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THE INFLUENCE OF HOMOCYSTEINE ON THE PATOGENESIS OF JUVENIL RHEUMATOID ARTRITIS IN CHILDREN

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***Key words**: juvenile rheumatoid arthritis, homocysteine, disease activity, chronic inflammation, clinical forms, systemic manifestations, pediatric rheumatology, endothelial dysfunction, biomarkers.*

ABSTRACT
Juvenile rheumatoid arthritis is a chronic inflammatory disease in which metabolic factors may contribute to disease severity. This study evaluated serum homocysteine levels in 67 children with juvenile rheumatoid arthritis (mean age: 12.4 ± 3.7 years) and analyzed their association with age, disease duration, clinical form, and disease activity. The mean serum homocysteine concentration was 6.6 ± 2.6 µmol/L. Homocysteine levels showed no significant association with age and only a weak correlation with disease duration ($r = 0.249$; $p = 0.043$). In contrast, homocysteine concentration differed significantly according to clinical form ($p = 0.005$) and increased with higher disease activity ($p < 0.001$). A moderate positive correlation was observed between disease activity and homocysteine level ($r = 0.457$; $p < 0.001$). These findings suggest that serum homocysteine reflects inflammatory burden and clinical severity in juvenile rheumatoid arthritis and may have potential value as a disease activity biomarker.

BOLALARDA GOMOTSISTEINNING YUVENIL REVMATOID ARTRIT PATOGENEZIGA TA'SIRI

***Kalit so'zlar**: yuvenil revmatoid artrit, gomotsistein, kasallik faolligi, surunkali yallig'lanish, klinik shakllar, tizimli namoyonlar, bolalar revmatologiyasi, endotelial disfunktsiya, biomarkerlar.*

ANNOTATSIYA
Yuvenil revmatoid artrit – bu surunkali yallig'lanish kasalligi bo'lib, unda metabolik omillar kasallikning og'irlik darajasiga ta'sir ko'rsatishi mumkin. Ushbu tadqiqotda yuvenil revmatoid artrit bilan og'rigan 67 nafar bolada (o'rtacha yosh: 12,4 ± 3,7 yil) qon zardobidagi gomotsistein darajasi baholandi hamda uning yosh, kasallik davomiyligi, klinik shakli va kasallik faolligi bilan bog'liqligi tahlil qilindi. Qon zardobidagi gomotsisteinning o'rtacha konsentratsiyasi 6,6 ± 2,6 µmol/L ni tashkil etdi. Gomotsistein darajasi yosh bilan ishonchli bog'liqlik ko'rsatmadi va kasallik davomiyligi bilan faqat kuchsiz korrelyatsiya aniqlandi ($r = 0,249$; $p = 0,043$). Aksincha, gomotsistein konsentratsiyasi klinik shakliga qarab ishonchli farqlandi ($p = 0,005$) va kasallik faolligi oshishi bilan sezilarli ravishda ortdi ($p < 0,001$). Kasallik faolligi va gomotsistein darajasi o'rtasida o'rtacha kuchdagi musbat korrelyatsiya kuzatildi ($r = 0,457$; $p < 0,001$). Ushbu natijalar gomotsisteinning yuvenil revmatoid artritda yallig'lanish yuklamasi va klinik og'irlik darajasini aks ettirishi hamda kasallik faolligining potentsial biomarkeri sifatida ahamiyatga ega bo'lishi mumkinligini ko'rsatadi.

INTRODUCTION

Understanding the role of homocysteine in the development and progression of juvenile rheumatoid arthritis (JRA) represents a timely and clinically significant area of medical research. Investigating the involvement of homocysteine in the pathogenesis of JRA provides deeper insight into the inflammatory and autoimmune mechanisms underlying the disease, which is essential for the development of more effective and targeted therapeutic strategies. Given the chronic nature of JRA and its systemic manifestations, identifying modifiable biochemical factors remains a priority in pediatric rheumatology. Beyond its role in inflammatory processes, homocysteine has been extensively studied in relation to cardiovascular pathology. Elevated homocysteine levels are strongly associated with an increased risk of cardiovascular diseases and endothelial dysfunction, potentially leading to structural and functional vascular impairment [2,4].

These effects are particularly relevant in patients with rheumatoid arthritis, who are known to have a higher prevalence of cardiovascular comorbidities compared to the general population.

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High concentrations of homocysteine have been linked to the development of heart and vascular disorders through mechanisms involving oxidative stress, endothelial damage, and prothrombotic states. Since individuals with rheumatoid arthritis frequently exhibit concurrent vascular abnormalities, exploring the relationship between homocysteine levels and disease activity may open new perspectives for integrated therapeutic approaches aimed at managing both articular inflammation and cardiovascular risk [3].

Moreover, homocysteine plays a role in immune system modulation, including the activation of immune cells and the regulation of cytokine production. Its involvement in pro-inflammatory and immune-mediated pathways is particularly relevant for understanding autoimmune processes in juvenile rheumatoid arthritis [6]. Elucidating these mechanisms may contribute to the identification of novel targets for immunomodulatory therapy.

In addition, homocysteine has potential clinical value as a biomarker. Its measurement could enhance early diagnosis, improve disease monitoring, and assist in predicting disease progression and treatment response in pediatric patients with JRA [5].

Purpose of the study.

The aim of this study was to evaluate the role of homocysteine in the pathogenesis of juvenile rheumatoid arthritis through the analysis of cysteine–homocysteine (CS) test outcomes, thereby clarifying its clinical and pathogenetic significance.

METHODOLOGY

This study evaluated serum homocysteine levels in 67 patients diagnosed with juvenile rheumatoid arthritis (JRA). The mean age of the patients was 12.4 ± 3.7 years, with an age range covering childhood and early adolescence. The study population consisted of 39 boys (58.2%) and 28 girls (41.8%).

All patients underwent a comprehensive clinical and laboratory examination in accordance with standard rheumatological protocols. Disease activity and clinical course were assessed using established criteria for JRA. Blood samples were collected under standardized conditions, and serum homocysteine concentration was determined using routine biochemical methods. The obtained values were expressed in μmol/L.

To explore potential associations, homocysteine levels were analyzed in relation to sex, age, and disease characteristics, including disease activity and type of clinical course. Statistical analysis included descriptive statistics, comparison of means, and correlation analysis. A p-value of < 0.05 was considered statistically significant.

RESULTS AND DISCUSSION

Autoimmune involvement of the nervous system was assessed indirectly through indicators of B-lymphocyte activation, reflecting the systemic immune dysregulation characteristic of JRA. Given the known role of endothelial dysfunction in autoimmune and inflammatory diseases, particular attention was paid to serum homocysteine levels. Hyperhomocysteinemia is widely recognized as an independent risk factor for endothelial damage and ischemic vascular processes, which may contribute to both systemic and neurological complications.

Homocysteine levels were measured in all 67 patients. The mean serum homocysteine concentration in the study population was 6.6 ± 2.6 μmol/L, remaining within reference values for most patients. Analysis by sex demonstrated no statistically significant differences between boys and girls ($t = 0.108$; $p = 0.914$), suggesting that sex did not have a substantial influence on homocysteine metabolism in this cohort.

Correlation analysis between patient age and serum homocysteine concentration revealed a weak positive relationship ($r = 0.160$). However, this association did not reach statistical significance ($p = 0.195$), indicating that age alone was not a decisive factor influencing homocysteine levels in patients with JRA.

Further analysis involved comparison of homocysteine levels across predefined age groups. Patients in the middle and older age groups demonstrated higher mean homocysteine concentrations (6.9 ± 2.7 μmol/L) compared with patients in the younger age group (5.8 ± 1.9 μmol/L). Despite this observed trend, the difference between the groups was not statistically significant, which may be attributed to the relatively small sample size and interindividual variability (Table 1).

The absence of statistically significant differences across sex and age groups suggests that homocysteine levels in JRA patients may be influenced more by disease-related mechanisms than by demographic factors alone. Chronic inflammation, immune activation, and possible metabolic alterations associated with autoimmune disease may play a role in endothelial dysfunction even in pediatric populations.

These findings highlight the importance of monitoring vascular risk markers in children with JRA, even in the absence of overt cardiovascular symptoms. Further studies with larger sample sizes and longitudinal follow-up are required to clarify the clinical significance of homocysteine as a potential biomarker of vascular and neurological involvement in juvenile rheumatoid arthritis.

Table 1. Serum Homocysteine Levels by Age Group

Age group	Serum homocysteine (μmol/L)	95% CI	n	F	p
	Mean ± SD				
6–10 years	5.8 ± 1.9	4.9–6.7	21	1.524	0.226
11–14 years	6.9 ± 2.7	5.7–8.2	20		
≥15 years	6.9 ± 2.7	5.8–8.0	26		

Note: Differences between age groups were analyzed using one-way analysis of variance (ANOVA). No statistically significant differences were observed ($p > 0.05$).

Analysis of homocysteine levels by age group

As shown in Table 1, serum homocysteine concentrations were analyzed across three age categories. In children aged 6–10 years, the mean homocysteine level was 5.8 ± 1.9 μmol/L (95% CI: 4.9–6.7; $n = 21$). Higher mean values were observed in the 11–14 years group (6.9 ± 2.7 μmol/L, 95% CI: 5.7–8.2; $n = 20$) and in patients aged 15 years and older (6.9 ± 2.7 μmol/L, 95% CI: 5.8–8.0; $n = 26$).

Despite the observed increase in mean homocysteine concentration with age, one-way analysis of variance (ANOVA) did not reveal statistically significant differences between the age groups ($F = 1.524$; $p = 0.226$). This indicates that, within the studied cohort, age-related differences in homocysteine levels were not sufficiently pronounced to reach statistical significance. The relatively wide confidence intervals and overlap between groups suggest substantial interindividual variability.

These findings are consistent with earlier correlation analysis, which demonstrated only a weak, non-significant association between chronological age and serum homocysteine concentration.

Relationship Between Homocysteine Level and Disease Duration

In contrast to age-related analysis, assessment of the relationship between homocysteine levels and disease duration (Figure 1) revealed a weak but statistically significant positive correlation ($r = 0.249$; $p = 0.043$). This finding suggests that longer disease duration may be associated with gradual increases in homocysteine concentration.

Although the strength of the correlation was modest, its statistical significance indicates a potential cumulative effect of chronic inflammation and prolonged immune activation on homocysteine metabolism and endothelial function. This observation supports the hypothesis that disease-related factors, rather than demographic characteristics alone, may play a more important role in influencing vascular risk markers in juvenile rheumatoid arthritis.

Taken together, these results indicate that while homocysteine levels do not differ significantly across age groups, disease duration appears to be a more relevant determinant. This highlights the importance of long-term monitoring of biochemical markers associated with endothelial dysfunction in pediatric patients with chronic autoimmune diseases.

Figure 1 illustrates the association between serum homocysteine concentration (μmol/L) and disease duration. Each point represents an individual patient, while the solid line indicates the linear regression trend with a shaded area corresponding to the 95% confidence interval.

Correlation analysis demonstrated a weak positive relationship between homocysteine levels and duration of disease ($r = 0.249$), which reached statistical significance ($p = 0.043$). This finding suggests that longer disease duration is associated with a gradual increase in serum homocysteine concentration.

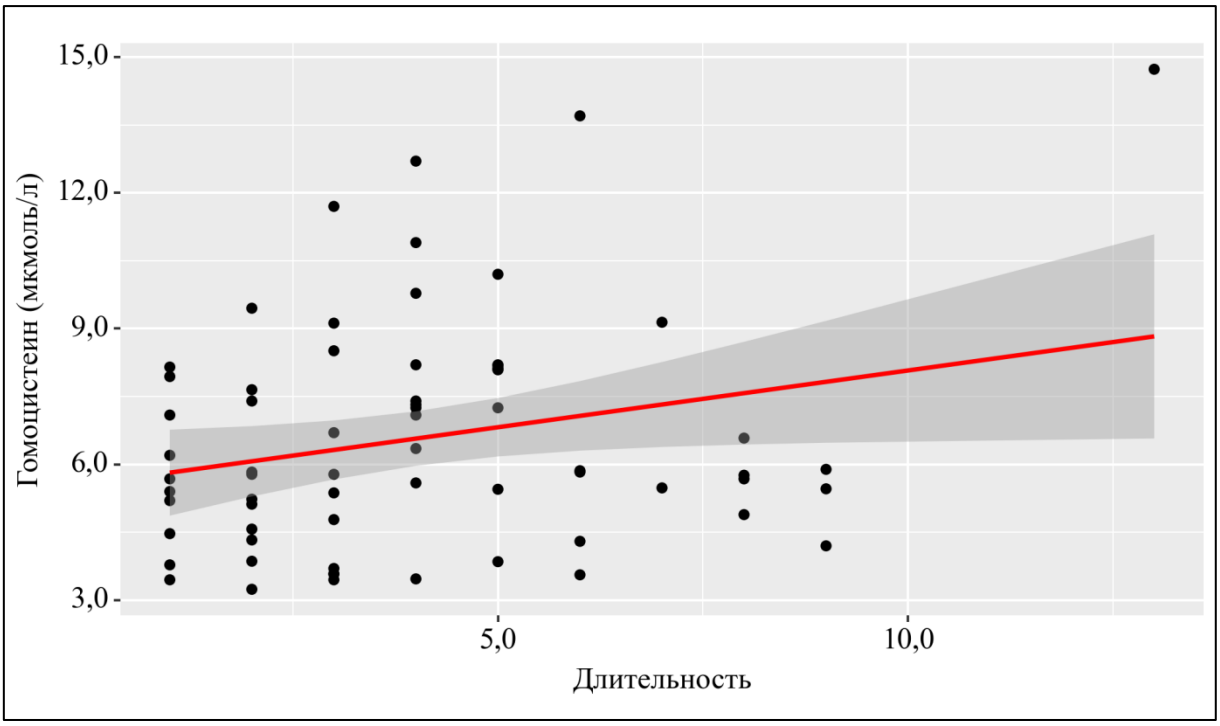

Figure 1. Linear regression illustrating the relationship between serum homocysteine concentration and disease duration in patients with juvenile rheumatoid arthritis.

Although the strength of the correlation was modest, the observed trend supports the hypothesis that prolonged chronic inflammation and sustained immune activation may contribute to alterations in homocysteine metabolism over time. The relatively wide confidence interval reflects interindividual variability and indicates that additional factors may influence homocysteine levels in pediatric patients with juvenile rheumatoid arthritis.

Table 2. Serum Homocysteine Levels According to the Clinical Form of Juvenile Rheumatoid Arthritis

Clinical form	Serum homocysteine (µmol/L)	95% CI	n	F	p
	Mean ± SD				
Oligoarthritis	5.4 ± 2.1	4.6–6.3	25	5.69	0.005
Polyarthritis	6.9 ± 2.4	6.0–7.8	30		
Systemic	8.1 ± 2.7	6.3–9.8	12		

Post hoc analysis: Difference between **oligoarticular and systemic forms** was statistically significant ($p = 0.006$).
Note: One-way analysis of variance (ANOVA) was applied.

Analysis of serum homocysteine levels according to the clinical form of juvenile rheumatoid arthritis revealed a statistically significant difference between groups (F = 5.69; p = 0.005). The lowest mean homocysteine concentration was observed in patients with the oligoarticular form (5.4 ± 2.1 µmol/L), whereas higher values were detected in patients with polyarticular (6.9 ± 2.4 µmol/L) and systemic disease (8.1 ± 2.7 µmol/L).

Post hoc comparison demonstrated a significant increase in serum homocysteine levels in patients with the systemic form compared with those with oligoarthritis (p = 0.006). Although patients with polyarthritis exhibited higher homocysteine levels than those with oligoarthritis, this difference did not reach statistical significance, indicating partial overlap between these groups.

The observed gradient of increasing homocysteine concentration from oligoarticular to systemic disease suggests a potential association between disease severity and endothelial dysfunction. This pattern may reflect the impact of more pronounced systemic inflammation and immune activation in severe forms of juvenile rheumatoid arthritis.

In the analysis of disease activity, a similar trend was identified: serum homocysteine levels increased with higher disease activity ($F = 8.492$; $p < 0.001$). This finding further supports the hypothesis that homocysteine may serve as a biochemical marker associated with inflammatory burden and vascular involvement in pediatric autoimmune rheumatic diseases.

Table 3. Serum Homocysteine Levels According to Disease Activity in Juvenile Rheumatoid Arthritis

Disease activity	Serum homocysteine (μmol/L)	95% CI	n	F	p
	Mean ± SD				
Activity I	5.2 ± 1.6	4.5–5.8	23	8.492	<0.001
Activity II	6.9 ± 2.9	5.8–7.9	29		
Activity III	8.2 ± 1.8	7.2–9.2	15		

Post hoc analysis:

- Activity I vs Activity II: $p = 0.027$
- Activity I vs Activity III: $p < 0.001$

Note: One-way ANOVA was applied.

As presented in Table 3, serum homocysteine concentrations differed significantly according to the degree of disease activity ($F = 8.492$; $p < 0.001$). The lowest mean level was observed among patients with Activity I (5.2 ± 1.6 μmol/L), whereas progressively higher concentrations were detected in patients with Activity II (6.9 ± 2.9 μmol/L) and Activity III (8.2 ± 1.8 μmol/L), indicating a clear activity-dependent gradient.

Post hoc analysis demonstrated that these differences were not confined to comparisons between extreme activity categories. Statistically significant differences were identified both between Activity I and Activity II ($p = 0.027$) and between Activity I and Activity III ($p < 0.001$). These results suggest a stepwise increase in serum homocysteine levels with rising disease activity, reflecting a close association between inflammatory burden and disruptions in homocysteine metabolism.

Correlation analysis further supported this relationship. As illustrated in Figure 2, assessment of the association between disease activity and serum homocysteine concentration revealed a moderate positive correlation ($r = 0.457$), which was highly statistically significant ($p < 0.001$). This finding indicates that increased disease activity is consistently accompanied by elevated homocysteine levels.

Collectively, these findings suggest that serum homocysteine may serve as a sensitive biochemical marker of disease activity in juvenile rheumatoid arthritis. The observed association underscores the potential role of homocysteine as an indicator of systemic inflammation and endothelial dysfunction in pediatric autoimmune conditions. Regression analysis confirmed a positive and statistically significant association between disease activity and serum homocysteine concentration. According to the regression model, an increase in disease activity by one unit was associated with an estimated rise in serum homocysteine level of 1.538 μmol/L. This quantitative relationship further strengthens the evidence for a dose-dependent link between inflammatory activity and homocysteine elevation.

This finding is consistent with the moderate positive correlation observed between disease activity and serum homocysteine levels ($r = 0.457$; $p < 0.001$), suggesting that homocysteine concentrations increase proportionally with the severity of inflammatory processes.

From a pathophysiological perspective, elevated homocysteine levels may reflect enhanced oxidative stress, endothelial injury, and altered methylation pathways associated with chronic inflammation. In juvenile rheumatoid arthritis, sustained immune activation may impair homocysteine clearance or promote its accumulation, thereby linking metabolic imbalance with inflammatory disease progression.

These results highlight the potential clinical utility of serum homocysteine as an adjunct biomarker for monitoring disease activity and treatment response in juvenile rheumatoid arthritis. Future longitudinal studies are warranted to clarify the causal mechanisms underlying this association and to determine whether therapeutic modulation of homocysteine levels could contribute to improved disease control and long-term cardiovascular risk reduction in affected children.

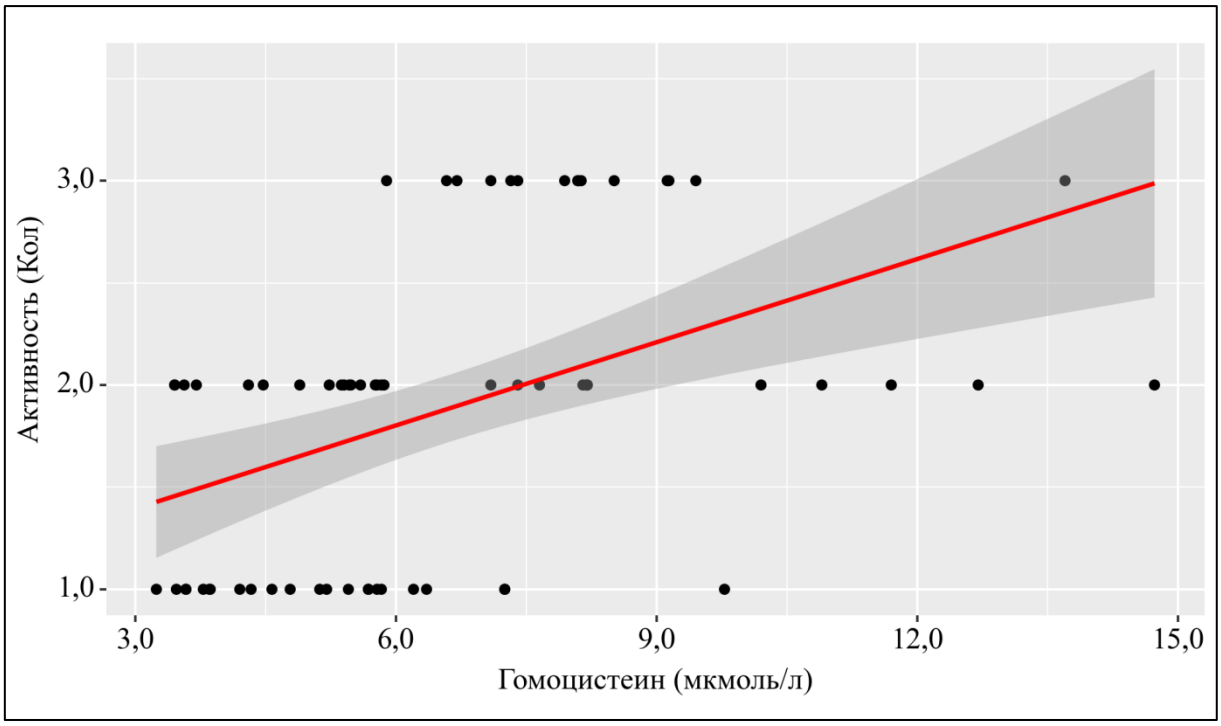

Figure 2. Linear regression illustrating the relationship between serum homocysteine concentration and disease activity in juvenile rheumatoid arthritis.

CONCLUSION

This study demonstrates that serum homocysteine levels increase in parallel with disease activity and clinical severity, including systemic manifestations, in patients with juvenile rheumatoid arthritis. In contrast, chronological age showed no significant association with homocysteine concentration, while disease duration exhibited only a weak relationship. These findings indicate that elevated homocysteine levels are more closely linked to the current inflammatory burden than to cumulative disease exposure.

The consistent association between serum homocysteine concentration and disease activity highlights its potential value as a biochemically informative marker reflecting immune-mediated inflammation and endothelial dysfunction in juvenile rheumatoid arthritis. Improved understanding of the mechanisms underlying homocysteine dysregulation may therefore enhance insight into disease pathophysiology, support more accurate risk stratification, and inform the development of targeted diagnostic and therapeutic strategies aimed at reducing systemic and vascular complications.

Future research should prioritize longitudinal, multicenter investigations integrating inflammatory, metabolic, and vascular biomarkers to clarify the causal role of homocysteine. Such studies may further determine its utility as a predictive marker of disease activity and progression, as well as explore its potential as a therapeutic target in the comprehensive management of juvenile rheumatoid arthritis.

REFERENCES

1. **Balabanova RM, Kuzmina NN, Erdez SF.** Rheumatic diseases in children and adolescents in the Russian Federation (2009–2010). *Scientific and Practical Rheumatology*. 2013;51(4):446–450.
2. **Khamidova NA, Madjidova YN.** Role of homocysteine in the development of juvenile rheumatoid arthritis in children. *Bulletin of the Tashkent Pediatric Medical Institute*. 2025;6(3):27–33.
3. **Khamidova NA, Madjidova YN.** *Clinical and neurophysiological features of neurological complications in children with juvenile rheumatoid arthritis: methodological recommendations*. Tashkent; 2025. p. 22.
4. **Watutin NT, Smirnova AS, El-Khatib A.** Principles of using imaging methods in the diagnosis and treatment of juvenile idiopathic arthritis. *Modern Rheumatology Journal*. 2015;(4):44–47.
5. **Gaidar EV, Kostik MM, Dubko MF, et al.** Efficacy of adalimumab in chronic anterior uveitis associated with juvenile idiopathic arthritis resistant to methotrexate therapy: a retrospective case series. *Pediatric Pharmacology*. 2016;(4):340–344.
6. **Grom AA.** Systemic juvenile idiopathic arthritis: mechanisms of development and targets for biologic therapy. *Issues of Modern Pediatrics*. 2012;(3).
7. **Kuzmina NN, Vorontsov IM, Nikishina IP, Salugina SO.** Evolution of terminology and classification concepts in juvenile chronic arthritis. *Scientific and Practical Rheumatology*. 2001;(1).
8. **Makunina YU, Yuarov ES, Melnichenko OA.** Structure of rheumatic diseases in children based on data from a pediatric hospital clinic over the last 30 years. *Bulletin of Medical and Clinical Research*. 2014;(4).

EDITORIAL REVIEW

The article recently accepted for publication makes an important contribution to the growing body of research addressing the systemic and metabolic dimensions of juvenile rheumatoid arthritis (JRA). By focusing on serum homocysteine levels and their relationship with disease activity, clinical form, and severity, the authors advance current understanding of JRA beyond traditional joint-centered and inflammatory paradigms.

Juvenile rheumatoid arthritis, increasingly referred to as juvenile idiopathic arthritis, is a chronic autoimmune disease of childhood with heterogeneous clinical manifestations and long-term consequences. While inflammation-driven joint damage remains the defining feature, accumulating evidence indicates that systemic and vascular processes also play a significant role in disease progression and long-term morbidity. In this context, the accepted study is both timely and relevant, as it examines homocysteine—a metabolically active amino acid with established links to endothelial dysfunction and inflammation—as a potential biomarker of disease activity in pediatric patients.

A major strength of the accepted article lies in its systematic analysis of homocysteine levels across multiple disease dimensions. The authors demonstrate that serum homocysteine concentration increases significantly with higher disease activity and is most pronounced in patients with systemic forms of JRA. In contrast, patient age showed no significant association, and disease duration was only weakly correlated with homocysteine levels. These findings suggest that homocysteine reflects current inflammatory burden rather than cumulative disease exposure, reinforcing its relevance as a dynamic disease-related marker.

The distinction drawn between demographic factors and disease-specific characteristics is particularly noteworthy. By showing that homocysteine elevation is not driven by age-related physiological changes, the study strengthens the argument that metabolic dysregulation is closely linked to immune activity in JRA. This positions homocysteine as a potentially informative adjunct to conventional inflammatory markers, such as erythrocyte sedimentation rate and C-reactive protein, which do not fully capture vascular or metabolic involvement.

From a mechanistic perspective, the findings reported in the accepted article are biologically plausible. Chronic systemic inflammation may interfere with folate-dependent metabolic pathways, reduce homocysteine clearance, and promote oxidative stress. Elevated homocysteine, in turn, may exacerbate endothelial dysfunction and amplify inflammatory signaling, creating a self-reinforcing cycle. Importantly, such processes may remain clinically silent during childhood while contributing to long-term cardiovascular and systemic risk. The clinical implications of the accepted work are substantial. Incorporating homocysteine assessment into the evaluation of children with active or severe JRA may improve risk stratification and provide a more comprehensive picture of disease burden. Moreover, because homocysteine levels are potentially modifiable, the findings open perspectives for future interventional studies exploring nutritional or metabolic adjuncts to immunosuppressive therapy.

In conclusion, the article accepted for publication offers meaningful insights into the metabolic correlates of disease activity in juvenile rheumatoid arthritis. By integrating immunological, clinical, and biochemical perspectives, it supports a more holistic approach to disease assessment and lays the groundwork for future longitudinal and mechanistic studies aimed at improving long-term outcomes in pediatric rheumatology.

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