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Review Article

Serum levels of Galectin-1 and Galectin-3 for monitoring rheumatoid arthritis severity: a meta-analysis

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Abstract: Background: Galectin-1 (Gal-1) and galectin-3 (Gal-3) have emerged as promising biomarkers for diagnosing and managing rheumatoid arthritis (RA). This study aims to synthesize current evidence through a systematic review and meta-analysis to evaluate the clinical effectiveness of serum Gal-1 and Gal-3 as biomarkers for monitoring disease severity and predicting clinical outcomes in RA patients. Methods: We conducted a comprehensive literature search in PubMed, Web of Science, and the Cochrane Library, including studies published up to March 2024. Eleven observational studies were selected based on predefined inclusion criteria. The risk of bias in these studies was assessed using the Cochrane Handbook for Systematic Reviews of Interventions. Data synthesis and meta-analysis were performed using RevMan 5.3 software, with the study protocol registered at INPLASY (ID: 202460103). Results: The meta-analysis included 1213 participants (comprising 809 RA patients and 404 healthy controls) from 11 studies. Serum levels of Gal-1 and Gal-3 were significantly higher in RA patients compared to controls (MD=25.09 ng/ml, 95% CI: 24.18-26.00 ng/ml, P<0.00001; MD=30.51 ng/ml, 95% CI: 29.10-31.93 ng/ml, P<0.00001). Moreover, Gal-1 levels exhibited a positive correlation with RA disease activity markers, such as the Erythrocyte Sedimentation Rate (ESR) and the Disease Activity Score 28 (DAS28). The analysis demonstrated a correlation coefficient of r=0.24 (95% CI: 0.14-0.33, P<0.00001) between Gal-1 and RA disease activity, highlighting a notable association. Similarly, Gal-3 showed significant positive correlations with ESR (r=0.29, 95% CI: 0.18-0.40, P<0.00001), DAS28 (r=0.25, 95% CI: 0.13-0.37, P<0.00001), and C-reactive protein (CRP) (r=0.15, 95% CI: 0.05-0.26, P<0.00001). The overall correlation between circulating Gal-3 levels and RA disease severity indices was r=0.23 (95% CI: 0.16-0.29, P<0.00001). Conclusion: Gal-1 demonstrates significant potential as a biomarker for diagnosing and managing RA. Monitoring Gal-1 and Gal-3 levels may provide valuable insights into early disease assessment and progression, potentially improving treatment outcomes for RA patients.

Keywords: Galectin-1, Galectin-1, disease activity, rheumatoid arthritis, meta analysis

Introduction

Rheumatoid arthritis (RA) is a chronic autoimmune disorder that predominantly affects the joints, causing significant morbidity and disability worldwide [1]. This condition is characterized by inflammation of the synovial membrane, resulting in pain, swelling, stiffness, and potentially severe joint deformity and functional impairment [2]. RA has systemic effects, affecting other organs such as the skin, blood vessels, heart, lungs, and muscles, making it a complex and multifaceted disease [3]. While the exact etiology remains elusive, RA is thought to result from a complex interplay of genetic predisposition, environmental triggers, and immune dysfunction. Potential contributing factors, including infections, hormonal imbalances, and psychological stress, are thought to play a role in disease onset [4]. Given the progressive nature of RA, early diagnosis and intervention are critical to preventing cumulative joint damage and preserving patients' quality of life. A deeper understanding of the disease's pathogenesis is crucial for developing innovative diagnostic markers and treatment approaches.

The galectin family of proteins, including Gal-1 and Gal-3, is known for their binding affinity to

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beta-galactoside sugars [5]. Both Gal-1 and Gal-3 are involved in a range of cellular processes, including cell growth, adhesion, apoptosis, and inflammation [6]. These proteins have been implicated in the regulation of immune responses, inflammation, and cancer progression [7]. In the context of RA, Gal-1 and Gal-3 have emerged as important immunoregulatory factors with potential roles in disease diagnosis and pathogenesis [8]. Growing evidence suggests a correlation between RA disease activity and the expression of Gal-1 and Gal-3. In particular, research has shown a close correlation between Gal-1 levels and RA disease activity, suggesting its utility as a diagnostic marker [9]. In addition, Gal-3 plays a key role in regulating inflammatory responses and apoptosis, which are integral to arthritis progression and pathophysiology [10]. However, the exact association between Gal-1/3 expression and RA disease activity remains unclear, with the existing literature offering variable and sometimes conflicting results.

In this study, we performed a systematic review and meta-analysis of available literature to evaluate the diagnostic significance of Gal-1/3 in RA and to explore their correlation with disease activity. Our goal is to provide a clearer understanding of the roles of these proteins in RA, which could inform future diagnostic and therapeutic approaches.

Materials and methods

Search strategy

This study followed the PRISMA guidelines for systematic reviews and meta-analyses [11]. Two investigators independently searched the PubMed, Web of Science, and Cochrane Library databases for eligible publications. The search spanned from the establishment of the databases to March 2024. We focused on English-language articles related to RA and the biomarkers Gal-1 and Gal-3. The quality of the publications was evaluated using the Newcastle-Ottawa Scale for observational research. The literature search employed the following terms: “Rheumatoid Arthritis”, “Arthritis, Rheumatoid”, “RA”, “Galectin-1”, “Galectin-3”, “Galactoside-Binding Lectin 1”, “Gal-3”, “Galactoside-Binding Lectin 3”, and “Gal-1”. Additionally, we reviewed the references of selected studies to identify any further relevant literature. The meta-analysis protocol was prospectively registered at INPLASY (International Platform of Registered Systematic Review and Meta-analysis Protocols, 202460103).

Study selection

Inclusion criteria: (1) Participants who met internationally recognized diagnostic criteria for RA; (2) Studies involving adult populations (age ≥18 years); (3) Observational investigations.

Exclusion criteria: (1) Secondary literature (systematic reviews, meta-analyses, narrative reviews); (2) Duplicate publications or studies with overlapping datasets; (3) Non-research publications (e.g., case reports, commentaries, and letters); (4) Studies lacking sufficient primary data for meaningful analysis. Any disagreements during the literature screening process were resolved through third-party arbitration by an independent reviewer to ensure methodological rigor.

Data extraction and quality assessment

Data extraction was performed by two independent researchers, who systematically collected specific information from each study. The data retrieved included patient demographics, study design, sample size, presence of Gal-1 and Gal-3 in RA patients, disease activity measures, and relevant outcomes. Disagreements between the researchers were resolved through discussion and consensus.

An extraction table was employed to organize the collected data, ensuring consistency and accuracy. To evaluate the quality and risk of bias in the included articles, we followed the criteria outlined in the Cochrane Handbook for Systematic Reviews of Interventions. The evaluation encompassed seven methodological domains: (1) random allocation procedures, (2) adequacy of allocation concealment, (3) effectiveness of participant/provider blinding, (4) objectivity of outcome assessor blinding, (5) management of missing outcome data, (6) strategies for minimizing selective reporting, and (7) identification of potential confounding factors. Each criterion was stratified into three

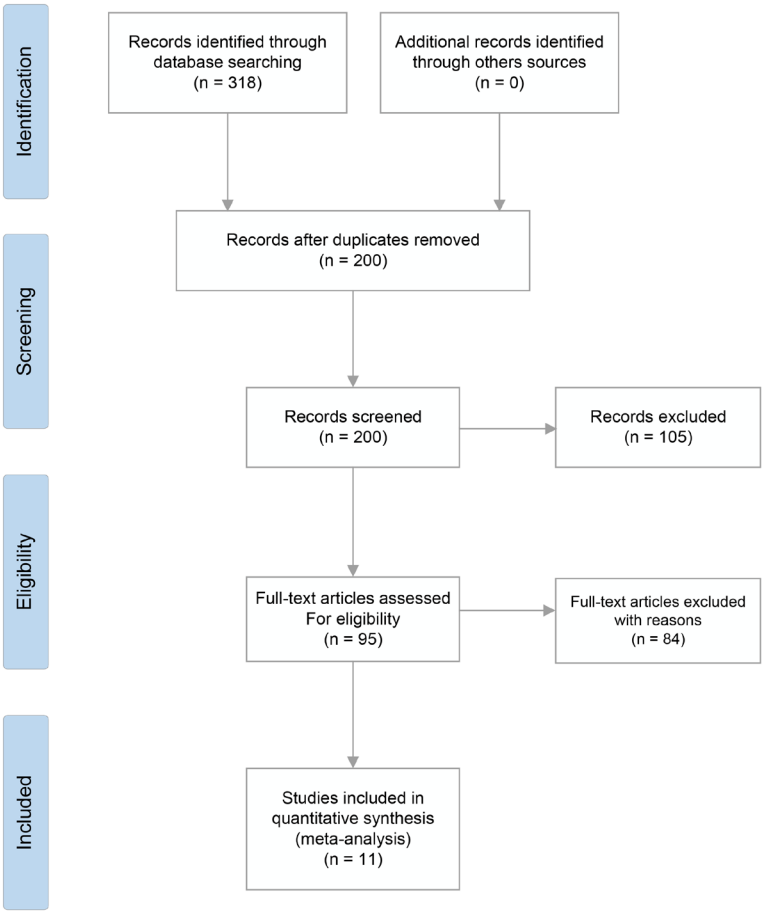

Figure 1. A schematic diagram demonstrating the process of study selection for the analysis.

classifications: low, high, or unclear risk of bias, facilitating a thorough quality assessment of the included studies.

Data analysis

Data analysis was conducted using RevMan 5.3. For continuous variables, we calculated the mean difference (MD) and corresponding 95% confidence intervals (CI). Heterogeneity was assessed using the Q test and the I^2 statistic, with a significance threshold set at $P<0.10$. In meta-analysis, the choice of model hinges on the degree of heterogeneity. The fixed-effects model is typically employed when heterogeneity is low, while the random-effects model is used for studies with high heterogeneity. In this study, based on the observed heterogeneity as defined by the I^2 value, a random-effects model was applied to estimate the summary effect when I^2 fell within the 75-100% (high-heterogeneity) range. Subgroup analyses were performed to explore potential sources of heterogeneity and evaluate the robustness of our findings across different subpopulations or study characteristics. Sensitivity analyses were also conducted to evaluate the stability of our results, by excluding individual studies to determine their impact on the overall meta-analysis outcomes. Fisher's Z-transformation was used to standardize the correlation coefficients from individual studies, enabling the calculation of weighted averages and 95% confidence intervals (CI).

Results

Study selection

Figure 1 illustrates the search results and the systematic process of study selection. A total of 318 articles were identified through searches in the PubMed, Web of Science, and Cochrane Library databases. After excluding 118 duplicates and 105 studies deemed irrelevant based on titles and abstracts, we reviewed the full texts of the remaining 95 studies. Following a thorough evaluation, 84 articles were excluded for various reasons, leaving 11 observational studies for inclusion in our meta-analysis. A summary of the basic characteristics of each included study is presented in **Table 1**.

Assessment of study quality

Methodological quality was evaluated using the Cochrane Collaboration's standardized bias assessment framework (**Figure 2**). All studies reported adequate procedural transparency regarding randomization sequence generation. Furthermore, completeness of reporting was achieved via protocol adherence audits to minimize selective outcome reporting. Notably, over 75% of the studies implemented outcome evaluator masking protocols and accounted for par-

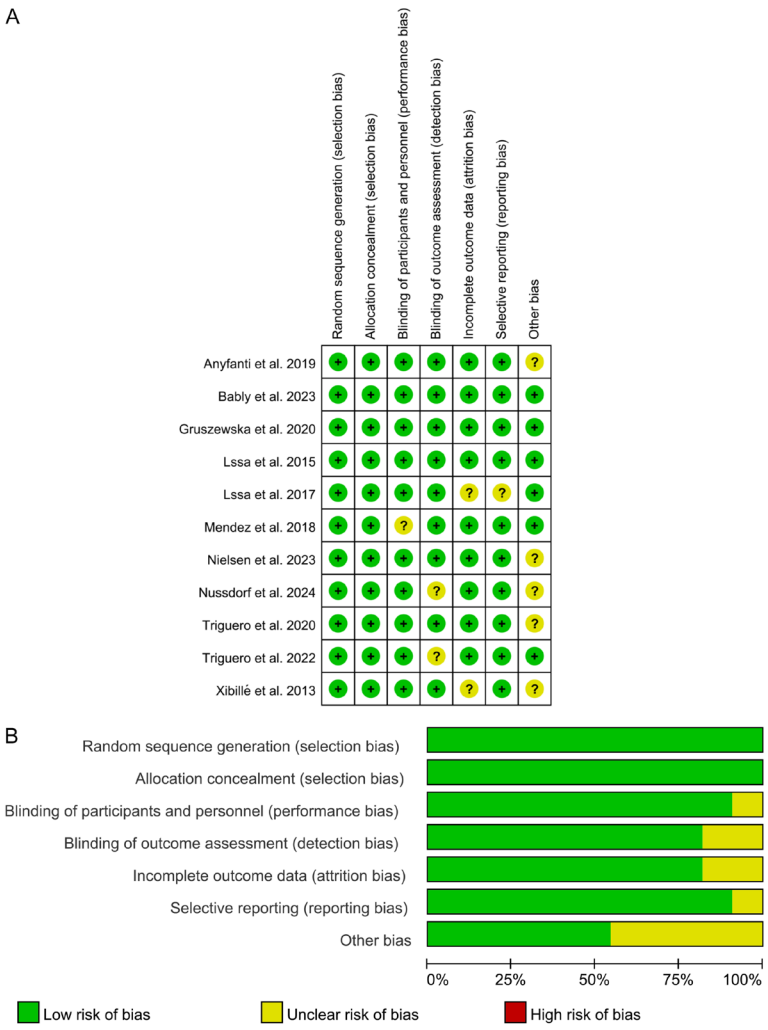

Figure 2. Graphic representation of the risk of bias and summary of the risk of bias. A: Bias risk graph: evaluation displaying the percentage of all studies with potential bias risks in the research. B: Summary of the risk of bias: assessment of all bias items for each study.

ticipant attrition, enhancing the overall methodological rigor.

Serum Gal-1 levels in RA patients and healthy controls

Five publications, involving 296 RA individuals and 185 healthy controls, reported Gal-1 levels in RA patients and healthy controls. The serum Gal1 concentration was significantly elevated in RA patients compared to healthy controls (MD=25.09 ng/ml, 95% CI: 24.18-26.00 ng/ml, P<0.00001, **Figure 3**). This finding indicates that serum Gal-1 levels are markedly elevated in RA patients, suggesting its potential as a biomarker or diagnostic indicator for the disease.

Serum Gal-3 levels in RA patients and healthy controls

This meta-analysis included five publications with 389 RA patients and 219 healthy controls for the assessment of serum Gal-3 levels. RA patients exhibited a marked growth in serum Gal-3 concentration compared to controls (MD=30.51 ng/ml, 95% CI: 29.10-31.93 ng/ml, P< 0.00001, **Figure 4**). This indicates that elevated Gal-3 levels may serve as a promising biomarker or diagnostic marker for RA.

Relationship between Gal1 and RA disease activity score

The meta-analytic framework incorporated Fisher's Z-transformation for standardization of the extracted correlation coefficients, followed by inverse-variance weighted pooling to estimate precision-adjusted 95% CIs. This dual-stage analytical protocol enabled cross-study harmonization of effect sizes, culminating in a synthesized correlation estimate with quantified statistical uncertainty. The association between Gal-1 levels and ESR was evaluated in two studies. A positive association was found between Gal-1 and ESR (r=0.30, 95% CI: 0.14-0.46, P=0.0003), as well as DAS28 (r=0.26, 95% CI: 0.11-0.42, P=0.0006). However, no significant association was observed between Gal-1 and CRP (r=0.12, 95% CI: -0.06-0.30, P=0.19). The comprehensive analysis of Gal-1 levels and RA disease activity yielded a correlation coefficient of r=0.24 (95% CI: 0.14-0.33, P<0.00001) (**Figure 5**).

Relationship between Gal3 and RA disease activity score

The relationship between Gal-3 levels and indices of RA disease activity, including ESR,

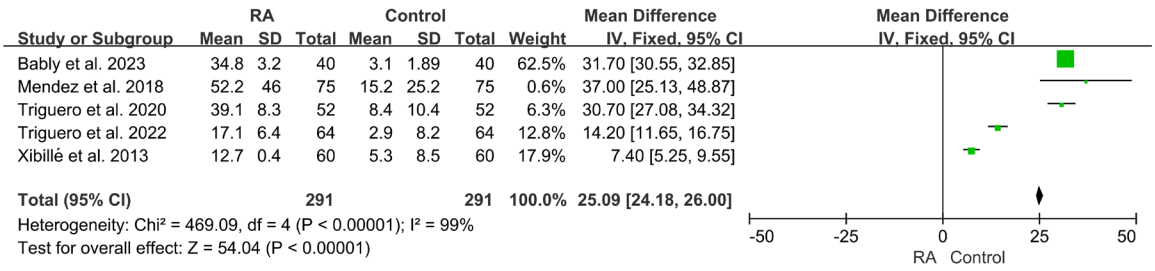

Figure 3. Forest-plot of the serum Gal1 levels in RA patients and healthy controls. Gal-1: Galectin-1; RA: rheumatoid arthritis.

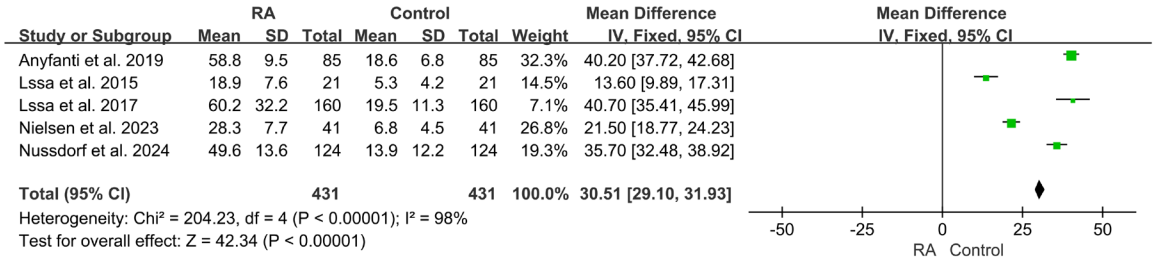

Figure 4. Forest-plot of the serum Gal3 levels in RA patients and healthy controls. Gal-3: Galectin-1; RA: rheumatoid arthritis.

DAS28, and CRP, was assessed across multiple studies. Our analysis revealed a significant positive correlation between Gal-3 and ESR (r=0.29, 95% CI: 0.18-0.40, P<0.00001), DAS28 (r=0.25, 95% CI: 0.13-0.37, P<0.00001), and CRP (r=0.15, 95% CI: 0.05-0.26, P< 0.00001), as illustrated in **Figure 6**. The overall analysis of Gal-3 levels and RA disease activity yielded a correlation coefficient of r=0.23, 95% CI: 0.16-0.29, P<0.00001 (**Figure 6**). These findings suggest a potential link between elevated Gal-3 levels and increased RA disease activity.

Risk of bias

Considering the limited number of studies, with each analysis involving fewer than 10 studies for individual risk factors, the sample sizes were insufficient to achieve sufficient statistical power to distinguish between random fluctuations and true asymmetry in the funnel plots. As such, the assessment of funnel plots for estimating the risk of bias was not conducted due to the insufficient number of data points.

Discussion

In this study, we conducted a systematic review and meta-analysis to investigate the relationship between Gal-1/3 expression and disease activity in RA, aiming to provide valuable insights into clinical diagnosis and treatment. We collected data on Gal-1/3 expression levels and disease activity measures from relevant studies and performed a meta-analysis to determine the overall correlation between these galectins and disease activity. The results revealed a notable positive correlation between the expression levels of both Gal-1 and Gal-3 and the disease activity in RA patients, suggesting that elevated levels of these galectins are closely associated with increased disease activity. This implies that higher expression of Gal-1/3 might serve as crucial markers of more aggressive RA symptoms and progression.

Both Gal-1 and Gal-3 are well-recognized as key inflammatory factors in RA, deeply implicated in the activation and regulation of immune cells, as well as in the complex inflammatory cascade [16]. Research has highlighted their critical role in the pathogenesis of RA, with high expression levels frequently observed in the synovial tissue of affected individuals, where they are closely linked to disease activity [17]. The prominent expression of Gal-1 and Gal-3 in synovial tissue has sparked interest in their potential as biomarkers for the diagnosis of RA and tracking disease progression. No-

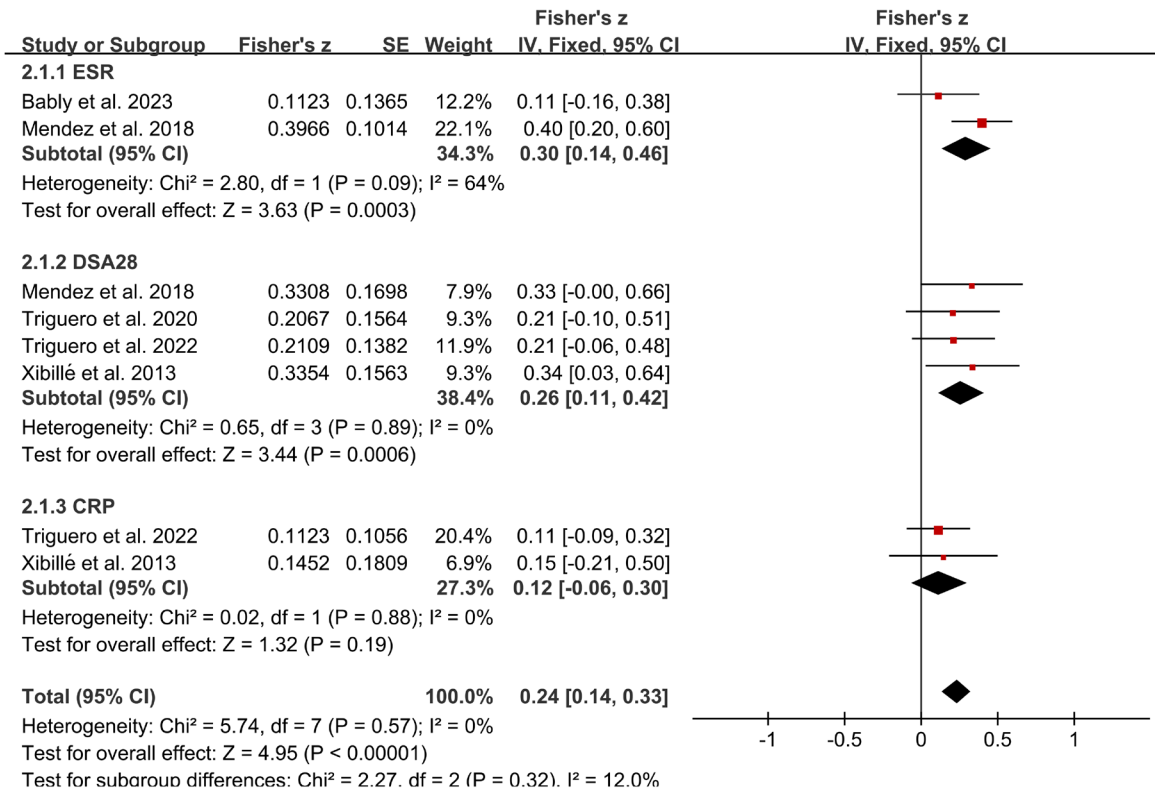

Figure 5. Forest chart of the relationship between disease activity and serum Gal-1 levels in RA patients. Gal-1: Galectin-1; ESR: erythrocyte sedimentation rate; DAS28: Disease Activity Score; RA: rheumatoid arthritis; CRP: C-reactive protein.

tably, recent studies suggest that measuring serum levels of Gal-1 and Gal-3 can be instrumental in identifying RA patients, particularly in the early stages of the disease, when conventional clinical markers may not be discernible [18]. Monitoring fluctuations in Gal-1 and Gal-3 levels can also provide valuable insights into treatment efficacy and the likelihood of disease recurrence [19]. In addition, Gal-1 and Gal-3 have been proposed to influence joint damage and repair by adhering to vascular endothelial cells, promoting angiogenesis, and driving inflammatory responses [20]. Furthermore, these galectins interact with a variety of inflammatory mediators and cytokines, further contributing to the complex pathological mechanisms underlying RA [21, 22].

Our results clearly established an association between both Gal-1 and Gal-3 and key indicators of RA disease activity. Specifically, we observed a positive correlation between these galectins and disease activity, as measured by indices such as DAS28 and ESR, which are standard tools for evaluating the severity of RA and guiding therapeutic inventions [23, 24]. These findings have important clinical implications. The potential of Gal-1 and Gal-3 as reliable biomarkers for disease activity could enhance early RA detection, predict disease progression, and assess treatment response. Additionally, targeting Gal-1 and Gal-3 in therapeutic strategies may offer innovative avenues for managing RA and improving patient outcomes.

In addition, we explored the diagnostic and prognostic value of Gal-1 in RA. Our findings suggest that Gal-1 holds promise as both a diagnostic and prognostic marker for RA. Mendez et al. reported an AUC of 0.82 for Gal-1 in distinguishing RA patients from healthy controls, while Barly et al. identified Gal-1 as a potential biomarker with an exceptionally high AUC of 0.999. Triguero et al. also reported AUC values of 0.783 and 0.761 for RA prediction based on Gal-1. While the correlation between Gal-1 and RA disease activity is promising, further analyses are required to validate these findings. The efficacy of Gal-1 as a biomarker

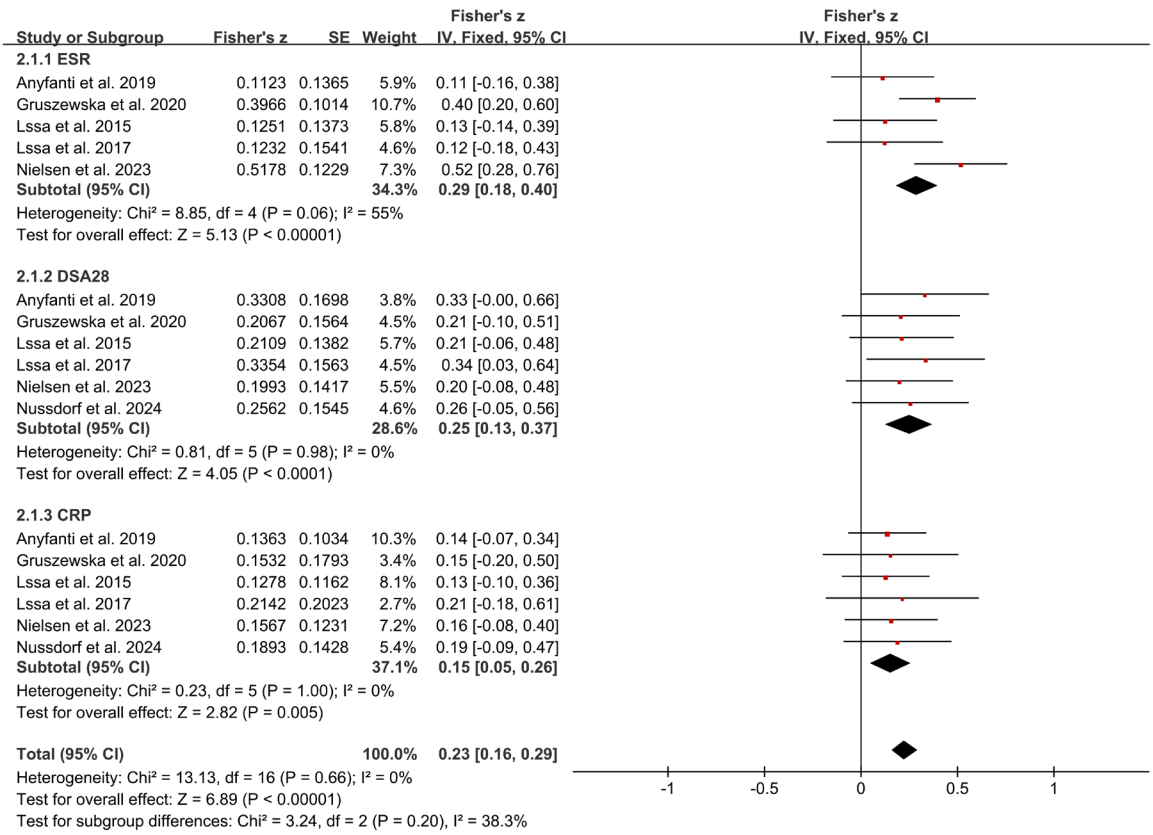

Figure 6. Forest chart of the relationship between disease activity and serum Gal-3 levels in RA patients. Gal-3: Galectin-3; ESR: erythrocyte sedimentation rate; DAS28: Disease Activity Score; CRP: C-reactive protein.

Table 1. Key features of the observational studies that were part of the meta-analysis

Author (year)	Detection method	Cases of RA	Controls	Age* (years)	RA duration
Mendez et al. (2018) [12]	ELISA	80	29	45 (39-66)	8.5 (1-28)#
Triguero et al. (2020)[15]	ELISA	52	24	52 (60-65)	4.6 (2.5-8.25)&
Triguero et al. (2022) [14]	ELISA	64	52	55 (42-67)	4.9 (2.2-9.4)&
Xibillé et al. (2013) [25]	ELISA	60	60	45.1±10.3	NA
Bably et al. (2023) [13]	ELISA	40	20	42.65±12.90	7.9±3.1#
Lssa et al. (2017) [26]	ELISA	160	119	53 (42-63)	14 (11-20)
Nussdorf et al. (2024) [27]	ELISA	124	NA	56.5±12.26	6.8
Anyfanti et al. (2019) [28]	ELISA	85	39	59.6±11.6	10 (5-18)
Gruszewska et al. (2020) [29]	Chemiluminescent microparticle immunoassay	82	30	58.8 (20-85)	NA
Nielsen et al. (2023) [30]	ELISA	41	15	50 (42-63)	3-6
Lssa et al. (2015) [31]	ELISA	21	16	62 (46-69)	4.3 (3.3-5.06)

Data were represented as median (range) or mean ± SD; # represents years; & represents months. ELISA stands for enzyme-linked immunosorbent assay; NA means not available; RA stands for rheumatoid arthritis.

must be evaluated in larger patient populations and prospective studies. This research could ultimately lead to improved strategies for managing RA and enhancing patients' quality of life.

Our study has several limitations that should be considered. First, variations in study design, sample size, and patient disease status may limit the accuracy of interpreting data compre-

hensively. Moreover, the inclusion of only English-language studies may have introduced a language bias. Given the limited number of published studies in this field, the small number of reports included in this meta-analysis may lead to insufficient statistical power and reduce the reliability of the results. While meta-analysis may have limitations when the available literature is sparse, it still provides valuable insights for clinical decision-making and future research when appropriate measures are taken.

In conclusion, our study highlights the significant association between Gal-1/3 expression levels and disease activity in RA patients. Both Gal-1 and Gal-3 exhibit strong potential as biomarkers for the diagnosis and treatment of RA. Monitoring the expression levels of Gal-1 and Gal-3 can facilitate early diagnosis and assessment of disease activity, potentially improving treatment strategies and patient outcomes. Nevertheless, further research is necessary to validate these findings and explore the underlying mechanisms of Gal-1 and Gal-3 in RA, which could pave the way for enhanced diagnostic and therapeutic approaches for RA patients.

Disclosure of conflict of interest

None.

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