QUALITY IN SPORT
eISSN 2450-3118 · Open Access · Peer-reviewed
apcz.umk.pl/QS Nicolaus Copernicus University in Toruń

Cite as: ZIĘBA, Natalia, CZECH, Mateusz, TARCZYŃSKI, Dominik, MAZUR, Anna, CIESIELSKA, Dominika, CZMOK, Jakub, KOCIUBA, Katarzyna, CHOJĘTA, Michał, BRZUSEK, Mikołaj, and CISŁAK, Miłosz. The Role of Pesticide Exposure and Chronic Physical Exertion in Rheumatoid Arthritis Development – Mechanisms and Clinical Implications – A Narrative Review. Quality in Sport. 2026;62:73246. https://doi.org/10.12775/QS.2026.62.73246

ARTICLE TIMELINE
Received: 10.06.2026. Revised: 30.06.2026. Accepted: 04.07.2026. Published: 12.07.2026.

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 Role of Pesticide Exposure and Chronic Physical Exertion in Rheumatoid Arthritis Development – Mechanisms and Clinical Implications - A Narrative Review

Natalia Zięba

ORCID: https://orcid.org/0009-0005-4554-9536

natalia.zieba03@gmail.com

Medical University of Lublin

Aleje Racławickie 1; 20-059 Lublin; Poland

Mateusz Czech

ORCID: https://orcid.org/0009-0008-6980-6930

mateuszcz102@gmail.com

Medical University of Lublin

Aleje Racławickie 1; 20-059 Lublin; Poland

Dominik Tarczyński
ORCID: https://orcid.org/0009-0004-5307-0130
dominik.tarczynski02@gmail.com
Medical University of Lublin
Aleje Racławickie 1; 20-059 Lublin; Poland

Anna Mazur
ORCID: https://orcid.org/0009-0001-2568-0781
mazurania2801@gmail.com
Medical University of Lublin
Aleje Racławickie 1; 20-059 Lublin; Poland

Dominika Ciesielska
ORCID: https://orcid.org/0009-0007-8548-1112
dciesielska600@gmail.com
Medical University of Lublin
Aleje Racławickie 1; 20-059 Lublin; Poland

Jakub Czmok
ORCID: https://orcid.org/0009-0006-0869-9305
Kubel2003@gmail.com
Medical University of Lublin
Aleje Racławickie 1; 20-059 Lublin; Poland

Katarzyna Kociuba
ORCID: https://orcid.org/0009-0003-1265-2452
katarzyna.kociuba1115@gmail.com
Medical University of Lublin
Aleje Racławickie 1; 20-059 Lublin; Poland

Michał Chojęta
ORCID: https://orcid.org/0009-0007-0911-971X
michalchojeta9@gmail.com
Medical University of Lublin
Aleje Racławickie 1; 20-059 Lublin; Poland

Mikołaj Brzusek
ORCID: https://orcid.org/0009-0005-6266-6988
mrib3002@gmail.com
Medical University of Lublin
Aleje Racławickie 1; 20-059 Lublin; Poland

Miłosz Cisłak
ORCID: https://orcid.org/0009-0006-3999-6185
milosz.cislak@gmail.com
Medical University of Lublin
Aleje Racławickie 1; 20-059 Lublin; Poland

Corresponding Author: Natalia Zięba, natalia.zieba03@gmail.com

Abstract

Background: Rheumatoid arthritis (RA) is a chronic autoimmune disease influenced by both genetic and environmental factors. Increasing evidence suggests that exposure to pesticides and excessive physical activity resulting in physiological overload may influence the risk of developing RA.

Aim: To summarize current knowledge on the impact of pesticide exposure and physical activity on the risk of developing RA.

Material and methods: A narrative literature review was conducted using publications from PubMed and Google Scholar, supplemented with studies from major scientific publishers and medical databases. Epidemiological studies, systematic reviews, meta-analyses, and selected experimental studies on RA risk factors were analyzed.

Results: Exposure to pesticides, including insecticides, herbicides, and fungicides, as well as other occupational hazards such as organic solvents, silica, asbestos, animal dust, and engine exhaust emissions, was associated with an increased risk of RA. In contrast, studies on per- and polyfluoroalkyl substances (PFAS) suggested a possible protective effect in women. Regular moderate physical activity was linked to a reduced risk of RA development.
Conclusions: Environmental and occupational exposures may contribute to RA development, whereas moderate physical activity may have a protective effect. Further research is needed to clarify these relationships and support effective prevention strategies.
Keywords: Rheumatoid arthritis, Rheumatoid arthritis and risk factors, Rheumatoid arthritis and environmental factors, Physical activity and rheumatoid arthritis

1. Introduction

Rheumatoid arthritis (RA) is a chronic, systemic connective tissue disease with an autoimmune background [1]. Depending on the presence of characteristic antibodies, such as rheumatoid factor (RF) of the IgM class and anti-citrullinated peptide antibodies (ACPA), seropositive and seronegative forms of the disease are distinguished.

General symptoms of RA include fever, fatigue, loss of appetite, and weight loss. A characteristic feature of this disease is the involvement of joints, mainly synovial joints. Over time, the progressive inflammatory process leads to damage of the articular cartilage and the formation of bone erosions, which may result in permanent deformities and impaired joint function [2,3]. The lesions are usually symmetrical and may involve both small and large joints. The most typical manifestations include involvement of the metacarpophalangeal and proximal interphalangeal joints of the hands [4].

Early diagnosis and prompt initiation of appropriate treatment are crucial for preventing the progression of irreversible destructive changes within the joints. Extra-articular manifestations may also occur in the course of RA, involving multiple organs and body systems. The etiopathogenesis of the disease is complex and multifactorial, with genetic, immunological, cellular, and environmental factors playing significant roles [3,5].

The aim of this study is to summarize the current knowledge regarding the impact of chronic physical exertion and pesticide exposure as potential risk factors for the development of

rheumatoid arthritis. Particular attention was paid to possible pathophysiological mechanisms associated with chronic activation of inflammatory processes, oxidative stress, and disturbances in immune response, which may play an important role in the initiation and progression of the disease.

2. Materials and methods

2.1 Literature search and study selection

This paper is a narrative review aimed at summarizing current knowledge regarding risk factors for rheumatoid arthritis, particularly physical activity and environmental exposure, with special emphasis on pathomechanisms and prevention in this disease entity.

The literature review was conducted based on publications available in the PubMed and Google Scholar databases. Additional sources were obtained from reputable scientific publishers and medical databases, including Elsevier, Springer, Wiley, BMJ, BMC, Frontiers Media, Nature Publishing Group, and the NCBI platform, mainly covering the years 2011-2026. The search strategy was based on keywords such as: Rheumatoid arthritis, Rheumatoid arthritis and risk factors, Rheumatoid arthritis and environmental factors, Physical activity and rheumatoid arthritis.

The analysis included epidemiological studies, meta-analyses, systematic reviews, and selected experimental studies. The selection of publications was selective and focused on articles relevant to the subject of this paper, with particular emphasis on the most recent scientific reports. Due to the narrative and cross-sectional nature of the study, no formal quality assessment or statistical analysis was performed.

2.2 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.

3. Research results

3.1 Epidemiology of rheumatoid arthritis

Rheumatoid arthritis is a disease commonly occurring worldwide. According to the Global Burden of Disease Study, approximately 17.6 million people were living with RA in 2020. A further increase in the number of cases is projected, with estimates indicating that the number of individuals affected by RA will reach 31.7 million by 2050. An increasing trend in RA incidence was also observed in a study covering the years 1990-2019, which demonstrated a systematic rise in the number of individuals affected by this disease entity [6,7].

The average prevalence of RA is estimated at 208.8 cases per 100,000 inhabitants. Women develop RA approximately 2.5 times more frequently than men [6]. In a nationwide study conducted in 2021, women accounted for 72% of all patients diagnosed with RA. The highest incidence rates are observed between the sixth and seventh decades of life [8].

Mortality directly associated with RA remains relatively low, amounting to 0.47 deaths per 100,000 individuals [6]. No significant epidemiological differences have been identified between rural and urban areas [9]. However, lower incidence rates were observed in regions characterized by a greater proportion of green spaces [10]. At the same time, access to primary healthcare physicians and rheumatology specialists differs substantially between urban and rural areas, representing an important social and organizational challenge for the healthcare system [11].

These findings indicate that RA remains a significant public health issue with steadily increasing epidemiological importance. The growing number of cases and the aging population highlight the need for preventive measures, early diagnosis, and improved access to specialized rheumatological care.

3.2 Pathophysiology of rheumatoid arthritis

The pathophysiology of RA is complex and multifactorial. Both genetic predisposition and environmental factors play significant roles in the development of the disease. The most

important molecular processes involved in the pathomechanism of RA include activation of fibroblast-like synoviocytes, immune response dysregulation, abnormalities in cytokine signaling, and oxidative stress [12].

The chronic inflammatory state characteristic of this disease results from abnormal activation of numerous cells involved in the immune response. An important role is played by cells present in the synovial membrane, referred to as non-immune cells. These include fibroblast-like synoviocytes (FLS), which under pathological conditions undergo excessive proliferation, produce pro-inflammatory cytokines, and participate in cartilage and bone destruction. Macrophage-like synoviocytes (MLS) are also important, as they actively sustain the inflammatory process through the secretion of TNF-α and IL-1 [13]. Early activation of synovial fibroblasts is observed at the initial stages of the disease and may increase the risk of developing clinical symptoms [14].

Cells of the immune system are also involved in the inflammatory process, including B lymphocytes producing autoantibodies such as rheumatoid factor (RF) and anti-citrullinated protein antibodies (ACPA). T lymphocytes, which secrete pro-inflammatory cytokines, also play a crucial role. Furthermore, neutrophils, macrophages, and dendritic cells participate in the pathomechanism of RA. The combined activity of these cells leads to increased production of inflammatory mediators such as TNF-α, IL-1β, IL-6, IL-17, GM-CSF, and the chemokines CCL17, CCL22, CXCL8, and CXCL10 [13].

Oxidative stress is also an important element in the pathogenesis of RA. During this process, excessive amounts of reactive oxygen species (ROS) are generated, leading to cellular damage and intensification of the inflammatory response. Bioinformatic analyses have identified 89 genes associated with oxidative stress. The most important genes correlated with RA include STAT1, MMP9, MYC, CCL5, and JUN, which may potentially serve as diagnostic biomarkers of the disease [15].

An important role is also played by the p53 protein, which is responsible for regulating cellular responses to DNA damage and controlling inflammatory processes. Mutations in the p53 gene have been observed in patients with RA, and oxidative stress is considered one of the factors that may contribute to their development. Furthermore, p53 is involved in regulating reactive oxygen species levels through its influence on the activity of NADPH oxidase 4 (NOX4) [16].

Hormonal factors also play a significant role in the pathomechanism of RA. A study conducted in a large cohort from the UK Biobank demonstrated that menarche after the age of 14 years, menopause before the age of 45 years, and a shorter reproductive lifespan were associated with an increased risk of developing RA, suggesting an important role of estrogen exposure duration in the pathogenesis of the disease. Moreover, women who underwent hysterectomy or oophorectomy exhibited a higher risk of developing RA. An increased risk of the disease was also observed among women using hormone replacement therapy [17].

3.3 Environmental factors in the development of rheumatoid arthritis

A study conducted among licensed pesticide applicators demonstrated that, among insecticides, the greatest increase in the risk of rheumatoid arthritis (RA) was associated with the use of malathion. Malathion is a synthetic organophosphate insecticide, and its use was associated with a 77% increase in the risk of developing RA. An elevated risk was also observed for phorate, a highly toxic compound belonging to the same chemical group, which increased the risk by 40%. Among carbamates, carbaryl and carbofuran were identified as significant risk factors, increasing the risk of RA by 65% and 41%, respectively. Increased risk was also reported for exposure to herbicides such as metolachlor, alachlor, S-ethyl dipropylthiocarbamate, and metribuzin. Among fungicides, particular attention was drawn to benomyl, which was associated with a 56% increase in the risk of RA. Furthermore, a relationship between the duration and intensity of exposure and the risk of disease development was observed, indicating that prolonged and more intensive exposure to certain pesticides is associated with a greater risk of RA [18].

Epidemiological studies have also shown that RA is associated with the regular use of chemical fertilizers, exposure to organic solvents excluding gasoline, and engagement in painting-related activities [19]. Furthermore, an analysis based on data from the Women's Health Initiative revealed that exposure to insecticides increases the risk of developing autoimmune rheumatic diseases [20].

Additional evidence was provided by a meta-analysis published in 2026, which demonstrated that exposure to silica, asbestos, organic solvents, pesticides, fertilizers, animal dust, and engine exhaust emissions constitutes a significant risk factor for the development of RA [21].

In recent years, attention has also been directed toward perfluoroalkyl and polyfluoroalkyl substances (PFAS), which exhibit immunosuppressive properties. Some studies suggest that higher concentrations of selected PFAS compounds may be associated with a lower risk of RA among women, potentially due to their effects on immune system function. However, the mechanisms underlying this association have not yet been fully elucidated [22].

3.4 Physical activity and the risk of rheumatoid arthritis

A nationwide study involving 2.1 million individuals demonstrated that men performing physically demanding work were at a higher risk of developing RA. In contrast, no such association was observed among women [23]. These findings suggest that occupations involving substantial physical workload may increase the risk of RA. One potential mechanism underlying this relationship is chronic mechanical stress on the joints, which may contribute to the initiation and maintenance of inflammatory processes involved in disease development [24].

At the same time, moderate physical activity has been associated with a reduced risk of developing RA [25]. Furthermore, in patients with well-controlled disease, high-intensity exercise and resistance training should be considered, as they may improve physical function, muscle strength, and quality of life without increasing disease activity [26].

3.5 Prevention of rheumatoid arthritis

The prevention of rheumatoid arthritis (RA) is challenging because its exact etiology has not been fully elucidated. However, it is known that both genetic and environmental factors play a significant role in its development. Therefore, preventive strategies focus primarily on reducing modifiable risk factors such as smoking, diet, obesity, occupational exposure, periodontal disease, low educational level, and socioeconomic status [27, 28].

Diet plays an important role in the course of RA, although it is neither a direct cause nor a treatment method for the disease. Nevertheless, increasing evidence suggests that appropriate dietary habits may have a beneficial effect on disease progression. The Mediterranean diet, rich in vegetables, fruits, whole grains, nuts, and olive oil, is particularly well documented and may improve treatment outcomes in patients with RA. Beneficial effects have also been observed in relation to adherence to general healthy eating guidelines, including Irish dietary recommendations [29]. Higher consumption of fruits and vegetables is associated with

a reduced risk of disease development, whereas a diet high in red and processed meat may increase the risk of seropositive RA [30].

Obesity is another important modifiable risk factor. Excess adipose tissue does not serve solely as an energy storage site but also exhibits metabolic activity by secreting numerous pro-inflammatory mediators. These substances may exacerbate chronic inflammation and thus worsen the course of RA. Studies have shown that overweight increases the risk of RA by approximately 13%, while obesity increases it by about 25% [31].

Body mass index (BMI) is a widely used tool for assessing body weight. It has been shown that each 2.5 kg/m² increase in BMI is associated with a 9% increase in the risk of developing RA [32].

Another key modifiable risk factor is cigarette smoking. Tobacco smoke promotes chronic inflammation and may negatively affect immune mechanisms involved in disease development. Smokers have approximately twice the risk of developing RA compared to non-smokers [28].

Therefore, RA prevention should include maintaining a healthy body weight, following a diet rich in fruits and vegetables, engaging in regular moderate physical activity, and complete avoidance of cigarette smoking.

4. Summary and conclusions

The aim of this study was to present current knowledge regarding the impact of environmental factors and physical activity on the development of rheumatoid arthritis (RA), as well as to discuss the most important pathophysiological mechanisms involved in the development of this disease.

The conducted analysis demonstrated that exposure to insecticides, herbicides, fungicides, chemical fertilizers, organic solvents, silica, asbestos, animal dust, and engine exhaust emissions may increase the risk of developing RA. In contrast, studies investigating the relationship between per- and polyfluoroalkyl substances (PFAS) and RA suggest a potential protective effect among women. These findings indicate that reducing exposure to environmental factors with documented harmful effects may constitute an important element of disease prevention.

The analysis of available studies on physical activity suggests that moderate exercise may exert a protective effect against RA. In contrast, long-term heavy physical work leading to substantial joint loading may increase the risk of disease development. These findings highlight the importance of appropriately tailored physical activity both in the prevention of RA and in the management of individuals at increased risk of developing the disease.

Disclosure

The authors report no disclosures.

Author's contribution

Conceptualization: Natalia Zięba

Methodology: Anna Mazur, Dominik Tarczyński, Dominika Ciesielska

Software: Not applicable

Check: Jakub Czmok

Formal analysis: Katarzyna Kociuba, Mateusz Czech

Investigation: Michał Chojęta

Resources: Mikołaj Brzusek

Data curation: Miłosz Cisłak

Writing-rough preparation: Natalia Zięba, Mateusz Czech, Katarzyna Kociuba, Mikołaj Brzusek

Writing review and editing: Anna Mazur, Dominik Tarczyński, Miłosz Cisłak

Visualization: Jakub Czmok, Michał Chojęta

Supervision: Natalia Zięba

Project administration: Natalia Zięba, Dominika Ciesielska

Receiving funding: Not applicable.

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

Funding Statement:

This research received no external funding.

Institutional Review Board Statement:

Not applicable.

Informed Consent Statement:

Not applicable.

Data Availability Statement:

Not applicable.

Acknowledgments:

Not applicable.

Conflict of Interest Statement:

The authors declare no conflict of interest.

References

1. Di Matteo, A., & Emery, P. (2024). Rheumatoid arthritis: a review of the key clinical features and ongoing challenges of the disease. Panminerva Medica, 66(4), 427–442. https://doi.org/10.23736/S0031-0808.24.05272-8
2. Chauhan, K., Jandu, J. S., Brent, L. H., & Al-Dhahir, M. A. (2026). Rheumatoid Arthritis. StatPearls Publishing. http://www.ncbi.nlm.nih.gov/books/NBK441999/
3. Srivastava, S., & Rasool, M. (2024). Genetics, epigenetics and autoimmunity constitute a Bermuda triangle for the pathogenesis of rheumatoid arthritis. Life Sciences, 357, 123075. https://doi.org/10.1016/j.lfs.2024.123075
4. Jayatilleke, A. (2025). Arthritis: Rheumatoid Arthritis. FP Essentials, 548, 25-30. (Brak numeru DOI na zrzucie ekranu)

5. Han, P., Liu, X., He, J., Han, L., & Li, J. (2024). Overview of mechanisms and novel therapies on rheumatoid arthritis from a cellular perspective. Frontiers in Immunology, 15, 146175. https://doi.org/10.3389/fimmu.2024.146175
6. GBD 2021 Rheumatoid Arthritis Collaborators. (2023). Global, regional, and national burden of rheumatoid arthritis, 1990–2020, and projections to 2050: A systematic analysis of the Global Burden of Disease Study 2021. The Lancet Rheumatology, 5(10), e594–e610. https://doi.org/10.1016/S2665-9913(23)00021-4
7. Wang, Z., Gu, Y., Wang, R., He, Y., Ge, H., Yang, Z., Jin, Z., Jin, H., Lv, S., & Zhan, H. (2024). The global magnitude and temporal trend of rheumatoid arthritis burden attributable to smoking from 1990 to 2019. Rheumatology, 63(3), 689–697. https://doi.org/10.1093/rheumatology/kead269
8. Krajewska-Włodarczyk, M., Szeląg, M., Batko, B., Żuber, Z., Orleański, M., Podwójcic, K., Sowiński, J., Jopek, J., Świderek, M., Maluchnik, M., Brzosko, M., Śmiglewska, A., & Kwiatkowska, B. (2024). Rheumatoid arthritis epidemiology: A nationwide study in Poland. Rheumatology International, 44(6), 1155–1163. https://doi.org/10.1007/s00296-024-05591-8
9. Pianarosa, E., Chomistek, K., Hsiao, R., Anwar, S., Umaefulam, Valerie., Hazlewood, G., & Barnabe, C. (2022). Global rural and remote patients with rheumatoid arthritis: A systematic review. Arthritis Care & Research, 74(4), 598–606. https://doi.org/10.1002/acr.24513
10. Hu, J., Shen, P., Mao, Y., Qiu, J., Xu, L., Wu, Y., Wang, Y., Ding, Y., Lin, H., Shui, L., Feng, T., Wang, J., & Chen, K. (2024). Association of fine particulate matter and residential green space with rheumatoid arthritis. Environmental Research, 263(Pt 2), Artykuł 120151. https://doi.org/10.1016/j.envres.2024.120151
11. Liu, X., Patel, A. B., Seidel, J. E., Mosher, D. P., Hagens, J., & Marshall, D. A. (2025). Informing equitable access to care: A cross-sectional study of travel burden to primary and rheumatology care for people with rheumatoid arthritis. International Journal for Equity in Health, 24(1), Artykuł 84. https://doi.org/10.1186/s12939-025-02439-w
12. Kumar, A., Singh, S., Goel, F., Pandey, R. K., Singh, L., Kumar, A., & Dobhal, V. (2026). Molecular mechanisms and risk factors in rheumatoid arthritis: a comprehensive review. Inflammopharmacology, 34(1), 125–144. https://doi.org/10.1007/s10787-025-02073-6

13. Ganeshalingam, S., Wilson, N. J., Ciciriello, S., Antonipillai, J., & Achuthan, A. A. (2025). Cellular interactions in maintaining an inflammatory microenvironment in rheumatoid arthritis. Molecular Immunology, 184, 112–122. https://doi.org/10.1016/j.molimm.2025.06.008
14. Atzinger, A., Tascilar, K., Kleyer, A., Hueber, A. J., Schmidkonz, C., Kuwert, T., Beck, M., Fagni, F., Corte, G., Ramming, A., Schett, G., & Bayat, S. (2025). Synovial fibroblast activation occurs before the onset of rheumatoid arthritis and influences the risk of developing disease. RMD Open, 11(4), Article e005775. https://doi.org/10.1136/rmdopen-2025-005775
15. Gao, Z., Wu, A., Hu, Z., & Sun, P. (2025). Bioinformatics analysis of oxidative stress and immune infiltration in rheumatoid arthritis [Bioinformatics analysis of oxidative stress and immune infiltration in rheumatoid arthritis]. Journal of Southern Medical University, 45(4), 862–870. https://doi.org/10.12122/j.issn.1673-4254.2025.04.22
16. Robat-Jazi, B., Farhadi, E., Mahmoudi, M., Jamshidi, A., Akhlaghi, M., & Vaziri, A. S. (2025). Function of p53 in angiogenesis and oxidative stress in rheumatoid arthritis. Clinical and Experimental Medicine, 25(1), Article 322. https://doi.org/10.1007/s10238-025-01867-w
17. Jiang, L.-Q., Zhang, R.-D., Musonye, H. A., Zhao, H.-Y., He, Y.-S., Zhao, C.-N., He, T., Tian, T., ... Pan, H.-F. (2024). Hormonal and reproductive factors in relation to the risk of rheumatoid arthritis in women: a prospective cohort study with 223 526 participants. RMD Open, 10(1), Article e003338. https://doi.org/10.1136/rmdopen-2023-003338
18. Parks, C. G., Leyzarovich, D., Hamra, G. B., Costenbader, K. H., Chen, D., Hofmann, J. N., Freeman, L. E. B., & Sandler, D. P. (2024). Associations between pesticide use and rheumatoid arthritis among older farmers in the Agricultural Health Study. Scientific Reports, 14(1), Artykuł 29978. https://doi.org/10.1038/s41598-024-76179-2
19. Parks, C. G., Meyer, A., Beane Freeman, L. E., Hofmann, J. N., & Sandler, D. P. (2019). Farming tasks and the development of rheumatoid arthritis in the agricultural health study. Occupational and Environmental Medicine, 76(4), 243–249. https://doi.org/10.1136/oemed-2018-105361
20. Parks, C. G., Walitt, B. T., Pettinger, M., Chen, J.-C., de Roos, A. J., Hunt, J., Sarto, G., & Howard, B. V. (2011). Insecticide use and risk of rheumatoid arthritis and systemic

lupus erythematosus in the Women's Health Initiative Observational Study. Arthritis Care & Research, 63(2), 184–194. https://doi.org/10.1002/acr.20335

21. Liu, Q., Song, X., Mauro, E., Jiang, Y., Shchetynsky, K., Klareskog, L., Alfredsson, L., & Jiang, X. (2026). Exposure to Occupational Inhalants and the Risk of Developing Rheumatoid Arthritis: A Systematic Review and Meta-Analysis. Arthritis & Rheumatology, 78(4), 830–847. https://doi.org/10.1002/art.43446
22. Qiao, J. C., Li, Z. H., Ma, Y. B., Ma, H. Y., Zhang, M. Y., Zhang, X. J., & Hu, C. Y. (2024). Associations of per- and polyfluoroalkyl substances (PFAS) and their mixture with risk of rheumatoid arthritis in the U.S. adult population. Environmental Health, 23, Artykuł 38. https://doi.org/10.1186/s12940-024-01073-3
23. Nielsen, H. B., Gupta, N., Andersen, A. J., Dreyer, L. W., Flachs, E. M., Madsen, I. E. H., Kolstad, H. A., Kromhout, H., Sejbaek, C. S., & Hougaard, K. S. (2026). Physical work demands and risk of rheumatoid arthritis, systemic sclerosis, and systemic lupus erythematosus. A Danish longitudinal cohort study. Journal of Autoimmunity, 158, Artykuł 103514. https://doi.org/10.1016/j.jaut.2025.103514
24. van Dijk, B. T., Boeren, A. M. P., Khidir, S. J. H., den Hollander, N. K., & van der Helm-van Mil, A. H. M. (2024). Work-related physical strain and development of joint inflammation in the trajectory of emerging inflammatory and rheumatoid arthritis: a prospective cohort study. RMD Open, 10(2), Artykuł e003895. https://doi.org/10.1136/rmdopen-2023-003895
25. Ni, J., Zhou, Q., Meng, S.-Y., Zhou, T.-D., Tian, T., & Pan, H.-F. (2024). Sleep patterns, physical activity, genetic susceptibility, and incident rheumatoid arthritis: a prospective cohort study. BMC Medicine, 22(1), Artykuł 390. https://doi.org/10.1186/s12916-024-03615-5
26. Bilberg, A., Mannerkorpi, K., Borjesson, M., Svedlund, S., Sivertsson, J., Klingberg, E., & Bjersing, J. (2024). High-intensity interval training improves cardiovascular and physical health in patients with rheumatoid arthritis: a multicentre randomised controlled trial. British Journal of Sports Medicine, 58(23), 1409–1418. https://doi.org/10.1136/bjsports-2024-108369
27. Schäfer, C., Keyßer, G., & Reuß-Borst, M. (2024). [Influence of smoking, nutrition and other modifiable environmental factors on rheumatoid arthritis]. Zeitschrift für Rheumatologie, 83(9), 706–720. https://doi.org/10.1007/s00393-024-0155-y

28. Maisha, J. A., El-Gabalawy, H. S., & O'Neil, L. J. (2023). Modifiable risk factors linked to the development of rheumatoid arthritis: evidence, immunological mechanisms and prevention. Frontiers in Immunology, 14, Article 1221125. https://doi.org/10.3389/fimmu.2023.1221125
29. Raad, T., George, E., Griffin, A., Larkin, L., Fraser, A., Kennedy, N., & Tierney, A. (2024). Effects of a telehealth-delivered Mediterranean diet intervention in adults with Rheumatoid Arthritis (MEDRA): a randomised controlled trial. BMC Musculoskeletal Disorders, 25(1), Article 631. https://doi.org/10.1186/s12891-024-07742-1
30. Bäcklund, R., Bergström, U., Compagno, M., Arvidsson, L., Rydell, E., Sonestedt, E., & Turesson, C. (2025). Quantified intakes of key diet components and the risk of developing rheumatoid arthritis: Results from a nested case–control study. Annals of the Rheumatic Diseases, 84(11), 1791–1800. https://doi.org/10.1016/j.ard.2025.06.212
31. Cao, F., Kang, X., Wang, W., & Huang, S. (2025). Association of body mass index with the risk of rheumatoid arthritis: a systematic review and meta-analysis. Frontiers in Medicine, 12, Article 1750640. https://doi.org/10.3389/fmed.2025.1750640
32. Dong, Y., Greenwood, D. C., Hardie, L. J., & Cade, J. E. (2025). Measures of Adiposity and Risk of Rheumatoid Arthritis in Middle-Aged UK Women: A Prospective Cohort Study. Nutrients, 17(9), Article 1557. https://doi.org/10.3390/nu17091557