophages and mast cells is evident, and neutrophils are the most abundant in cases of papulopustular rosacea. There is also evidence of plasma cell activation with significant antibody production as early as the ETR (erythematotelangiectatic rosacea) phase, followed by an increase in severity toward nodular rosacea [7,8].

Many studies have shown that the innate immune system is aberrantly activated by some skin microorganisms through Toll-like receptor 2 (TLR 2). Increased TLR2 expression may lead to abnormal production of antimicrobial peptides, as well as to increased expression and activity of serine kallikrein (KLK-5) [9]. Increased activity of serine protease kallikrein 5 (KLK5) leads to proteolytic cleavage of the cathelicidin precursor hCAP18 into its active form LL-37, which exhibits pro-inflammatory and pro-angiogenic properties in rosacea [10].

ENVIROMENTAL FACTORS

As mentioned above, factors that can exacerbate the symptoms of rosacea include hot spices, alcohol consumption, prolonged exposure to high temperatures and sun, also microorganisms that live on our skin. Spicy foods containing capsaicin and hot beverages activate transient receptor potential (TRP) channels, particularly TRPV1, leading to neurovascular dysregulation, vasodilation, and flushing [8, 28]. Alcohol, particularly red wine, promotes vasodilation through prostaglandin release and additionally contains histamine and sulfites that compound the inflammatory response.

Microorganisms include, amongst others, *Demodex folliculorum*, *Bacillus oleronius*, *Helicobacter pylori*, *Staphylococcus epidermidis* and *Chlamydia pneumoniae* [2]. The presence of microorganisms triggers an inflammatory response in our body, causing blood vessels to dilate and inflammatory cells to migrate to the site.

According to the study “Genetic vs Environmental Factors That Correlate with Rosacea: A Cohort-Based Survey of Twins” the most significant factor influencing the onset of symptoms was UV radiation [11].

GUT-SKIN AXIS

In recent years, there has been increasing discussion about the importance of the gut-skin axis in the pathogenesis of rosacea.

The gut-skin axis represents a conceptual framework that connects gastrointestinal function with skin health and has been increasingly implicated in the development of chronic

inflammatory conditions. Cutaneous homeostasis and adaptive responses are strongly influenced by gastrointestinal status through intricate interactions involving the immune, metabolic, and neural systems [9, 12]. Within this axis, the gut microbiota plays a pivotal role, as it modulates host immune responses in a bidirectional manner and helps maintain systemic equilibrium [13].

The major products from the good working microbial fermentative activity in the gut are SCFAs, in particular: acetate, propionate, and butyrate [14].

Short-chain fatty acids (SCFAs) have beneficial effects on the body; among other things, they regulate glucose and lipid metabolism, nourish colonocytes and reduce inflammation [15]. In cases of intestinal dysbiosis, the production of short-chain fatty acids (SCFAs), such as butyrate, propionate and acetate, is significantly reduced.

Short-chain fatty acids, particularly butyrate, enhance intestinal barrier integrity by promoting tight junction assembly via AMPK activation, while also exerting anti-inflammatory effects; disruption of these mechanisms may increase gut permeability and systemic inflammation, potentially contributing to the development or exacerbation of rosacea [16].

GUT MICROBIOTA

EUBIOSIS

“Eubiosis” is a word derived from Greek. It can be broken down into two parts: “eu” means “good,” and “bios” means “life”; thus, eubiosis means “good life” or “what is good for life.” Gut eubiosis is a state of balance in the gut ecosystem in which the composition and functions of the microbiota are optimal for the host organism. It is characterized by: high microbial diversity, a predominance of protective bacteria, preserved integrity of the intestinal barrier, and proper modulation of the immune system [18]. It is impossible to compile a list of bacteria that have only positive effects on our bodies. The microbiome is an ecosystem, so the same bacterium can be beneficial in one context and harmful in another; moreover, what matters is the proportion, diversity, and function, not merely the presence of the bacteria. It is also worth noting that many bacteria are so-called pathobionts—normally harmless, but problematic when present in excess [19].

Factors influencing healthy gut microbiota include diet, infrequent use of antibiotics, high levels of physical activity, and limiting alcohol consumption and smoking. That is why it is so important to minimize factors that have a negative impact on gut microbiota. This is why it is so important to minimize factors that negatively impact the microbiota.

DYSBIOSIS

“Dysbiosis” is a term derived from Greek, where “dys” means “bad,” “difficult,” or “disordered,” and “bios” means “life”; thus, dysbiosis can be understood as a state of “disordered life”. In the context of the gut, dysbiosis refers to a disturbance of the intestinal ecosystem in which both the composition and functional activity of the microbiota are altered, potentially leading to adverse effects on the host organism [20]. It is characterized by reduced microbial diversity, a decline in beneficial commensal bacteria, and an increased abundance of opportunistic or potentially pathogenic microorganisms, including pathobionts, often accompanied by disruption of intestinal barrier integrity and altered immune responses [19]. Dysbiosis is therefore not defined by the presence of a single pathogenic species, but rather by an imbalance in the overall structure and function of the microbial community, which may promote inflammatory processes and contribute to disease development [20]. These alterations may contribute to chronic low-grade systemic inflammation, which is a key element in the pathogenesis of rosacea. Dysbiosis may also impair intestinal barrier function, leading to increased permeability and translocation of microbial products such as lipopolysaccharides (LPS) into the circulation [9].

Furthermore, disturbances in gut microbiota are associated with reduced production of short-chain fatty acids (SCFAs), which normally exert protective and anti-inflammatory effects. A decrease in SCFA levels may therefore contribute to both intestinal and systemic immune dysregulation [14,15].

CHANGES IN BACTERIAL COMPOSITION

Recent studies have demonstrated that rosacea is associated with specific alterations in gut microbial composition [9,12]. Typical findings include decreased levels of *Bifidobacterium* and *Lactobacillus* and increased presence of pro-inflammatory bacterial species, including *Helicobacter Pylori* [21,23,27].

Microbial metabolites produced in the gut may act as signaling molecules that influence distant organs, including the skin. This supports the hypothesis that gut microbiota plays a systemic role in rosacea development, extending beyond the gastrointestinal tract [9].

Disturbances in both the composition and relative abundance of the microbiome observed in patients with rosacea have prompted the incorporation of microbiome-targeted strategies into therapeutic approaches. These strategies include the administration of probiotics, defined as selected strains of beneficial bacteria or yeasts that facilitate the

restoration of microbial homeostasis, as well as prebiotics, which function as substrates that selectively promote the growth and activity of such microorganisms. Furthermore synbiotics, combinations of probiotics and prebiotics, are increasingly employed, particularly within dermatological and cosmetic contexts [21, 22].

SIBO AND GASTROINTESTINAL DISORDERS

An increased incidence of gastrointestinal disorders, including SIBO and other inflammatory bowel diseases, has been observed among patients with rosacea. SIBO is small intestine bacterial overgrowth. This is a condition in which there is an increased number of bacteria in the small intestine. This condition can lead to digestive and absorption problems. An excessive build-up of sugars in the intestines, combined with a drop in pH, can lead to bowel movement problems, flatulence and a feeling of bloating. An increased amount and activity of bacteria in the intestines can cause a rise in ammonia levels, which damages the intestinal mucosa. [24]

H. pylori infection is often found in patients with rosacea. The bacteria cause an overproduction of nitric oxide, which dilates the blood vessels and may exacerbate the symptoms of rosacea [26]. Bacterial strains that possess the *cagA* gene (*cagA*⁺) are more dangerous than those that do not. The CagA protein disrupts normal tissue function and increases inflammation. During inflammation of the gastric mucosa, gastric acid secretion is reduced, leading to an increased number of bacteria in the intestines. This results in the development of SIBO, which also exacerbates inflammation [25].

DIET AND TREATMENT

Diet plays a key role both in regulating the gut microbiota and in exacerbating the symptoms of rosacea. A carefully chosen diet can help patients extend periods of remission. Avoiding foods that trigger redness, such as hot spices, alcohol or hot meals, can reduce the number of flare-ups [1].

Furthermore, ensuring the gut microbiota is properly nourished can reduce inflammation in the body and thus aid in the treatment of rosacea. Diets rich in refined sugars, processed foods, and saturated fats may additionally contribute to gut dysbiosis and reduce short-chain fatty acid (SCFA) production, thereby promoting systemic inflammation and compromising intestinal barrier integrity, mechanisms already discussed in the context of the gut-skin axis [29].

To support our gut microbiota, it is important to follow a suitable diet. The Mediterranean diet- characterized by high intake of olive oil, fatty fish, vegetables, legumes, whole grains, and polyphenol-rich foods, alongside low consumption of red meat and processed foods, promotes gut microbiome diversity, enhances SCFA production, and reduces systemic inflammatory markers, all of which are pathways directly relevant to rosacea pathophysiology [30].

Fiber-rich and prebiotic foods, including whole grains, legumes, chicory, and garlic, selectively promote the growth of beneficial bacteria such as *Lactobacillus* and *Bifidobacterium*, supporting intestinal barrier integrity through butyrate-mediated tight junction assembly. [16, 28].

In addition to following a suitable diet, pharmacological treatment may be helpful in some cases. In patients diagnosed with SIBO, treatment with rifaximin yields good results and results in the complete healing of skin lesions in 78% of participants. [30]. It is a broad-spectrum antibiotic that is absorbed into the bloodstream to a minimal extent and acts locally within the gastrointestinal tract. [31].

Patients with *H. pylori* infection also benefit from drug treatment. The standard eradication regimen in Poland is the so-called quadruple therapy with bismuth: PPI + bismuth + metronidazole + tetracycline [24]. In this case, too, a reduction in the number of skin lesions was observed in patients [26].

CONSLUSION

Rosacea is a multifactorial chronic inflammatory disease whose pathogenesis extends beyond the skin to encompass complex bidirectional interactions between the gastrointestinal tract and the cutaneous immune system.

The gut-skin axis represents an increasingly well-supported conceptual and mechanistic framework for understanding these interactions. Dysbiosis, reduced SCFA production, increased intestinal permeability, and translocation of microbial products into systemic circulation collectively contribute to the immune dysregulation and neurovascular instability characteristic of rosacea. The strong epidemiological and clinical association between rosacea and gastrointestinal conditions, particularly SIBO and *H. pylori* infection, alongside the therapeutic benefit observed following their eradication, provides compelling evidence that the gut is not merely a comorbid site but an active contributor to rosacea pathogenesis.

Dietary modification, including adherence to anti-inflammatory dietary patterns such as the Mediterranean diet, avoidance of known neurovascular triggers, and targeted supplementation with prebiotics, and probiotics, represents a promising adjunctive therapeutic strategy that addresses both gut microbiome health and systemic inflammation simultaneously.

Pharmacological targeting of the gut, through rifaximin in SIBO, positive patients and bismuth-based quadruple eradication therapy in *H. pylori*-positive patients, may yield significant dermatological benefit in appropriately selected individuals.

However, the evidence base in this field retains important limitations. The majority of studies demonstrating the rosacea-gut connection are observational or mechanistic in design, and robust randomized controlled trials specifically evaluating gut-targeted interventions as rosacea therapies remain scarce. Furthermore, significant interindividual variability in trigger responses and microbiome composition complicates the generalization of dietary and probiotic recommendations. Future research should prioritize well-designed, placebo-controlled trials investigating the clinical efficacy of dietary interventions, probiotic supplementation, and SIBO eradication on validated rosacea severity outcomes, as well as longitudinal studies characterizing gut microbiome dynamics in rosacea across subtypes and disease phases.

DISCLOSSURE

Author's contribution:

Conceptualization: Martyna Zielnik, Olga Błażowska, Weronika Nowak

Methodology: Martyna Zielnik, Katarzyna Stróżna, Maria Styczyńska

Investigation: Martyna Zielnik, Alicja Jurewicz, Amir Naziri

Data curation: Martyna Zielnik, Łukasz Kowalewski, Wiktoria Kostuch

Writing - rough preparation: Martyna Zielnik, Oliwia Gutowska, Maria Styczyńska

Writing - review and editing: Olga Błażowska, Katarzyna Stróżna, Amir Naziri

Visualization: Alicja Jurewicz, Wiktoria Kostuch, Łukasz Kowalewski

Supervision: Martyna Zielnik, Oliwia Gutowska, Weronika Nowak

Software: Martyna Zielnik, Wiktoria Kostuch, Alicja Jurewicz

Check: Maria Styczyńska, Amir Naziri, Łukasz Kowalewski

Formal analysis: Weronika Nowak, Oliwia Gutowska, Katarzyna Stróżna

Resources: Olga Błażowska, Alicja Jurewicz, Amir Naziri

Project administration: Wiktoria Kostuch, Łukasz Kowalewski, Martyna Zielnik

Receiving funding: Maria Styczyńska, Olga Błażowska, Katarzyna Stróżna

Funding: The study did not receive external funding.

Institutional Review Board Statement: Not applicable.

Informed Consent Statement: Not applicable.

Data Availability Statement: Not applicable.

Acknowledgements: Not applicable.

Conflicts of Interest: The authors declare no conflicts of interest.

All authors have read and agreed with the published version of the manuscript.

AI.

AI was utilized for two specific purposes in this research. Text analysis of clinical reasoning narratives to identify linguistic patterns associated with specific logical fallacies. Assistance in refining the academic English language of the manuscript, ensuring clarity, consistency, and adherence to scientific writing standards. **AI** were used for additional linguistic refinement of the research manuscript, ensuring proper English grammar, style, and clarity in the presentation of results. It is important to emphasize that all AI tools were used strictly as assistive instruments under human supervision. The final interpretation of results, classification of errors, and conclusions were determined by human experts in clinical medicine and formal logic. The AI tools served primarily to enhance efficiency in data processing, pattern recognition, and linguistic refinement, rather than replacing human judgment in the analytical process.

REFERENCES:

1. Skin color: a practical guide to dermatologic diagnosis and treatment. Springer, New York, NY; 2013:21-36.
2. Dermatologia współczesna Vol. 2 L. Rudnicka, M. Olszewska, A. Rakowska, M. Sar-Pomian Chapter 67 Rosacea Pages 26-29
3. Powell FC. Clinical practice: rosacea. *N Engl J Med.* 2005;352(8):793-803. doi:10.1056/NEJMcp041894
4. Crawford GH, Pelle MT, James WD. Rosacea: I -etiology, pathogenesis, and subtype classification. *J Am Acad Dermatol.* 2004;51(3):327-341. doi:10.1016/j.jaad.2004.03.030

5. van Zuuren EJ. Rosacea. *N Engl J Med*. 2017;377(18):1754-1764. doi:10.1056/NEJMcp1506630
6. Gallo RL, Granstein RD, Kang S, Mannis M, Steinhoff M, Tan J, et al. Standard classification and pathophysiology of rosacea: the 2017 update by the National Rosacea Society Expert Committee. *J Am Acad Dermatol*. 2018;78(1):148-155. doi:10.1016/j.jaad.2017.08.037
7. Buhl T, Sulk M, Nowak P, Buddenkotte J, McDonald I, Aubert J, et al. Molecular and morphological characterization of inflammatory infiltrate in rosacea reveals activation of Th1/Th17 pathways. *J Invest Dermatol*. 2015;135(9):2198-2208. doi:10.1038/jid.2015.141
8. Schwab VD, Sulk M, Seeliger S, Nowak P, Aubert J, Mess C, et al. Neurovascular and neuroimmune aspects in the pathophysiology of rosacea. *J Invest Dermatol Symp Proc*. 2011;15(1):53-62. doi:10.1038/jidsymp.2011.6
9. Zhu W, Hamblin MR, Wen X. Role of the skin microbiota and intestinal microbiome in rosacea. *Front Microbiol*. 2023;14:1106253. doi:10.3389/fmicb.2023.1106253
10. Yamasaki K, Di Nardo A, Bardan A, Murakami M, Ohtake T, Coda A, et al. Increased serine protease activity and cathelicidin promotes skin inflammation in rosacea. *Nat Med*. 2007;13(8):975-980. doi:10.1038/nm1616
11. Aldrich N, Gerstenblith M, Fu P, et al. Genetic vs environmental factors that correlate with rosacea: a cohort-based survey of twins. *JAMA Dermatol*. 2015;151(11):1213-1219. doi:10.1001/jamadermatol.2015.2230
12. Daou H, Paradiso M, Hennessy K, Seminario-Vidal L. Rosacea and the microbiome: a systematic review. *Dermatol Ther (Heidelb)*. 2021;11(1):1-12. doi:10.1007/s13555-020-00460-1
13. Hill C, Guarner F, Reid G, Gibson GR, Merenstein DJ, Pot B, et al. The International Scientific Association for Probiotics and Prebiotics consensus statement on the scope and appropriate use of the term probiotic. *Nat Rev Gastroenterol Hepatol*. 2014;11(8):506-514. doi:10.1038/nrgastro.2014.66
14. Cummings JH, Pomare EW, Branch WJ, Naylor CP, Macfarlane GT. Short chain fatty acids in human large intestine, portal, hepatic and venous blood. *Gut*. 1987;28(10):1221-1227. doi:10.1136/gut.28.10.1221
15. Park JS, Lee EJ, Lee JC, Kim WK, Kim HS. Anti-inflammatory effects of short chain fatty acids in IFN- γ -stimulated RAW 264.7 murine macrophage cells: involvement of

- NF- κ B and ERK signaling pathways. *Int Immunopharmacol.* 2007;7(1):70-77. doi:10.1016/j.intimp.2006.08.015
16. Peng L, Li ZR, Green RS, Holzman IR, Lin J. Butyrate enhances the intestinal barrier by facilitating tight junction assembly via activation of AMP-activated protein kinase in Caco-2 cell monolayers. *J Nutr.* 2009;139(9):1619-1625. doi:10.3945/jn.109.104638
 17. Chen Y, Shi Y, Chen Y, Li Y, Zhang Y. Gut microbiota dysbiosis in rosacea: a potential role in disease pathogenesis. *Front Microbiol.* 2021;12:686304. doi:10.3389/fmicb.2021.686304
 18. Gałęcka M. Dysbioza jelitowa. Wydawnictwo Lekarskie PZWL; 2021
 19. Human Microbiome Project Consortium. Structure, function and diversity of the healthy human microbiome. *Nature.* 2012;486(7402):207-214. doi:10.1038/nature11234
 20. Saleem MM, Masood S, Rahmatullah MM, Imdad IA, Sange AMA, Nasr D. Gut microbiota dysbiosis and its role in the development of irritable bowel syndrome. *Cureus.* 2023;15(5):e39786. doi:10.7759/cureus.39786
 21. Yu J, Duan Y, Zhang M, Li Q, Cao M, Song W, et al. Effect of combined probiotics and doxycycline therapy on the gut-skin axis in rosacea. *Front Microbiol.* 2022;13:1034258. doi:10.3389/fmicb.2022.1034258
 22. Manzhali E, Hornuss D, Stremmel W. Intestinal-borne dermatoses significantly improved by oral application of *Escherichia coli* Nissle 1917. *World J Gastroenterol.* 2016;22(23):5415-5421. doi:10.3748/wjg.v22.i23.5415
 23. Li M, Fu J, Wei J, Wan M, Li L, Kong M, et al. Correlations between gut microbiota and serum metabolomics in patients with neurogenic rosacea. *Front Microbiol.* 2023;14:1125973. doi:10.3389/fmicb.2023.1125973
 24. Szczeklik A, Gajewski P, eds. *Interna Szczeklika*. Medycyna Praktyczna; 2023.
 25. Keikha M, Sahebkar A, Yamaoka Y, Karbalaee M. *Helicobacter pylori* CagA status and gastric mucosa-associated lymphoid tissue lymphoma: a systematic review and meta-analysis. *J Health Popul Nutr.* 2022;41:2. doi:10.1186/s41043-021-00280-9
 26. Gravina AG, Federico A, Ruocco E, Lo Schiavo A, Masarone M, Tuccillo C, et al. *Helicobacter pylori* infection but not small intestinal bacterial overgrowth may play a pathogenic role in rosacea. *United European Gastroenterol J.* 2015;3(1):17-24. doi:10.1177/2050640614559262

27. Frydrych ZL, Chwarścianek N, Błaszak K, Czajkowski R. The potential role of *Helicobacter pylori* and other gut dysbiosis factors in the development of rosacea. *Clin Dermatol Rev.* 2022;6(2):75-80. doi: 10.5603/fd.96224
28. Manfredini M, Barbieri M, Milandri M, Longo C. Probiotics and diet in rosacea: current evidence and future perspectives. *Biomolecules.* 2025;15(3):411. doi:10.3390/biom15030411
29. Chen P, Yang Z, Fan Z, Wang B, Tang Y, Xiao Y, et al. Associations of adherence to Mediterranean-like diet pattern with incident rosacea: a prospective cohort study of government employees in China. *Front Nutr.* 2023;10:1092781. doi:10.3389/fnut.2023.1092781
30. Parodi A, Paolino S, Greco A, Drago F, Mansi C, Rebora A, et al. Small intestinal bacterial overgrowth in rosacea: clinical effectiveness of its eradication. *Clin Gastroenterol Hepatol.* 2008;6(7):759-764. doi:10.1016/j.cgh.2008.02.054
31. Antybiotykoterapia praktyczna. Dzierżanowska D., wydanie VI, s. 210.