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Research Paper

# Rheumatoid Factor in Long-term Sulfur Mustard Survivors: Associations With Sex Differences and Clinical Complications

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## ABSTRACT

**Background:** Rheumatoid factor (RF) is a classical autoantibody reflecting B-cell dysregulation and autoimmunity. Sulfur mustard (SM) is a potent chemical warfare agent that induces chronic pulmonary, dermatological, and ocular injuries, often accompanied by persistent inflammation, oxidative stress, and immune perturbation. This study aimed to investigate RF levels in SM-exposed individuals compared to unexposed controls, and to explore differences based on sex and clinical manifestations.

**Materials and Methods:** A total of 139 SM-exposed veterans and 109 unexposed controls were enrolled. Serum RF concentration was measured using ELISA. Participants were stratified by sex and by severity of lung, skin, and eye involvement.

**Results:** RF levels were significantly lower in the exposed group compared to controls ($P<0.01$). Sex-stratified analysis revealed a pronounced reduction in RF levels in exposed females compared to control females ($P<0.01$), whereas RF levels in males did not differ significantly. Analysis of clinical manifestations demonstrated consistently lower RF levels in exposed individuals across lung, skin, and eye involvement, with significant differences in most comparisons versus controls. Notably, RF reduction did not correlate with disease severity, suggesting that exposure, rather than disease severity, drives this effect.

**Conclusion:** Long-term SM exposure is associated with reduced RF levels, particularly in women, and with differences in clinical manifestations. These findings suggest chronic immune remodeling or B-cell dysfunction rather than classical autoantibody-driven autoimmunity. The observed heterogeneity compared with previous studies underscores the need for longitudinal and mechanistic investigations, including B-cell phenotyping, cytokine and oxidative stress profiling, and sex-specific analyses, better to understand the long-term immunologic consequences of SM exposure.

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## Introduction

Rheumatoid factor (RF) is a classical autoantibody directed against the Fc portion of immunoglobulin G (IgG) and represents a key marker of B-cell dysregulation and humoral autoimmunity. Although RF is most widely recognized in the diagnosis of rheumatoid arthritis, elevated levels may also occur in chronic inflammatory lung diseases, persistent infections, autoimmune disorders, and conditions characterized by sustained immune activation. RF production is strongly influenced by prolonged antigenic stimulation, cytokine imbalance, and aberrant activation of polyclonal B-cell responses, making it a sensitive indicator of chronic or systemic immune perturbation [1-3].

Sulfur mustard (SM), a potent alkylating chemical warfare agent, causes extensive acute tissue injury and long-term complications involving the skin, eyes, and respiratory system. Decades after exposure, many survivors develop chronic bronchial injury, bronchiolitis obliterans, airway remodeling, and a clinical condition often termed "mustard lung." These chronic pulmonary and epithelial abnormalities are accompanied by persistent inflammation, oxidative stress, and airway structural damage. Increasing evidence indicates that SM exposure also induces long-lasting alterations in the immune system, including disturbed cytokine networks, impaired regulatory mechanisms, and features of premature immunosenescence [4, 5]. Such chronic immune dysregulation persists for decades and may contribute to the emergence of autoimmune phenomena in exposed patients.

Autoantibody production has been reported in various chemical and environmental exposures, supporting the concept that ongoing tissue injury and unresolved inflammation can promote loss of immunological tolerance [6]. Given that the respiratory system is one of the primary targets of SM toxicity, the continuous presence of damaged epithelial cells, inflammatory mediators, and persistent antigenic stimulation in mustard lung may influence RF induction. However, despite extensive research on the respiratory and systemic consequences of SM exposure, little is known about RF alterations in long-term survivors.

Understanding whether RF levels differ between SM-exposed individuals and unexposed controls can provide insight into chronic immune activation and may help identify biomarkers that reflect long-term immunotoxicity. Moreover, sex-specific differences in autoimmune responses—including RF production—have been documented in numerous chronic diseases, suggesting the potential relevance of gender-stratified analysis in this population.

This study aimed to investigate whether long-term survivors of SM exposure exhibit altered RF profiles compared to unexposed individuals. By evaluating RF levels across the total and subgroups, this study provides new information on the immunological consequences of SM exposure decades after the initial insult. These findings contribute to a better understanding of chronic immune perturbation in chemical-exposed populations and help guide future research on late-onset autoimmunity associated with mustard lung.

## Materials and Methods

### Study participants, ethical approval, and clinical assessment and categorization

This study included two groups: 1) 139 individuals with documented exposure to SM during the Iran–Iraq war (33 years ago), and 2) an unexposed, healthy control group consisting of 109 individuals. The Medical Committee of the Foundation of Martyrs and Veterans Affairs confirmed the veterans' SM exposure. All procedures were conducted in accordance with institutional ethical guidelines, and written informed consent was obtained from all participants before enrollment. A comprehensive clinical assessment was conducted, and participants were categorized by sex (male or female). In total, patients were also enrolled in three study groups: chemical veterans with pulmonary, skin, and eye complications according to their medical records. These participants were further stratified into "normal–mild" and "moderate–severe" categories based on standardized clinical and paraclinical assessments. Pulmonary severity, assessed by a pulmonologist, was based on symptoms and pulmonary function tests, with normal–mild, including normal or mildly impaired function, and moderate–severe, including clinically significant disease (e.g. forced expiratory volume $(FEV)_1$<80% predicted). Dermatological severity, assessed by a dermatologist, was determined by the extent and persistence of lesions (e.g. pruritus, dryness, pigmentation changes, alopecia, lichenification, excoriation), with moderate–severe for moderate to extensive or persistent involvement and normal–mild for absent or mild, localized findings. Ocular severity, assessed by an ophthalmologist using slit-lamp and ophthalmoscopy, was classified as moderate–severe for significant corneal pathology or visual impairment, and normal–mild for absent or mild symptoms.

Common pulmonary symptoms associated with SM exposure included dyspnea, sputum, chronic cough, and hemoptysis. Notably, none of the patients had workplace-related pollution exposure.

Peripheral blood samples were collected under standardized conditions, and serum was separated, aliquoted, and stored until analysis.

### Sample collection

Blood samples were collected in Vacutainer tubes (BD Biosciences). Serum was obtained by centrifugation at 2000×g for 20 minutes at 4 ˚C, aliquoted under sterile conditions, and stored at −80 ˚C until analysis.

### RF serum levels measurement

Serum RF concentrations (U/) were measured using a RF Screen ELISA kit (DRG Instruments GmbH, Germany), following the manufacturer's instructions. This assay specifically quantifies IgM isotype RF (RF-IgM).

### Statistical analysis

Laboratory data were summarized using median, Mean±SD. Differences between study groups were evaluated using the Mann–Whitney U test. Statistical analyses were performed using SPSS software, version 26 (SPSS Inc., Chicago, IL, USA), and a P<0.05 was considered statistically significant.

## Results

### RF levels in exposed individuals compared to controls

The analysis in Table 1 showed a statistically significant difference in RF levels between the control and exposed groups.

RF levels were lower in the exposed group than in the control group (P=0.005), indicating that exposure was associated with a reduction in RF levels.

### Gender-stratified RF levels: Exposed vs control groups

Table 2 presents the RF levels for males and females in the control and exposed groups. In the controls, females had higher RF levels than males, whereas the opposite was observed in the exposed group, although neither difference was statistically significant. Overall, no statistically significant differences were observed between men and women, except in exposed women compared to control women (P=0.005), suggesting that exposure may exert a stronger effect on RF levels in women. Notably, RF levels were lower in exposed females compared to control females, and the high variability within the exposed female group indicates wide individual differences in response to exposure. These findings indicate a potential sex-specific influence of exposure on RF, with women showing a significant association.

### Clinical complication–stratified RF levels: Exposed vs control groups

Table 3 presents RF levels in control individuals and in exposed individuals stratified by severity of organ-specific involvement in the lung, skin, and eyes.

Lung involvement: Exposed individuals with normal-to-mild lung involvement had lower RF levels compared to controls, with a statistically significant difference (P=0.035). Individuals with moderate-to-severe lung involvement also had lower RF levels, showing a significant difference versus controls (P=0.010). However, when comparing normal-mild versus moderate-severe lung groups, the difference was not significant, suggesting that RF reduction is associated with exposure rather than severity of lung involvement.

**Table 1.** RF levels in exposed individuals compared to controls (n=109)

| Variables | RF | | | |
|---|---|---|---|---|
| | Median | Q1 | Q3 | P |
| Control | 8 | 6.4 | 10.8 | 0.005 |
| Exposed | 6.6 | 5 | 10 | |

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Note: Statistical analysis was performed using the Mann–Whitney U test. P<0.05 were considered statistically significant. P: Control vs exposed group.

**Table 2.** Sex-based comparison of RF levels in control and exposed groups

| Variables | | RF | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | | No. | Median | Q1 | Q3 | P1 | P2 | P3 |
| Control | Male | 70 | 7.65 | 6.1 | 11. | 0.297 | | |
| | Female | 39 | 8.6 | 7 | 10.8 | | | |
| Exposed | Male | 100 | 6.9 | 5.2 | 10 | 0.294 | 0.132 | 0.005 |
| | Female | 39 | 6.2 | 5 | 10.4 | | | |

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Note: Statistical analysis was performed using the Mann–Whitney U test. P<0.05 were considered statistically significant. P1: Female vs male; P2: Male subgroups; P3: Female subgroups.

**Table 3.** Comparison of RF levels across skin, lung, and eye involvement categories

| Lung | RF | | | | | |
|---|---|---|---|---|---|---|
| | No. | Median | Q1 | Q3 | P1 | P2 |
| Control | 109 | 8 | 6.4 | 10.8 | | |
| Normal-mild | 67 | 6.8 | 4.9 | 10.5 | 0.035 | |
| Moderate-severe | 72 | 6.45 | 5.4 | 9 | 0.01 | 0.802 |

| Skin | RF | | | | | |
|---|---|---|---|---|---|---|
| | No. | Median | Q1 | Q3 | P1 | P2 |
| Control | 109 | 8 | 6.4 | 10.8 | | |
| Normal-mild | 131 | 6.5 | 5 | 10 | 0.004 | |
| Moderate-severe | 8 | 8 | 6.2 | 10.1 | 0.863 | 0.375 |

| Eye | RF | | | | | |
|---|---|---|---|---|---|---|
| | No. | Median | Q1 | Q3 | P1 | P2 |
| Control | 109 | 8 | 6.4 | 10.8 | | |
| Normal-mild | 117 | 6.8 | 5.2 | 10 | 0.011 | |
| Moderate-severe | 20 | 6.15 | 5.1 | 9.65 | 0.054 | 0.701 |

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Note: Statistical analysis was performed using the Mann–Whitney U test. P<0.05 were considered statistically significant. P: Control vs normal-mild/moderate-severe; P2: Normal-mild vs moderate-severe.

Skin involvement: RF levels in exposed individuals with normal-to-mild skin involvement were significantly lower than in controls (P=0.004). In the moderate-to-severe skin group, RF levels did not differ significantly from controls or the normal-mild skin group, possibly reflecting the small sample size (n=8) or greater variability in this subgroup.

Eye involvement: RF levels were significantly lower in exposed individuals with normal-to-mild eye involvement (P=0.011) and moderate-to-severe eye involvement (P=0.054) compared to controls. The comparison between the two severity groups was not significant, indicating that RF levels are affected by exposure, regardless of the severity of eye involvement.

Overall, these data suggest that RF levels tend to decrease in exposed individuals across different organ involvements, with statistical significance in most comparisons versus controls. The lack of significant differences between severity groups indicates that exposure itself, rather than the degree of disease, may be the main factor influencing RF levels. High variability in some subgroups, particularly moderate-to-severe cases, highlights individual differences in response to exposure.

## Discussion

In our study, we observed a significant reduction in RF levels in SM-exposed individuals compared to unexposed controls. However, it is important to note that all measured RF levels were within the normal clinical range (<20 IU/mL), and the observed difference, while statistically significant, is not of a magnitude considered clinically significant for disease diagnosis.

RF reduction was consistent across sexes, with a particularly notable effect in exposed women, and across different complications (lung, skin, eye), independent of the clinical severity of damage. These findings suggest that long-term SM exposure may lead to immunological remodeling or suppression rather than classical autoantibody-driven autoimmunity. The decrease in RF, rather than an increase, points toward possible impairment of autoreactive B-cells or a shift in the immune milieu unfavourable for autoantibody production.

However, our results contrast with our study in SM-exposed veterans with severe pulmonary disease, where RF, antinuclear antibodies (ANA), anti-DNA antibodies, and C-reactive protein (CRP) were all significantly elevated decades after exposure [7]. This discrepancy might be explained by differences in sample selection: while their study focused on individuals with advanced lung pathology, our samples may include a wider spectrum of clinical severity. In individuals with more severe organ damage, persistent inflammation triggers ongoing autoantibody production, while in others, chronic immune exhaustion or B-cell dysfunction predominates.

Immunophenotyping studies in SM-exposed veterans with serious problems have revealed long-term alterations in immune cell populations. In one study, peripheral blood analysis showed increased total white blood cell (WBC) and lymphocyte counts in exposed individuals versus controls [8].

Another analysis of innate immune function ~30 years after exposure showed preserved phagocytosis, complement activity, and NBT-reducing ability, but a decrease in α-globulin, possibly implicating α-antitrypsin deficiency in the chronic pathology of these veterans [9]. These findings align with the notion that some systems remain functional, while others undergo subtle long-term adaptation or damage.

Paraclinical reviews of Iranian SM survivors have also documented positive RF and ANA, reduced helper T-cell and natural killer (NK)-cell numbers, and dysregulated immunoglobulins [10].

This study supports the idea that autoantibody perturbations are common in SM-exposed cohorts. However, the direction and magnitude likely depend on individual factors, such as exposure dose, disease severity, and time since exposure.

The role of oxidative stress is especially pertinent: data analysis of SM-exposed veterans found persistent elevation of lipid peroxidation (malondialdehyde), increased catalase activity, and reduced glutathione even decades after exposure [11, 12].

Chronic oxidative stress can impair B-cell viability, promote immune senescence, or disrupt germinal center reactions, potentially contributing to lower RF production rather than promoting autoimmunity [13, 14].

A key question arising from our data is whether the observed reduction in RF-IgM reflects a pathophysiologically meaningful state, such as B-cell dysfunction or immune exhaustion, or if it is merely an epiphenomenon without direct clinical consequences. Several lines of evidence favor a dysfunctional/adaptive model rather than benign incidental findings. First, the suppression was remarkably consistent across distinct organ complication groups (lung, skin, and eye) and severity strata, suggesting it is a systemic, exposure-linked trait rather than a random fluctuation. Second, the parallel evidence of long-term oxidative stress [11, 12] and altered immune cell populations [8-10] in SM survivors provides a plausible biological platform for B-cell impairment, as oxidative damage is known to compromise lymphocyte function and survival [13, 14]. Third, the stronger suppression observed in women hints at a regulated, sex-dimorphic process, which is less characteristic of a non-specific epiphenomenon. While we cannot definitively rule out that low RF is a passive marker without clinical impact, its association with other persistent immunological disturbances makes this less likely. Therefore,

we posit that the RF reduction is more consistent with a state of chronic immune adaptation or antigen-driven B-cell exhaustion resulting from decades of managing SM-induced tissue damage and inflammation. This state may represent one end of a spectrum of immune outcomes in SM survivors, contrasting with the elevated autoantibody profiles observed in cohorts with more severe active inflammatory disease [7].

Skin pathology in SM survivors may also be relevant. A large retrospective cohort study found a much higher prevalence of chronic dermatoses (itching, blistering, xerosis, and excoriated dermatitis) in exposed individuals compared to controls [15]. Chronic skin inflammation and tissue remodeling may contribute to systemic immune dysregulation.

From an epidemiological standpoint, a World Health Organization (WHO) and International League against Rheumatism Community Oriented Program for Control of Rheumatic Diseases (WHO-ILAR COPCORD) study among chemical-warfare veterans reported a significant burden of rheumatic complaints, including possible rheumatoid arthritis, though definitive serological confirmation was often limited [16].

That study underscores that clinical rheumatic disease is not uncommon in SM survivors, even if RF levels in some cohorts (like ours) are lower. Based on these findings, several mechanistic hypotheses have emerged: 1) B-cell dysfunction or antigen-driven exhaustion: Chronic SM-induced inflammation and oxidative stress may lead to functional impairment, selective depletion, or exhaustion of autoreactive B-cell clones, resulting in a diminished capacity to produce RF-IgM and potentially other antibodies. 2) Senescence and microenvironment disruption: SM-induced oxidative stress and senescence in stromal or immune-supporting tissues may degrade the architecture required for autoreactive B-cell survival. 3) Unfavorable cytokine milieu: Exposure may shift cytokine balance toward a suppressed, exhausted immune state, rather than a continuously activated, autoantibody-promoting environment in the long term. 4) Antioxidant depletion and oxidative stress: Persistent oxidative damage may compromise B-cell function or survival, explaining reduced RF despite immune system activation in other compartments.

The limitations of our study are as follows: the exposed cohort is likely heterogeneous in clinical severity, which may obscure subgroups with different immune trajectories. Also, some subgroups (e.g. moderate-to-severe skin involvement) had small sample sizes, limiting statistical power. Furthermore, the ELISA method employed in this study specifically measured IgM isotype RF (RF-IgM). We did not assess other RF isotypes (e.g. IgG or IgA), which may have distinct biological roles and clinical associations.

Future studies on SM survivors should longitudinally track autoantibody trajectories (RF, ANA, anti-DNA) and include detailed B-cell phenotyping to assess subset depletion or dysfunction. Parallel profiling of cytokines, oxidative stress, and senescence markers would clarify the immune context of RF suppression. The stronger RF reduction in women warrants investigation of sex-specific mechanisms, while linking immunological changes to clinical outcomes could define the prognostic significance of altered RF in this population.

## Conclusion

Our findings of reduced RF levels in long-term SM–exposed individuals challenge the assumption that SM exposure inevitably leads to autoantibody-driven autoimmunity. Instead, our data, together with the broader literature, suggest a more complex picture of immune remodeling, possibly involving suppression, B-cell dysfunction, or adaptation, rather than persistent autoimmunity. Contrasting results in other cohorts (with raised RF) highlight heterogeneity among survivors and underscore the need for mechanistic, longitudinal, and stratified research to fully understand the immunologic legacy of SM.

## Ethical Considerations

### Compliance with ethical guidelines

This study was approved by the Ethics Committee of Shahed University, Tehran, Iran (Code: Shahed.REC.1399.091).

### Funding

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### Authors' contributions

Conceptualization and investigation: Tooba Ghazanfari; Writing the original draft: Tahereh Jamali; Supervision, review and editing: Sussan Kaboudanian Ardestani.

### Conflicts of interest

The author declared no conflict of interest.

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