EUROPEAN OPEN SCIENCE SPACE

COLLECTION OF SCIENTIFIC PAPERS

9th International Scientific and Practical Conference

EVOLVING SCIENCE: THEORIES, DISCOVERIES AND PRACTICAL OUTCOMES

ISSUE №110

eoss-conf.com

October 19-21, 2026
Zurich, Switzerland

EUROPEAN OPEN SCIENCE SPACE

Proceedings of the 9th International Scientific and Practical Conference

"Evolving Science: Theories, Discoveries and Practical Outcomes"

October 19-21, 2026

Zurich, Switzerland

Collection of Scientific Papers

Switzerland, 2026

UDC 01.1

Collection of Scientific Papers with the Proceedings of the 9th International Scientific and Practical Conference «Evolving Science: Theories, Discoveries and Practical Outcomes» (October 19-21, 2026, Zurich, Switzerland). European Open Science Space. 2026.

ISBN 979-8-89704-958-5 (series)
DOI 10.70286/EOSS-19.10.2026

The conference is included in the Academic Research Index ReserchBib International catalog of scientific conferences.

The conference is registered in the database of scientific and technical events of UkrISTEI to be held on the territory of Ukraine (Certificate №564 dated 28.07.2026).

The materials of the conference are publicly available under the terms of the CC BY-NC 4.0 International license.

The materials of the collection are presented in the author's edition and printed in the original language. The authors of the published materials bear full responsibility for the authenticity of the given facts, proper names, geographical names, quotations, economic and statistical data, industry terminology, and other information.

ISBN 979-8-89704-958-5

EUROPEAN OPEN SCIENCE SPACE

© Participants of the conference, 2026
© Collection of scientific papers, 2026
© European Open Science Space, 2026

Section: Medicine

Уколова М.Є., Візір М.О.
АРТЕРІАЛЬНА ГІПЕРТЕНЗІЯ, АСОЦІЙОВАНА ІЗ ЗАСТОСУВАННЯМ КОМБІНОВАНИХ ОРАЛЬНИХ КОНТРАЦЕПТИВІВ: ФАКТОРИ РИЗИКУ ТА ПАТОГЕНЕТИЧНІ МЕХАНІЗМИ……... 192

Dunaieva I.P., Doroshenko O.M.
MRGPRX2 ANTAGONISTS IN ATOPIC DERMATITIS: PRECLINICAL FINDINGS AND LIMITATIONS OF CLINICAL EFFICACY…… 195

Фарзуллаєв Н.Н., Гончаров А.С., Герасименко Н.В., Криворучко І.А.
РАННЄ ЗАКРИТТЯ БОЙОВИХ РАН КІНЦІВОК: ВІД ХІРУРГІЧНОГО ДЕБРИДМЕНТУ ДО РЕКОНСТРУКЦІЇ……. 198

Kostyshyn O., Rudnikov Y., Nastenko I.A.
TRANSLATIONAL–ROTATIONAL BLOOD FLOW MODEL OF THE AORTA WITH AN ACTIVE WALL: THREE LEVELS OF DESCRIPTION AND THE NORMAL REFERENCE……. 202

Горбунова А.Ю., Візір М.О.
ВПЛИВ ХАРЧУВАННЯ НА РОЗВИТОК АТЕРОСКЛЕРОЗУ……... 208

Hryvkova L.V., Zotov O.S., Hryvkov S.S.
CHRONIC STRESS AS A MODULATOR OF THE TUMOR IMMUNE MICROENVIRONMENT……. 210

Андрєєв О. В., Тітаренко О. В., Тагунова І. К.
КЛЮЧОВІ ПРОБЛЕМИ КРІОХІРУРГІЇ В ОТОРИНОЛАРИНГОЛОГІЇ. ІНТЕЛЕКТУАЛЬНИЙ КОНТРОЛЬ ЗОНИ КРІОВПЛИВУ……... 218

Пилипенко О., Гончарова С.
ПОРІВНЯЛЬНА ХАРАКТЕРИСТИКА ВПЛИВУ ЦУКРУ ТА ЦУКРОЗАМІННИКІВ НА БІОХІМІЧНІ ВЛАСТИВОСТІ ЕМАЛІ…… 223

Chernikova H.
INTEGRATION OF DIGITAL HISTOLOGY WITH MULTIPLEX IMAGING AND SPATIAL TRANSCRIPTOMICS……. 225

5. Gunaratne MDSK, Thorsteinsdottir B, Garovic VD. Combined oral contraceptive pill-induced hypertension and hypertensive disorders of pregnancy: shared mechanisms and clinical similarities. Current Hypertension Reports. 2021;23(5):29. doi:10.1007/s11906-021-01147-4.
6. Державний експертний центр Міністерства охорони здоров'я України. Артеріальна гіпертензія: клінічна настанова, заснована на доказах. Київ; 2024. Реєстровий № КН 2024-1581.

MRGPRX2 ANTAGONISTS IN ATOPIC DERMATITIS: PRECLINICAL FINDINGS AND LIMITATIONS OF CLINICAL EFFICACY

Dunaieva I.P.
Doctor of Medical Sciences, Associate Professor
Doroshenko O.M.
Assistant
Department of pharmacology and pharmacotherapy
Kharkiv National Medical University
Kharkiv, Ukraine

Background. Atopic dermatitis (AD) is a chronic, relapsing inflammatory skin disease characterised by epidermal barrier dysfunction, immune dysregulation and neuroimmune mechanisms of pruritus. The Mas-related G protein-coupled receptor X2 (MRGPRX2) has attracted attention as a potential pharmacological target due to its involvement in IgE-independent mast cell activation. Neuropeptides such as substance P can activate this receptor, triggering mast cell degranulation and the subsequent release of pro-inflammatory mediators.

Experimental studies have shown that the MRGPRX2/MrgprB2–tryptase signalling pathway contributes to type 2 inflammatory responses in atopic dermatitis [1]. However, despite evidence supporting its role in disease pathogenesis, clinical results published in 2026 suggest that blocking MRGPRX2 alone may not confer sufficient therapeutic benefit.

Aim. To critically compare preclinical and clinical evidence concerning MRGPRX2 antagonists in AD, identify barriers to the clinical translation of this pharmacological approach and outline priorities for further research.

Materials and methods. A targeted narrative literature search and comparative analysis were conducted using publications indexed in PubMed and articles available through scientific publishers, with the search terms "MRGPRX2", "atopic dermatitis", "antagonist", "mast cell", "EP262" and "EVO756".

Results and discussion. In the study by Wong et al. (2024), the MRGPRX2 antagonist GE1111 reduced the expression of TSLP, IL-13, TNF-α and IL-1β in cell-

based experiments and attenuated erythema, skin thickening and histological signs of inflammation in a murine model of DNFB-induced dermatitis [2]. Nevertheless, chemically induced dermatitis in mice reproduces only selected aspects of human AD; therefore, these effects cannot be interpreted as direct evidence of clinical efficacy.

Further support for the pharmacological effects of MRGPRX2 blockade has come from studies of potent small-molecule antagonists. Wollam et al. (2024) found that these compounds inhibited MRGPRX2-mediated mast cell degranulation in cultured cells, in mice expressing the human receptor, and in human skin samples examined ex vivo [3]. In a subsequent study published in 2026, treatment with INCB000262 (EP262) attenuated house dust mite/staphylococcal enterotoxin B (HDM/SEB)-induced dermatitis in mice. The observed effects included reduced skin thickness, lower transepidermal water loss and improved composite clinical scores [4]. These findings indicate that pharmacological inhibition of MRGPRX2 can interfere with receptor-mediated signalling and alleviate certain inflammatory manifestations under experimental conditions.

By contrast, the randomised, double-masked, placebo-controlled phase IIa EASE trial (NCT06144424) yielded substantially different results. Thirty-two adults with AD were randomised to receive EP262 150 mg once daily (n = 21) or placebo (n = 11) for six weeks. The primary endpoint was safety and tolerability; secondary endpoints included changes in gene expression and skin histology, whereas clinical efficacy was assessed on an exploratory basis.

The mean percentage reduction in the Eczema Area and Severity Index (EASI) was 14.9% with EP262, compared with 40.7% with placebo. The proportions of patients achieving a validated Investigator Global Assessment for Atopic Dermatitis (vIGA-AD) score of 0 or 1, with an improvement of at least two points, were 21.1% and 27.3%, respectively. Reductions in pruritus intensity, assessed using the Peak Pruritus Numeric Rating Scale (PP-NRS), were comparable between groups (26.7% and 25.1%, respectively). No convincing between-group differences were identified in the assessed transcriptomic or histological parameters. Although no serious adverse events were observed, the limited number of participants and brief follow-up period make it difficult to assess the long-term safety of the treatment [5].

Further concerns were raised by an announcement from Evommune on 8 September 2026 concerning a phase IIb trial of the MRGPRX2 antagonist EVO756 involving 121 patients with moderate-to-severe AD. Over 12 weeks of treatment, neither the primary endpoint, percentage change in EASI, nor the secondary endpoints were met at any of the doses investigated. The developer subsequently announced that it would discontinue development of EVO756 for this indication [6]. As only summary results released by the company were available at the time of the analysis, they cannot be regarded as equivalent to the published EASI study in terms of detail or opportunities for independent appraisal.

Several hypotheses may account for the discrepancy between preclinical activity and clinical outcomes. First, experimental models predominantly assess preventive or early pharmacological effects on induced inflammation, whereas human AD is a chronic, heterogeneous disease involving multiple interconnected signalling pathways.

Second, systemic exposure to an antagonist does not establish adequate target engagement in lesional skin. In EASE, the predicted plasma concentrations of EP262 were achieved, but the extent of receptor blockade within the skin remained uncertain [5].

Third, the mean baseline EASI score in EASE was only 6.7, and treatment lasted six weeks, potentially limiting the study's sensitivity to detect a clinical effect. Nevertheless, the failure of two independent clinical programmes suggests that study design alone is unlikely to provide a sufficient explanation. A central unresolved question is whether MRGPRX2 represents a relevant therapeutic target across the broader AD population or predominantly in specific molecular endotypes.

Conclusions. MRGPRX2 antagonists have demonstrated reproducible pharmacodynamic activity in preclinical models; however, as of October 2026, convincing evidence of clinical efficacy in AD remains lacking. The EASE findings and preliminary phase IIb data for EVO756 do not support selective MRGPRX2 blockade as a stand-alone therapeutic strategy in an unselected population of patients with AD.

Further development will require validation of biomarkers of MRGPRX2-dependent inflammation, direct confirmation of target engagement in skin tissue, rational patient selection based on molecular endotype, and refinement of experimental models that better recapitulate the chronic course of AD. Negative clinical findings should be regarded as an important test of the underlying pharmacological hypothesis rather than as definitive evidence against the receptor's biological relevance.

References

1. Jia T., Che D., Zheng Y. et al. Mast Cells Initiate Type 2 Inflammation through Tryptase Released by MRGPRX2/MRGPRB2 Activation in Atopic Dermatitis. Journal of Investigative Dermatology. 2024;144(1):53–62.e2. DOI: 10.1016/j.jid.2023.06.201.
2. Wong T. K., Choi Y. G., Li P. H. et al. MRGPRX2 antagonist GE1111 attenuated DNFB-induced atopic dermatitis in mice by reducing inflammatory cytokines and restoring skin integrity. Frontiers in Immunology. 2024;15:1406438. DOI: 10.3389/fimmu.2024.1406438.
3. Wollam J., Solomon M., Villescaz C. et al. Inhibition of mast cell degranulation by novel small molecule MRGPRX2 antagonists. Journal of Allergy and Clinical Immunology. 2024;154(4):1033–1043. DOI: 10.1016/j.jaci.2024.07.002.
4. Wollam J., Solomon M., Villescaz C. et al. MRGPRX2 antagonist treatment prevents inflammation and disease in a mouse model of atopic dermatitis. Journal of Investigative Dermatology. 2026;146(10):2727–2735.e8. DOI: 10.1016/j.jid.2026.04.021.
5. Dehn S., Cooper D., Weyer C., et al. EP262, an MRGPRX2 Antagonist for the Treatment of Atopic Dermatitis: Results From the Phase 2 Randomised EASE Study. Dermatology and Therapy. 2026;16:5245–5255. DOI: 10.1007/s13555-026-01883-y.
6. Evommune, Inc. Evommune Announces Top-line Results from EVO756 Phase 2b Trial in Moderate-to-Severe Atopic Dermatitis. 8 September 2026. Available at: SEC filing (accessed 12 October 2026).

Proceedings of the 9th International Scientific
and Practical Conference
"Evolving Science: Theories, Discoveries and Practical Outcomes"
October 19-21, 2026
Zurich, Switzerland

Organizing committee may notagree with the authors'point of view.
Authors are responsible for the correctness of the papers' text.

Contact details of the organizing committee:
European Open Science Space
E-mail: info@eoss-conf.com
URL: https://www.eoss-conf.com/

EUROPEAN OPEN
SCIENCE SPACE