THE EUROPEAN SOCIETY OF MEDICINE
Medical Research Archives, Volume 13 Issue 11

RESEARCH ARTICLE

How Education Shapes Autoimmune Disease Risk: Evidence from Mendelian Randomization and Epidemiology

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OPEN ACCESS

PUBLISHED
30 November 2025

CITATION
Freire de Carvalho, J., 2025. How Education Shapes Autoimmune Disease Risk: Evidence from Mendelian Randomization and Epidemiology. Medical Research Archives, [online] 13(11). https://doi.org/10.18103/mra.v13i11.7077

DOI
https://doi.org/10.18103/mra.v13i11.7077

ISSN
2375-1924

ABSTRACT

Autoimmune diseases represent a major cause of chronic disability worldwide and arise from complex interactions between genetic, environmental, and social determinants. Among these factors, educational attainment has emerged as an important predictor of immune-mediated disease risk. This review synthesizes current evidence on the relationship between education and autoimmune diseases, with specific emphasis on Mendelian randomization studies, which use genetic variants as instruments to strengthen causal inference. A structured literature search identified genetic instrumental-variable analyses and observational studies evaluating education and autoimmune disease susceptibility. Consistent Mendelian randomization findings demonstrate that higher educational attainment reduces the risk of rheumatoid arthritis by approximately 50–60 percent, with nearly half of this protective effect mediated by modifiable factors such as smoking and body mass index. Emerging evidence for systemic lupus erythematosus, autoimmune thyroid disease, and psoriasis suggests similar protective trends, while associations with type 1 diabetes and latent autoimmune diabetes in adults appear heterogeneous. Educational attainment also influences autoimmune disease risk indirectly through occupational exposures, health behaviors, and systemic inflammation. Understanding these pathways highlights opportunities for prevention through behavioral, environmental, and policy-level interventions aimed at reducing disparities in autoimmune disease.

Keywords: rheumatoid arthritis, systemic lupus erythematosus, education, Mendelian randomization, social determinants of health, autoimmune disease.

Introduction

Autoimmune diseases (AIDs) are chronic inflammatory disorders caused by a breakdown in immunological self-tolerance, leading to tissue-specific or systemic damage. Rheumatoid arthritis (RA), systemic lupus erythematosus (SLE), systemic sclerosis, dermatomyositis, and polymyositis are among the most prevalent, imposing a substantial burden in terms of disability, healthcare utilization, and mortality.[1-3]

While genetic susceptibility is fundamental to AID pathogenesis, environmental and social determinants critically shape disease onset and outcomes. Educational attainment — a measurable indicator of socioeconomic status — strongly predicts health behaviors, occupational exposure, access to care, and lifestyle risk factors such as smoking and obesity.[4,5] In rheumatology, lower educational levels have been associated with greater disease activity, reduced adherence to treatment, and poorer long-term prognosis.[5]

Traditional observational studies consistently report an inverse relationship between education and autoimmune disease risk, but such studies are prone to residual confounding and reverse causation. To address these limitations, Mendelian randomization (MR) — a genetic epidemiological approach using germline variants as instrumental variables — provides a robust framework for causal inference.[6,7] Recent genome-wide association studies (GWAS), such as those from the Social Science Genetic Association Consortium (SSGAC) involving over 1.1 million participants, have generated validated genetic instruments for educational attainment.[8] These developments enable causal testing of the relationship between education and AID risk with unprecedented precision.

This review integrates MR evidence and supporting observational data to evaluate the causal role of education in autoimmune disease susceptibility, emphasizing mediating factors such as smoking, body mass index (BMI), and occupational exposures. By comparing findings across diseases, we aim to clarify the mechanistic pathways through which education shapes immune and inflammatory risk profiles.

Materials and Methods

SEARCH STRATEGY AND DATA SOURCES

A structured literature search was performed across PubMed, Scopus, and Web of Science databases up to September 2025, following recommendations for narrative reviews. The search terms included: *"educational attainment"*, *"schooling"*, *"socioeconomic status"*, *"autoimmune disease"*, *"rheumatoid arthritis"*, *"systemic lupus erythematosus"*, *"Mendelian randomization"*, *"GWAS"*, *"psoriasis"*, *"type 1 diabetes"*, *"latent autoimmune diabetes in adults"*, and *"silica exposure"*. No language restrictions were applied. Reference lists of included studies were screened to identify additional publications.

INCLUSION AND EXCLUSION CRITERIA

Eligible studies met the following criteria:

1. Peer-reviewed publications exploring the association between educational attainment and risk of autoimmune diseases.
2. Studies employing MR, cohort, case-control, or meta-analytic designs.
3. Reports providing quantitative risk estimates (e.g., odds ratios, β-coefficients) or mediation analyses involving education, smoking, BMI, or occupational factors.

Exclusion criteria: Non-original studies lacking analytical data, conference abstracts, or commentaries, and duplicates and reports without defined educational exposure.

DATA EXTRACTION AND SYNTHESIS

Data were extracted for disease type, study design, population ancestry, sample size, effect estimates, mediators, and methodological quality (instrument strength and pleiotropy assessment for MR studies). Quality assessment followed GRADE guidelines where applicable, and MR-specific rigor was evaluated via F-statistics (>10), MR-Egger regression for pleiotropy,

and MR-PRESSO outlier detection. A PRISMA-style flowchart (to be included separately) summarizes the selection process.

Results

A total of 10 studies met inclusion criteria: six MR analyses, two large prospective cohorts, one multi-cohort inflammatory biomarker study, and one meta-analysis on occupational exposure. Collectively, they covered more than 1.5 million individuals across multiple ancestries. See Table 1 for a summary of all included studies.

RHEUMATOID ARTHRITIS (RA)

The most consistent MR evidence supports a causal inverse association between education and RA risk.

- Huang et al.[9] analyzed 14,361 rheumatoid arthritis cases and 43,923 controls and demonstrated that each standard deviation (~4.2 years) increase in education reduced rheumatoid arthritis risk by 58% (OR 0.42, 95% CI 0.34–0.52; $p < 10^{-13}$).
- Zhao et al.[10] confirmed these findings, reporting that 24% of the protective effect of education on rheumatoid arthritis risk was mediated by smoking and 17% by body mass index. Together, these results indicate that nearly half of the educational benefit operates through modifiable behavioral pathways.

These studies indicate that nearly half of the educational benefit operates through modifiable behavioral pathways, providing direct translational relevance for prevention strategies.

SYSTEMIC LUPUS ERYTHEMATOSUS (SLE) AND CONNECTIVE TISSUE DISEASES

Zeng et al.[11] examined five connective tissue diseases and found a protective effect for both RA (OR 0.63) and SLE (OR 0.34), but not for systemic sclerosis, dermatomyositis, or polymyositis. This heterogeneity suggests disease-specific immune and environmental mechanisms modulating educational effects.

PSORIASIS AND AUTOIMMUNE THYROID DISEASE

Li et al.[12] identified inverse associations between education and both psoriasis (OR 0.61) and autoimmune thyroid disease (OR 0.80), with partial mediation by smoking, BMI, and income. These findings extend the educational gradient in immune disease beyond rheumatology to dermatologic and endocrine autoimmunity.

TYPE 1 DIABETES (T1D) AND LADA

Evidence for diabetes-related autoimmunity shows a more complex pattern.

White et al.[13] demonstrated that low maternal education increases the risk of childhood-onset type 1 diabetes, mediated by childhood body mass index and exposure to serious life events.

In contrast, Olsson et Al.[14] reported that higher educational attainment was associated with increased risk of latent autoimmune diabetes in adults. This paradox may reflect differences in health-seeking behavior, diagnostic pathways, and demographic patterns across age groups rather than a biological effect.

BIOLOGICAL AND OCCUPATIONAL MEDIATORS

Lower educational attainment has been consistently linked to increased levels of inflammatory biomarkers such as C-reactive protein and fibrinogen, independent of smoking or body mass index. Maurel et al.[15] demonstrated that systemic low-grade inflammation may represent biological embodiment of social inequality.

Occupational exposures also play an important mediating role. Workers with lower education are more frequently exposed to crystalline silica, organic solvents, and other industrial agents. Silica exposure markedly increases the risk of rheumatoid arthritis and systemic sclerosis, with an even higher risk when combined with smoking, as shown by Morotti et al.[16] and Boudigaard et al.[17]

Table 1. Summary of the main studies evaluating the association between educational attainment and autoimmune diseases.

Autoimmune Disease	Study / Year	Design	Population / Ancestry	Genetic Instrument / F-statistic	Sample Size	Main Findings (Effect of Higher Education)	Mediators / Pathways	Key Observations	Major Limitations
Rheumatoid Arthritis (RA)	Huang et al., 2021 [10]	Mendelian Randomization (Two-sample)	European (UK Biobank; FinnGen)	1271 SNPs from SSGAC; F > 40	14,361 RA cases / 43,923 controls	OR 0.42 (95% CI 0.34–0.52) per +4.2 years schooling (~58% risk reduction)	Not assessed	Strong causal effect of education on RA risk reduction	European ancestry only; pleiotropy not fully excluded
Rheumatoid Arthritis (RA)	Zhao et al., 2022 [11]	Multivariable MR with mediation	European (UK Biobank)	1260 SNPs; F = 45	13,459 RA cases / 42,750 controls	OR 0.37 (95% CI 0.31–0.44)	Smoking (24%), BMI (17%) → total 47% mediation	Almost half of protective effect mediated by modifiable factors	Limited to behavioral mediators; no data on occupational factors
Systemic Lupus Erythematosus (SLE)	Zeng et al., 2024 [12]	MR (Five connective tissue diseases)	East Asian (China Kadoorie Biobank, FinnGen)	1,000+ SNPs; F > 30	SLE: 3,152 cases / 15,870 controls	OR 0.34 (95% CI 0.12–0.94)	Not assessed	Evidence of protective effect; less robust than for RA	Limited power; lack of replication in non-Asian populations
Systemic Sclerosis, Dermatomyositis, Polymyositis	Zeng et al., 2024 [12]	MR (same study)	East Asian	1,000+ SNPs; F > 30	4,320 combined cases	No significant association	—	No causal relationship detected	Small sample sizes; low statistical power
Psoriasis	Li et al., 2023 [13]	MR (Two-sample)	European (UK Biobank)	1218 SNPs; F > 35	10,231 cases / 25,486 controls	OR 0.61 (95% CI 0.52–0.72)	Smoking, BMI, Income (partial mediation)	Behavioral and socioeconomic mediation plausible	Education proxies not harmonized across datasets

Autoimmune Disease	Study / Year	Design	Population / Ancestry	Genetic Instrument / F-statistic	Sample Size	Main Findings (Effect of Higher Education)	Mediators / Pathways	Key Observations	Major Limitations
Autoimmune Thyroid Disease	Li et al., 2023 [13]	MR (Two-sample)	European (UK Biobank)	1218 SNPs; F > 35	9,504 cases / 28,900 controls	OR 0.80 (95% CI 0.72–0.88)	Smoking, BMI, Income (partial mediation)	Higher education associated with lower risk	Limited ancestry diversity
Type 1 Diabetes (Childhood)	White et al., 2023 [14]	Prospective Birth Cohort	Swedish national registries	Parental education reported; no MR	>1 million births; 3,721 T1D cases	Lower maternal education → higher T1D risk	Childhood BMI, stressful life events	Intergenerational transmission of social disadvantage	Observational; residual confounding possible
Latent Autoimmune Diabetes in Adults (LADA)	Olsson et al., 2011 [16]	Population-based Cohort	Swedish ESTRID study	No MR; educational level self-reported	377 LADA cases / 718 controls	Higher education associated with higher LADA risk	Not assessed	Possible diagnostic and healthcare access bias	Lack of genetic causal inference
Inflammatory Biomarkers	Maurel et al., 2020 [17]	Multi-cohort cross-sectional	European (France, UK, Denmark)	Not applicable	>20,000 participants	Lower education → higher CRP and fibrinogen	Independent of smoking and BMI	Indicates systemic low-grade inflammation as pathway	Observational; cannot infer causality
Occupational Exposure (Silica)	Morotti et al., 2022; Boudigaard et al., 2021 [18,19]	Meta-analysis and cohort studies	Multinational (Europe, USA)	Not applicable	Combined >30,000 exposed workers	Lower education → higher silica exposure and RA risk	Occupational exposure; synergy with smoking	Environmental mediator of education–RA link	Heterogeneity of exposure definitions

Discussion

The combined evidence from observational studies and Mendelian randomization (MR) analyses strongly supports a causal protective effect of higher educational attainment on the risk of autoimmune diseases, particularly rheumatoid arthritis (RA). The consistency of findings across multiple independent cohorts strengthens the biological and social plausibility of education as an upstream determinant of immune-mediated disease. MR studies reduce residual confounding and reverse causation by using genetic variants as instrumental variables, thus enhancing causal inference compared with conventional epidemiologic designs. In the largest MR analyses to date, Huang et al.[9] and Zhao et al.[10] demonstrated a 50–60% reduction in RA risk for each standard deviation increase in years of education. Zhao et al.[10] further showed that 24% of this protective effect was mediated by smoking and 17% by body mass index (BMI), indicating that nearly half of the educational benefit operates through modifiable behavioral mechanisms.

This association is biologically plausible. The impact of smoking and obesity on immune activation and autoantibody production is well established. Cigarette smoking is a recognized environmental trigger of RA, particularly anti–citrullinated peptide antibody–positive disease, because pulmonary citrullination generates neoantigens that drive loss of immune tolerance. Stolt et al.[18] provided compelling mechanistic evidence supporting this pathway. Obesity promotes chronic low-grade inflammation through adipokine release, macrophage infiltration into adipose tissue, and activation of the NLRP3 inflammasome, all influencing autoreactive T- and B-cell responses.[10] These pathways offer a biologically coherent link between education, metabolic health, and immune homeostasis.

Beyond behavioral factors, education appears to exert broader biological effects rooted in psychosocial and neuroendocrine mechanisms. Individuals with lower educational attainment are more likely to experience chronic psychosocial stress, financial strain, and reduced social support—conditions known to enhance activation of the hypothalamic–pituitary–adrenal (HPA) axis and the sympathetic nervous system. These neuroendocrine alterations contribute to glucocorticoid resistance and elevations in pro-inflammatory cytokines such as IL-6 and TNF-α. Maurel et al.[15] demonstrated that lower education is associated with higher levels of C-reactive protein and fibrinogen even after adjustment for smoking and BMI. Additional national cohort evidence from the MIDUS study similarly demonstrated that lower education correlated with markedly higher inflammatory biomarker levels, suggesting that systemic inflammation may represent a biological embedding of social disadvantage.[20] Likewise, analyses from the Panel Study of Income Dynamics confirmed that socioeconomic position, as measured by parental education and income, influenced immune biomarkers across the life course.[21] Together, these findings highlight psychosocial stress and neuroimmune dysregulation as additional mediators linking education to autoimmune disease risk.

Occupational exposures constitute another important mediating pathway. Individuals with lower educational attainment are more likely to work in manual labor or industrial settings, increasing exposure to crystalline silica, metal particulates, and organic solvents. Silica exposure markedly elevates risk for RA and systemic sclerosis and exerts a synergistic effect when combined with smoking. Morotti et al.[16] and Boudigaard et al.[17] demonstrated significant elevations in autoimmune disease risk among individuals exposed to respirable crystalline silica. A recent population-based study further showed that socioeconomic inequalities—including educational level—strongly predicted both the prevalence and severity of RA, reinforcing the influence of structural determinants in disease expression.[22]

Disease-specific patterns reveal further complexity. Zeng et al.[11] identified inverse associations between education and both RA and systemic lupus erythematosus but not systemic sclerosis, dermatomyositis, or polymyositis. Li et al.[12] extended

the educational gradient to psoriasis and autoimmune thyroid disease, with partial mediation through smoking, BMI, and income. Conversely, evidence related to diabetes-associated autoimmunity is heterogeneous. White et al.[13] found that low maternal education increased the risk of childhood type 1 diabetes through childhood BMI and exposure to serious life events, while Olsson et al.[14] reported higher education among adults with latent autoimmune diabetes in adults, potentially reflecting diagnostic or health-seeking patterns rather than a true biological effect. Recent genome-wide association studies also suggest that genetic liability to higher education reduces risk for several autoimmune diseases, mediated largely through behavioral and socioeconomic pathways.[22] These findings underscore that the impact of education may vary according to disease pathophysiology, life course timing, and health system dynamics.

Global epidemiological data further support the connection between educational inequality and autoimmune disease burden. A recent Global Burden of Disease analysis reported marked disparities in autoimmune diseases among children and adolescents in regions with lower educational and economic indices, suggesting that improving educational access may produce downstream benefits in reducing autoimmune disease incidence at the population level.[23] This aligns with broader ecological trends showing that autoimmune disease prevalence often mirrors regional differences in education, income, and occupational structures.

From a methodological standpoint, MR provides a powerful approach for assessing causal relationships in social epidemiology, but limitations remain. Although genetic instruments for education are strong and derived from large GWAS including over one million individuals, the exclusion-restriction assumption may be challenged by horizontal pleiotropy. Sensitivity analyses such as MR-Egger and MR-PRESSO reduce—but cannot fully eliminate—this risk. Furthermore, years of schooling used in GWAS reflect quantity rather than quality or context of education, which may vary substantially across societies. Another limitation is that most MR studies investigating education and autoimmune diseases are based on European-ancestry populations, raising concerns about generalizability. Extending MR approaches to multiethnic populations with diverse socioeconomic structures will be essential to determine whether these associations are consistent across populations.

Overall, the available evidence supports a multilevel, causal role of education in shaping autoimmune disease risk. Protective associations observed for RA, systemic lupus erythematosus, psoriasis, and autoimmune thyroid disease, combined with biological plausibility across behavioral, metabolic, psychosocial, and environmental pathways, provide a coherent mechanistic framework linking education to immune regulation. Although effect sizes and directionality vary across diseases, improving educational attainment and reducing educational disparities may represent long-term public-health strategies to reduce the burden of autoimmune diseases. Future research integrating MR, longitudinal epidemiology, and mechanistic immunology will be essential to clarify how social inequality becomes biologically embedded and to identify actionable targets for prevention across the life course.

Conclusion

In conclusion, based on the evidence presented here we believe that there is strong support for a multilevel and causal effect of education on autoimmune disease risk. The protective effects with RA, SLE, psoriasis and autoimmune thyroid disease seen along with biological plausibility in terms of behavioral, metabolic, psychosocial and environmental routes, offer a coherent biologic model for an education-immune regulatory relationship. Education seems to be a distal yet potent determinant that influences social, behavioral, and occupational contexts in which autoimmunity takes place. Although this effect size and the directionality may differ by disease, the steady increase of incidence rates with population education levels implies two

things: that improved educational attainment and decreased educational inequality can be a successful long-term public health strategy for lessening the occurrence as well as burden of diseases. Integrative MR-longitudinal epidemiology-mechanistic immunology designs in future research will help illuminate how social inequality becomes biologically embodied and, importantly, enable concrete targets of intervention at both an individual and population level to be identified.

Funding:

None

Conflict of interest:

None

Artificial intelligence (AI) use:

AI tools were utilized exclusively to assist in language editing and improving the clarity of sentences in the manuscript. All ideas, data synthesis, and conclusions presented in this study are entirely the responsibility of the authors.

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