TPM Vol. 32, No. S9, 2025
ISSN: 1972-6325
https://www.tpmap.org/

THE RELATIONSHIP BETWEEN ENVIRONMENTAL FACTORS AND THE DEVELOPMENT OF RHEUMATIC DISEASES: SYSTEMATIC REVIEW

MOAMEN ABDELFADIL ISMAIL[1], RAWABI TALAL ALJAYAANI[2], SHOWQ ABDULLAH ALSAEDI[3], SALMA AWADH ALHARTHI[4], LUJANE HAMDI SEIF ELDIN MOHAMED[5], RAGHAD SAAD HASAN ALSHAHRANI[6], SARAH SAAD SHARIDAH ALRUWAILI[7], BASHAYER IBRAHIM ALKHALIFAH[8], SIRIN OMAR AL-FAQIH[9], ABDULLAH ANWAR ALBADI[10], ABDULRAHMAN DHAIFALLAH ALTHOBAITI[11], FAISAL ABDULLAH ALRASHED[12], ATHEER ABDULLAH ALMARHABI[13]

[1.] INTERNAL MEDICINE CONSULTANT, KING ABDULAZIZ SPECIALIST HOSPITAL - SAKAKA – ALJOUF
[2.] INTERNAL MEDICINE, KING SAUD MEDICAL CITY, RIYADH
[3.] INTERNAL MEDICINE RESIDENT
[4.] INTERNAL MEDICINE, KING ABDULAZIZ SPECIALIST HOSPITAL-TAIF
[5.] GENERAL PRACTITIONER ORCID
[6.] INTERNAL MEDICINE, ARMED FORCES HOSPITAL SOUTHERN REGION
[7.] INTERNAL MEDICINE
[8.] INTERNAL MEDICINE, PSBJH
[9.] INTERNAL MEDICINE
[10.] INTERNAL MEDICINE
[11.] MEDICAL IINTERN, TAIF UNIVERSITY, EMAIL: abdalrhman-1422@hotmail.com
[12.] INTERNAL MEDICINE, ALRASHEDFAISAL52@GMAIL.COM
[13.] INTERN

Abstract

Background: Rheumatic diseases, such as rheumatoid arthritis (RA) and dermatomyositis, are chronic autoimmune conditions whose development cannot be fully explained by genetic predisposition alone. This systematic review investigates the role of environmental factors in the initiation and progression of these diseases.

Methods: Following PRISMA guidelines, a comprehensive search was conducted across multiple electronic databases (PubMed, Scopus, Web of Science, Google Scholar) for studies published up to December 2024. Eligible studies included original research examining environmental exposures (e.g., air pollution, occupational inhalants, smoking, diet, ultraviolet radiation) and their association with rheumatic disease outcomes. Data were extracted and synthesized narratively due to heterogeneity.

Results: From 1,246 identified records, 11 studies comprising over 500,000 participants were included. Air pollution ($PM_{2.5}$, SO_2, NO_2) was consistently associated with increased RA risk and autoantibody positivity (e.g., ACPA). Occupational exposures (e.g., silica, toluene) significantly elevated RA risk, particularly in men and when combined with smoking and genetic susceptibility (OR up to 18.22). Smoking showed mixed effects, while ultraviolet radiation increased dermatomyositis risk. A Mediterranean diet exhibited a protective effect, but only among smokers. Gene-environment interactions consistently amplified disease risk.

Conclusion: Environmental factors—including air pollution, occupational inhalants, smoking, UV exposure, and diet—play a significant and often synergistic role in the development of rheumatic diseases, especially in genetically susceptible individuals. These findings highlight the importance of environmental risk assessment and targeted public health interventions for disease prevention.

BACKGROUND

Rheumatic diseases, including rheumatoid arthritis (RA) and idiopathic inflammatory myopathies such as dermatomyositis, are chronic autoimmune conditions characterized by persistent inflammation, joint damage, and

systemic manifestations. While genetic predisposition is an established determinant of susceptibility, it cannot fully explain disease onset or variation across populations. This has led to growing recognition of **environmental factors** as crucial contributors to disease initiation and progression (Tang et al., 2023; Zhang et al., 2023).

Air pollution has emerged as one of the most consistent environmental risk factors for autoimmune rheumatic diseases. Fine particulate matter ($PM_{2.5}$) and gaseous pollutants such as SO_2 and NO_2 have been implicated in promoting systemic inflammation and immune dysregulation. Zhao et al. (2020) demonstrated that exposure to **$PM_{2.5}$ and SO_2 was significantly associated with anti-citrullinated protein antibody (ACPA) positivity**, a serological hallmark of RA. Similarly, Alex et al. (2020) showed that $PM_{2.5}$ exposure independently predicted **higher ACPA concentrations** in RA patients.

Evidence from large-scale cohorts further strengthens the role of pollution. In a prospective study of over 340,000 UK Biobank participants, Zhang et al. (2023) found that **combined exposure to multiple pollutants increased RA incidence**, with the highest quartile of exposure associated with a hazard ratio of 1.14. These findings suggest that chronic exposure to polluted environments may act as a trigger for autoimmunity, particularly in genetically susceptible individuals.

Occupational exposures represent another critical environmental determinant. Workers exposed to silica, solvents, and other inhalable agents have consistently shown higher risks of RA. Wrangel et al. (2021) demonstrated that **silica dust exposure increased the odds of both seropositive and seronegative RA in men** (OR 1.22–1.23). Likewise, Min et al. (2021) reported that Korean workers exposed to **toluene had a standardized admission ratio of 2.38 for seropositive RA**. These results reinforce the concept that occupational hazards significantly shape autoimmune disease risk.

Beyond industrial exposures, agricultural and military environments have also been implicated. Ebel et al. (2021) found that U.S. veterans exposed to **burn pits and military waste disposal** had markedly increased odds of testing positive for anti-CCP antibodies, particularly among carriers of the shared epitope allele (OR up to 5.69). These data suggest that toxic inhalants encountered in combat or occupational settings may play a role in disease pathogenesis.

The interaction between environmental triggers and genetic predisposition is a recurring theme in rheumatology research. Tang et al. (2023) showed that exposure to multiple inhalable agents increased the risk of **ACPA-positive RA**, but the effect was dramatically amplified when combined with **smoking and HLA-shared epitope alleles**, leading to an 18-fold increase in risk. Similarly, Zhang et al. (2023) observed that individuals with both high air pollution exposure and high genetic risk scores had nearly **double the incidence of RA** compared to low-risk groups.

Other lifestyle-related exposures further influence disease development. Smoking, a well-known modifiable risk factor, has been shown to interact with genetic susceptibility and occupational exposures to magnify RA risk (Tang et al., 2023). However, in smaller cohorts, its role in disease activity remains less clear. For instance, Gudelj Gračanin et al. (2020) reported no significant difference in RA activity scores between smokers and nonsmokers in a Croatian cohort of 89 patients. This highlights the complexity of disentangling smoking's independent effects from its interactions with other risk factors.

In contrast, protective factors such as diet may mitigate environmental risks. Nguyen et al. (2021) showed that adherence to the **Mediterranean diet** did not reduce RA risk overall but significantly lowered incidence among ever-smokers (HR 0.91 per unit increase in diet score). This suggests that anti-inflammatory dietary patterns may help counteract harmful exposures, particularly in high-risk groups.

Ultraviolet (UV) radiation exposure represents another environmental determinant, particularly in idiopathic inflammatory myopathies. Parks et al. (2020) reported that individuals with high sun exposure or frequent sunburns were significantly more likely to develop **dermatomyositis** compared to polymyositis or inclusion body myositis (OR 1.44–1.77). This finding supports the role of solar radiation as a potential environmental trigger for autoimmune diseases beyond RA.

Together, these studies highlight the multifaceted nature of environmental influences on rheumatic diseases. Air pollution, occupational exposures, smoking, UV radiation, and diet all play important roles in disease risk, often acting synergistically with genetic predisposition. While genetic factors provide the underlying susceptibility, environmental exposures appear to **determine whether and when disease manifests**. Understanding these interactions is therefore essential for developing preventive strategies, early identification, and tailored interventions for populations at risk.

METHODOLOGY

Study Design

This research was conducted as a **systematic review** of published literature examining the relationship between environmental factors and the development of rheumatic diseases. The study followed the **Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines** to ensure transparency and reproducibility.

Search Strategy

A comprehensive search was performed across electronic databases including **PubMed, Scopus, Web of Science, and Google Scholar** to identify eligible studies published up to December 2024. The following keywords and Boolean

operators were used: "environmental factors" OR "air pollution" OR "occupational exposure" OR "inhalants" OR "smoking" OR "diet" OR "ultraviolet radiation" AND "rheumatic diseases" OR "rheumatoid arthritis" OR "dermatomyositis" OR "autoantibodies." Reference lists of relevant studies were also screened to identify additional articles.

Eligibility Criteria

Studies were included if they met the following criteria:

1. Peer-reviewed original research (cohort, case-control, cross-sectional, or randomized controlled trial).
2. Investigated at least one environmental exposure (e.g., air pollution, occupational agents, smoking, diet, ultraviolet radiation).
3. Reported outcomes related to **rheumatic diseases** (e.g., RA, dermatomyositis, autoantibody positivity).
4. Provided sufficient quantitative data (odds ratios, hazard ratios, standardized ratios, or p-values).
5. Published in English.

Studies were excluded if they were case reports, reviews, animal studies, or lacked relevant exposure or outcome data.

Screening and Selection Process

All identified records were imported into reference management software, and **duplicate studies were removed**. Titles and abstracts were screened independently by two reviewers. Full texts of potentially eligible studies were retrieved and assessed against the inclusion criteria. Disagreements were resolved through consensus or by consulting a third reviewer.

Data Extraction

A standardized data extraction form was used. The following information was collected from each study:

- Author(s) and year of publication
- Country and study design
- Sample size and population characteristics
- Type of environmental exposure assessed
- Outcome measured (RA incidence, dermatomyositis, autoantibody status, treatment response)
- Effect estimates (odds ratios, hazard ratios, standardized ratios, with 95% confidence intervals)
- Key findings and conclusions

Quality Assessment

The quality of observational studies (cohort and case-control) was assessed using the **Newcastle–Ottawa Scale (NOS)**, while randomized controlled trials were assessed using the **Cochrane Risk of Bias Tool**. Each study was graded as low, moderate, or high risk of bias.

Data Synthesis

Due to heterogeneity in study designs, exposures, and outcomes, a **narrative synthesis** was conducted rather than a formal meta-analysis. Results were summarized in comparative tables, and findings were grouped by exposure category: **air pollution, occupational exposures, smoking, diet, ultraviolet radiation, and genetic-environment interactions.** Where possible, effect sizes were compared across studies to identify consistent associations.

Ethical Considerations

As this study synthesized data from published literature, **no ethical approval or informed consent was required**. However, all studies included were assumed to have been conducted in accordance with relevant ethical guidelines.

RESULTS

PRISMA Flow

A systematic search and screening process identified **11 eligible studies** investigating the relationship between environmental factors and rheumatic diseases.

- **Records identified through database searches**: 1,246
- **Records after duplicates removed**: 1,108
- **Records screened (titles & abstracts)**: 1,108
- **Full-text articles assessed for eligibility**: 42
- **Studies excluded (not relevant, wrong population, wrong outcome)**: 31
- **Studies included in qualitative synthesis**: **11**

Thus, 11 studies were included in the final analysis, encompassing data from **over 500,000 participants** across multiple countries.

Table 1. Characteristics of Included Studies

Author (Year)	Country	Sample Size	Exposure	Disease/Outcome	Key Findings

Wrangel et al. (2021)	Sweden	31,139 RA cases + controls	Silica dust	RA risk	↑ Risk in men (OR 1.22–1.23)
Min et al. (2021)	Korea	148,870 workers	Toluene	RA admissions	↑ SAR for seropositive RA (2.38)
Ebel et al. (2021)	USA	797 veterans	Military/agricultural inhalants	Autoantibodies in RA	Burn pits ↑ anti-CCP (OR 1.66–5.69 in SE+)
Parks et al. (2020)	USA	1,350 registry	UV radiation	Dermatomyositis	Sunburn ↑ risk (OR 1.44–1.77)
Nguyen et al. (2021)	France	62,629 women	Mediterranean diet	RA incidence	No overall effect; ↓ risk in smokers (HR 0.91)
Gudelj Gračanin et al. (2020)	Croatia	89 RA patients	Smoking	Disease activity	No significant effect (DAS28 similar)
Zhao et al. (2020)	Canada	7,600	SO_2, NO_2, $PM_{2.5}$	ACPA positivity	$PM_{2.5}$ ↑ ACPA (OR 1.19), combined pollutants ↑ ACPA (OR 1.36)
Tang et al. (2023)	Sweden	4,033 RA cases, 6,485 controls	Occupational inhalable agents	RA incidence	↑ ACPA+ RA (OR 1.25); ↑ risk with smoking + genetics (OR 18.22)
Alex et al. (2020)	USA	557 veterans	Air pollutants ($PM_{2.5}$, PM_{10}, NO_2, SO_2, O_3)	Autoantibody levels	$PM_{2.5}$ ↑ ACPA concentration (p=0.037)
Zhang et al. (2023)	UK	342,973	Combined air pollution	RA incidence	$PM_{2.5}$ ↑ risk (HR 1.07); high pollution + genetics ↑ RA risk (HR 1.73)
Lend et al. (2024)	Nordic countries	778 RA patients	Genetic (RF, ACPA, SE alleles) & treatments	Treatment response	Environmental-genetic not strongly predictive

Table 2. Environmental Factors and Associated Risks

Environmental Factor	Number of Studies	Consistent Findings	Effect on Rheumatic Diseases
Air pollution ($PM_{2.5}$, NO_2, SO_2)	4	Yes	↑ ACPA positivity, ↑ RA risk
Occupational exposures (silica, solvents, inhalants)	4	Yes	↑ RA risk, esp. ACPA+ and in smokers
Smoking	2	Mixed	↑ Risk in gene–environment interaction studies; no effect on disease activity in small cohort
Ultraviolet (UV) exposure	1	Yes	↑ Dermatomyositis risk with sunburn
Diet (Mediterranean diet)	1	Conditional	Protective effect in smokers only
Genetic interactions	3	Yes	Pollution + smoking + HLA alleles → synergistic risk

Narrative Interpretation

- **Air pollution ($PM_{2.5}$, SO_2, NO_2)** consistently showed positive associations with RA development and autoantibody positivity, with effect sizes ranging from **OR 1.19–1.36** and HRs around **1.07**.
- **Occupational exposures** (silica, toluene, inhalable agents) increased RA risk, especially in men and those with genetic susceptibility. The strongest synergy was observed in Tang et al. (2023), where **triple exposure (pollution + smoking + high GRS)** yielded an **18-fold higher risk**.
- **Smoking** alone was not associated with RA disease activity in one small cohort, but in larger studies it amplified risk when combined with occupational or genetic factors.
- **UV radiation** increased the risk of dermatomyositis, supporting sunlight as a trigger for autoimmune disease.
- **Diet** (Mediterranean pattern) had a protective effect only among smokers, suggesting diet may buffer but not eliminate environmental risk.
- **Genetic context** consistently modified environmental effects, highlighting the importance of gene–environment interactions.

DISCUSSION

This systematic review evaluated the role of environmental factors in the development of rheumatic diseases, synthesizing evidence from 11 studies that collectively included over half a million participants across multiple regions. The findings demonstrate that environmental exposures—including air pollution, occupational inhalants, smoking, ultraviolet (UV) radiation, and diet—contribute significantly to disease risk, progression, and immunological changes. Importantly, many of these effects are amplified in the presence of genetic susceptibility, underscoring the complexity of gene–environment interactions in autoimmune pathogenesis.

Air pollution emerged as a consistent risk factor for the development of rheumatoid arthritis (RA) and autoantibody positivity. Zhao et al. (2020) reported that long-term exposure to industrial **$PM_{2.5}$ and SO_2** was significantly associated with **anti-citrullinated protein antibody (ACPA) positivity** (OR 1.19 and OR 1.03, respectively). Similarly, Alex et al. (2020) found that fine particulate matter independently predicted **higher ACPA concentrations** in RA patients, strengthening the evidence that inhaled pollutants may trigger autoimmune responses.

Zhang et al. (2023) provided large-scale prospective evidence from the UK Biobank, demonstrating that **combined exposure to multiple air pollutants** significantly increased RA incidence, with a hazard ratio of **1.14 for the highest quartile of pollution exposure**. These results align with epidemiological findings by Zhao et al. (2020) and Alex et al. (2020), indicating that **fine particulate matter ($PM_{2.5}$)** is the most consistent pollutant associated with autoimmune activity.

Occupational exposures also emerged as strong predictors of RA development. Wrangel et al. (2021) identified that **silica dust exposure increased the odds of both seropositive and seronegative RA among men** (OR 1.22–1.23). Similarly, Min et al. (2021) found that workers exposed to **toluene** had a markedly elevated **standardized admission ratio for seropositive RA (SAR 2.38)**. These studies highlight that industrial and chemical exposures remain a substantial occupational health risk for rheumatic diseases.

More recent evidence has expanded the range of hazardous occupational exposures. Tang et al. (2023) reported that exposure to any **inhalable occupational agents** significantly increased the risk of **ACPA-positive RA (OR 1.25)**, with risks escalating as exposure duration or number of agents increased. Crucially, when combined with **smoking and high genetic risk**, the odds ratio rose dramatically to **18.22**, demonstrating a strong synergistic interaction.

The role of military exposures further supports this occupational link. Ebel et al. (2021) observed that U.S. veterans exposed to **burn pits and military waste disposal** were significantly more likely to test positive for anti-CCP antibodies, with odds ratios as high as **5.69 among HLA-shared epitope carriers**. These findings add weight to the growing concern over military-related inhalant exposures as autoimmune triggers.

Smoking remains a controversial environmental factor. Gudelj Gračanin et al. (2020) reported no significant association between smoking and RA disease activity, seropositivity, or demographic factors in a small Croatian cohort. However, larger gene–environment interaction studies provide a different perspective. Tang et al. (2023) demonstrated that smoking dramatically enhanced the effect of occupational exposures and genetic risk, indicating that while smoking alone may not always predict severity, it is a critical catalyst when combined with other risk factors.

Dietary patterns may offer protective effects. Nguyen et al. (2021) examined adherence to the **Mediterranean diet** in the E3N-EPIC cohort and found no overall effect on RA risk. However, among **ever-smokers**, higher adherence reduced RA risk (HR 0.91 per score unit). This suggests that diet may buffer the harmful effects of other exposures, particularly tobacco, and represents an important modifiable factor in disease prevention strategies.

Environmental UV exposure has been associated with dermatomyositis. Parks et al. (2020) reported that individuals with **repeated sunburns or high occupational/hobby-related sun exposure** had significantly increased odds of developing dermatomyositis compared to polymyositis or inclusion body myositis (OR 1.44–1.77). This indicates that not only inhalants and pollutants but also **solar radiation** can act as a disease trigger in susceptible populations.

The interplay between genetic predisposition and environmental exposures emerged as a unifying theme. Tang et al. (2023) and Ebel et al. (2021) both demonstrated that **HLA-DRB1 shared epitope alleles** amplify the effects of occupational and military exposures. Likewise, Zhang et al. (2023) observed that individuals with **high polygenic risk scores** and high pollution exposure had almost **double the incidence of RA** compared with those at low risk.

Interestingly, genetic biomarkers such as RF and ACPA were less predictive of treatment response to biologics, as shown by Lend et al. (2024). This suggests that environmental influences may be more critical in **disease onset and serological activity**, while genetic markers may have limited clinical relevance in predicting therapeutic outcomes.

The cumulative evidence suggests that **environmental exposures are not isolated risk factors**, but rather interact synergistically with lifestyle and genetic susceptibility. Air pollution and occupational exposures appear to act as **primary triggers**, while smoking and diet serve as **modifying factors** that can either exacerbate or mitigate disease risk.

Geographic and cultural factors also play a role in exposure patterns. For example, occupational inhalant risks were more evident in Swedish and Korean studies due to industrial exposures (Wrangel et al., 2021; Min et al., 2021), whereas UV-related dermatomyositis risk was highlighted in U.S. cohorts (Parks et al., 2020). These differences

suggest that **local environmental conditions influence disease patterns**, making regional public health strategies essential.

Collectively, the findings underscore the importance of integrating **environmental risk assessments into clinical practice**. Screening for occupational history, pollution exposure, and lifestyle factors such as diet and smoking may improve early identification of at-risk individuals. Moreover, genetic testing for high-risk alleles could help stratify patients for preventive interventions, especially in high-exposure environments.

This review highlights an urgent need for preventive policies targeting environmental risks. Reducing air pollution exposure, regulating workplace inhalants, addressing military toxic exposures, and promoting protective diets could significantly lower the burden of rheumatic diseases. Importantly, these strategies should be prioritized for individuals with known genetic predisposition, where risk amplification is greatest.

CONCLUSION

Environmental factors—including **air pollution, occupational inhalants, smoking, UV exposure, and diet—play a significant role in the development of rheumatic diseases**, particularly RA and dermatomyositis. The evidence consistently shows that these exposures increase autoantibody positivity and disease incidence, often synergizing with genetic predisposition. While not all exposures uniformly predict disease severity, their cumulative and interactive effects suggest that environmental modification remains a powerful tool in prevention.

Recommendations

1. **Public Health Policy**: Implement stricter regulations on air pollution and occupational inhalant exposures, with targeted protections for high-risk industries.
2. **Military and Occupational Health**: Expand monitoring and exposure reduction strategies for workers and veterans exposed to burn pits, silica, solvents, and industrial agents.
3. **Lifestyle Interventions**: Promote dietary patterns such as the Mediterranean diet, particularly among smokers and high-risk populations.
4. **Genetic–Environmental Risk Profiling**: Incorporate genetic testing into risk assessment to identify individuals most vulnerable to environmental triggers.
5. **Patient Education**: Increase awareness of UV protection, smoking cessation, and dietary modifications to mitigate disease risk.
6. **Future Research**: Conduct longitudinal studies integrating genetic, environmental, and lifestyle data to better predict and prevent autoimmune disease onset.

REFERENCES

1. Lend, K., Lampa, J., Padyukov, L., Hetland, M. L., Heiberg, M. S., Nordström, D. C., Nurmohamed, M. T., Rudin, A., Østergaard, M., Haavardsholm, E. A., Hørslev-Petersen, K., Uhlig, T., Sokka-Isler, T., Gudbjornsson, B., Grondal, G., Frazzei, G., Christiaans, J., Wolbink, G., Rispens, T., Twisk, J. W. R., … van Vollenhoven, R. F. (2024). Association of rheumatoid factor, anti-citrullinated protein antibodies and shared epitope with clinical response to initial treatment in patients with early rheumatoid arthritis: data from a randomised controlled trial. Annals of the rheumatic diseases, 83(12), 1657–1665. https://doi.org/10.1136/ard-2024-226024
2. Wrangel, O., Graff, P., Bryngelsson, I. L., Fornander, L., Wiebert, P., & Vihlborg, P. (2021). Silica Dust Exposure Increases Risk for Rheumatoid Arthritis: A Swedish National Registry Case-Control Study. Journal of occupational and environmental medicine, 63(11), 951–955. https://doi.org/10.1097/JOM.0000000000002281
3. Min, Y. S., Lee, S., Kim, M., & Ahn, Y. S. (2021). Rheumatoid arthritis in low-level toluene-exposed workers based on nationwide medical surveillance data in Korea. American journal of industrial medicine, 64(4), 245–250. https://doi.org/10.1002/ajim.23226
4. Ebel, A. V., Lutt, G., Poole, J. A., Thiele, G. M., Baker, J. F., Cannon, G. W., Gaffo, A., Kerr, G. S., Reimold, A., Schwab, P., Singh, N., Richards, J. S., Ascherman, D. P., Mikuls, T. R., & England, B. R. (2021). Association of Agricultural, Occupational, and Military Inhalants With Autoantibodies and Disease Features in US Veterans With Rheumatoid Arthritis. Arthritis & rheumatology (Hoboken, N.J.), 73(3), 392–400. https://doi.org/10.1002/art.41559
5. Parks, C. G., Wilkerson, J., Rose, K. M., Faiq, A., Noroozi Farhadi, P., Long, C. S., Bayat, N., Brunner, H. I., Goldberg, B., McGrath, J. A., Miller, F. W., & Rider, L. G. (2020). Association of Ultraviolet Radiation Exposure With Dermatomyositis in a National Myositis Patient Registry. Arthritis care & research, 72(11), 1636–1644. https://doi.org/10.1002/acr.24059
6. Nguyen, Y., Salliot, C., Gelot, A., Gambaretti, J., Mariette, X., Boutron-Ruault, M. C., & Seror, R. (2021). Mediterranean Diet and Risk of Rheumatoid Arthritis: Findings From the French E3N-EPIC Cohort Study. Arthritis & rheumatology (Hoboken, N.J.), 73(1), 69–77. https://doi.org/10.1002/art.41487

7. Gudelj Gračanin, A., Marković, I., Golob, M., Lucijanić, M., Valetić, A. M., & Morović-Vergles, J. (2020). The effect of smoking on disease activity in rheumatoid arthritis - our experience. Acta clinica Croatica, 59(2), 312–317. https://doi.org/10.20471/acc.2020.59.02.15
8. Zhao, N., Smargiassi, A., Hatzopoulou, M., Colmegna, I., Hudson, M., Fritzler, M. J., Awadalla, P., & Bernatsky, S. (2020). Long-term exposure to a mixture of industrial SO_2, NO_2, and $PM_{2.5}$ and anti-citrullinated protein antibody positivity. Environmental health : a global access science source, 19(1), 86. https://doi.org/10.1186/s12940-020-00637-3
9. Tang, B., Liu, Q., Ilar, A., Wiebert, P., Hägg, S., Padyukov, L., Klareskog, L., Alfredsson, L., & Jiang, X. (2023). Occupational inhalable agents constitute major risk factors for rheumatoid arthritis, particularly in the context of genetic predisposition and smoking. Annals of the rheumatic diseases, 82(3), 316–323. https://doi.org/10.1136/ard-2022-223134
10. Alex, A. M., Kunkel, G., Sayles, H., Flautero Arcos, J. D., Mikuls, T. R., & Kerr, G. S. (2020). Exposure to ambient air pollution and autoantibody status in rheumatoid arthritis. Clinical rheumatology, 39(3), 761–768. https://doi.org/10.1007/s10067-019-04813-w
11. Zhang, J., Fang, X. Y., Wu, J., Fan, Y. G., Leng, R. X., Liu, B., Lv, X. J., Yan, Y. L., Mao, C., & Ye, D. Q. (2023). Association of Combined Exposure to Ambient Air Pollutants, Genetic Risk, and Incident Rheumatoid Arthritis: A Prospective Cohort Study in the UK Biobank. Environmental health perspectives, 131(3), 37008. https://doi.org/10.1289/EHP10710