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PHYSICAL ACTIVITY BEFORE DISEASE ONSET AND ITS IMPACT ON THE RISK AND CLINICAL SEVERITY OF RHEUMATOID ARTHRITIS – A NARRATIVE LITERATURE REVIEW

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ABSTRACT

Background: Rheumatoid arthritis (RA) is a chronic, systemic connective tissue disease of immunological origin and unknown etiology, characterized by nonspecific symmetric joint inflammation, extra-articular manifestations, and systemic symptoms, ultimately leading to progressive joint destruction, disability, and premature death. The disease can be classified as seropositive or seronegative depending on the presence or absence of autoantibodies in the serum, such as IgM rheumatoid factor and/or anti-citrullinated peptide antibodies (ACPA) [1]. Increasing evidence suggests that environmental factors, including lifestyle and physical activity (PA), may influence both the risk of developing RA and the clinical course of the disease [2–4].
Aim: Assessment of the impact of physical activity undertaken prior to disease onset on the risk of developing rheumatoid arthritis and on the severity of symptoms at diagnosis.
Objective: To review the literature on whether PA prior to RA onset affects disease risk and severity at diagnosis.
Material and methods: A literature review was conducted using the PubMed, Scopus, and Web of Science databases, covering the years 2000–2025, with the following keywords:"Rheumatoid Arthritis, "“Physical Activity, "“Risk, "“Disease Severity." The review included cohort studies, cross-sectional studies, meta-analyses, and systematic reviews.
Results: A higher level of recreational physical activity during leisure time prior to disease onset is associated with a 20–35% reduction in the risk of developing RA [5–7], as well as with a milder disease course at diagnosis [8]. In contrast, high levels of occupational physical activity increase the risk of RA [9]. The protective mechanisms against disease development include modulation of the immune response, reduction of systemic inflammation, and improvement of metabolic profile [10–12].
Conclusions: Regular recreational physical activity may represent an important component of primary prevention of rheumatoid arthritis. Further prospective studies with objective measurements of physical activity are warranted.

KEYWORDS
Rheumatoid Arthritis, Physical Activity, Risk, Disease Severity, Prevention

CITATION

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1. Introduction

Rheumatoid arthritis (RA) is a chronic, systemic connective tissue disease of immunological origin and unknown etiology, characterized by nonspecific symmetric joint inflammation, extra-articular manifestations, and systemic symptoms, leading to progressive joint destruction, disability, and premature death. The disease can be classified as seropositive or seronegative, depending on the presence or absence of autoantibodies in the serum, such as IgM rheumatoid factor and/or anti-citrullinated peptide antibodies (ACPA) [1]. In Poland, the number of patients is estimated at approximately 400, 000–500, 000 [13]. Women are affected three times more frequently than men, and the peak incidence occurs in the fourth and fifth decades of life. In most patients, the disease is characterized by alternating periods of exacerbations and relative remission, with ongoing progressive joint destruction. Spontaneous remissions occur more frequently in men and in older patients. Typically, the disease develops insidiously over several weeks, although in some cases symptoms may appear suddenly within a few days.

The pathogenesis of rheumatoid arthritis (RA) is complex and involves the interaction of genetic, immunological, environmental, and lifestyle factors, including the level of physical activity. Genetic factors have a significant impact on the development of RA, as evidenced by the higher concordance rate among monozygotic twins. In cases where RA is diagnosed in one twin, the risk of developing the disease in the other is estimated at 30–50%. Immunological factors include disturbances of the adaptive immune response, which play a role in initiating and maintaining disease progression through T lymphocytes that recognize self-antigens, including citrullinated autoantigens, and support the production of autoantibodies with the same specificity, such as anti-CCP antibodies. The presence of the shared epitope increases the frequency of self-antigen presentation, thereby accelerating the transformation of mild arthritis into an aggressive inflammatory process. Moreover, B lymphocytes actively contribute to the development and perpetuation of inflammation. The consequence of polyclonal B-cell activation and an excessive humoral immune response is the production of various autoantibodies, including rheumatoid factor (RF), which is present in approximately 80% of RA patients and is associated with a more severe clinical course of the disease [14].

Environmental factors include previous infections (particularly viral), which stimulate the immune system in response to pathogens. In genetically predisposed individuals, this may trigger an autoimmune reaction directed against joint structures. Certain bacteria and viruses share antigenic similarities with HLA-DRB1 and HLA-DRB4 alleles, frequently observed in RA patients, which may promote the initiation of autoimmune processes. Furthermore, cigarette smoking, inadequate diet, and even obesity increase the risk of developing RA and may also contribute to a more severe disease course [15].

It has been demonstrated that lifestyle, particularly the level of physical activity, influences the pathogenesis of rheumatoid arthritis (RA) [16]. Physical activity is defined as any bodily movement produced by skeletal muscles that results in energy expenditure. This includes movement performed during leisure time as well as activities related to work, transportation, and household duties. The World Health Organization (WHO) recommends at least 150–300 minutes of moderate-intensity or 75–150 minutes of vigorous-intensity aerobic physical activity per week, supplemented by muscle-strengthening exercises. Insufficient physical activity is considered one of the major risk factors for non-communicable diseases and contributes to multiple health problems. Therefore, WHO emphasizes the promotion of physical activity as an essential component of a healthy lifestyle [17].

An increasing body of evidence indicates that physical activity (PA) influences immune responses by modulating cytokine production and immune cell populations. Both aerobic and resistance training lead to significant reductions in pro-inflammatory cytokines such as TNF-α, IL-6, and CRP, while simultaneously

increasing levels of the anti-inflammatory cytokine IL-10 [10, 18]. Moreover, regular physical activity enhances the number and activity of regulatory T lymphocytes (Treg), which exert protective effects in the development of autoimmune diseases, while reducing the proportion of Th17 lymphocytes. This phenomenon modifies the immune response and shifts the balance toward tolerance [12, 19].

2. Objective

The aim of this study is to analyze the impact of physical activity undertaken prior to disease onset on:

- The risk of developing rheumatoid arthritis.
- The severity of clinical symptoms at the time of diagnosis.

3. Materials and Methods

The literature review was conducted in August 2025 using the PubMed, Scopus, and Web of Science databases. The search covered the period from January 1, 2000, to August 1, 2025. A combination of MeSH terms and keywords was applied as follows:

("Rheumatoid Arthritis") and ("Physical Activity" OR Exercise) and (Risk OR Incidence OR"Disease Severity").

4. Results

4.1. Recreational physical activity and the risk of RA

Liu et al. [5], in the Nurses' Health Study II, which included 113, 366 women and spanned from 1989 to 2015 (mean follow-up 26 years), demonstrated a significant association between the level of recreational physical activity and the risk of developing rheumatoid arthritis (RA). Women who engaged in ≥7 hours per week of recreational activity had a 33% lower risk of developing RA compared to those with <1 hour per week (HR 0.67; 95% CI 0.47–0.98; trend p=0.02). Importantly, this effect was observed in analyses applying a time-lag of 2–8 years, thereby minimizing the influence of changes in physical activity related to early disease symptoms, and after adjustment for potential confounders including smoking, diet quality, body mass index (BMI) at age 18, and updated BMI during follow-up. Mediation analysis revealed that updated BMI accounted for 14% (p=0.002) of the effect on overall RA and 20% (p=0.001) of the effect on seropositive RA, indicating that part of the protective role of physical activity was mediated through changes in body weight [5].

In contrast, Di Giuseppe et al. [6], in a large prospective cohort study — the Swedish Mammography Cohort — which included 30, 112 women born between 1914 and 1948 and followed between 2003 and 2010 (226, 477 person-years), identified 201 incident cases of RA. The analysis demonstrated that women in the highest category of leisure-time physical activity—defined as at least 20 minutes of walking or cycling daily (median 40–60 minutes/day) combined with ≥1 hour of weekly exercise (median 2–3 hours/week)—had a significantly reduced risk of RA: 35% lower compared with women in the lowest activity category (RR 0.65; 95% CI 0.43–0.96). Notably, household activity was associated with approximately 32% risk reduction, whereas occupational activity did not confer a significant protective effect (approximately –15% risk). In contrast, complete absence of leisure-time activity was associated with a 27% increase in RA risk. Furthermore, the authors calculated the population prevented fraction, estimating that high physical activity in this category potentially prevented 22% of RA cases in the studied population [6].

In a meta-analysis, Sun et al. [7] evaluated four available observational studies (including a total of 255, 365 participants and 4, 213 incident RA cases) to assess the association between physical activity (PA) levels and the risk of developing the disease. Comparison of the highest versus lowest categories of PA demonstrated that individuals with the highest activity levels had a 21% lower risk of RA (RR 0.79; 95% CI 0.72–0.87). When overall activity was compared with complete inactivity, a 15% risk reduction was observed (RR 0.85; 95% CI 0.79–0.92). The authors also applied Mendelian Randomization (MR) analysis, using genetically determined instruments for different forms of physical activity, including both self-reported and accelerometer-measured variables. MR results did not statistically confirm a causal protective effect of PA on RA risk—odds ratio was 0.97 (95% CI 0.88–1.08) for each unit increase in accelerometer-assessed activity. Other activity variables, such as moderate-to-vigorous activity or ≥3 days/week of vigorous activity, also yielded neutral associations (OR 1.08 and OR 2.63, both non-significant). Due to the high heterogeneity of the included studies (I^2), differences in PA definitions, measurement methods, participant demographics, and study methodologies were identified. Therefore, further studies are needed, particularly with objective measurements of activity (e.g., accelerometers) and causal inference analyses in genetically predisposed populations [7].

4.2. Physical activity prior to disease onset and disease severity.

Sandberg et al. [8], in an analysis based on data from the Epidemiological Investigation of Rheumatoid Arthritis (EIRA) cohort including 617 patients with RA, compared individuals who had been physically active during the four years preceding diagnosis with those who were less active. Logistic regression analysis demonstrated that physically active patients had a 42% lower likelihood of presenting with high disease activity (DAS28 above the median) at diagnosis (OR = 0.58; 95% CI 0.42–0.81). The protective effect was also observed for physician's global assessment (OR = 0.67; 95% CI 0.47–0.95) and for patient-reported pain on the VAS scale (OR = 0.62; 95% CI 0.45–0.86), whereas no significant association was detected for HAQ scores. Moreover, a clear dose–response relationship was identified: higher pre-disease physical activity levels correlated with both reduced joint swelling and lower inflammatory markers (ESR, CRP), thereby influencing both objective and subjective components of DAS28. Importantly, these associations remained robust after adjustment for sex, age, year of diagnosis, smoking, BMI, alcohol consumption, socioeconomic status, and occupational physical load [8].

4.3. Occupational physical activity

Kronzer *et al.* [9], in a case-control study conducted within the Mayo Clinic Biobank cohort including 212 incident RA cases and 636 age-, sex-, and recruitment year–matched controls, assessed both occupational physical activity (work-related physical load) and dietary habits [9]. Logistic regression analysis demonstrated that high levels of occupational physical activity were associated with a threefold increased risk of developing RA compared to low activity or sedentary occupations (aOR 3.00; 95% CI 1.58–5.69).

Among the dietary habits analyzed, consumption of at least three daily servings of high-fat foods (e.g., fatty meats, fried foods, fast foods) was associated with an elevated RA risk (aOR 1.22; 95% CI 0.74–2.00). Conversely, intake of more than five servings of fruits and vegetables per day showed a protective trend, being associated with a lower RA incidence (aOR 0.75; 95% CI 0.51–1.11). Sensitivity analyses accounting for a minimum five-year lag period between exposure assessment and diagnosis yielded similar findings — a high-fat diet was linked to increased risk (aOR 1.80; 95% CI 0.69–4.71), whereas higher fruit/vegetable consumption maintained a protective trend (aOR 0.54; 95% CI 0.27–1.08).

The authors concluded that both intensive occupational physical activity and unfavorable dietary habits may represent risk-enhancing factors for RA, underscoring the need for further confirmatory studies [9].

4.4. Type of Physical Activity

Studies indicate that aerobic exercise more effectively reduces inflammatory markers than resistance training [20, 21]. Moreover, high-intensity interval training (HIIT) provides benefits not only for healthy older adults but also for patients with RA. In a pilot study by Bartlett *et al.* [22], 12 individuals with quiescent, stable RA (mean age: 64 ± 7 years) participated in a 10-week HIIT walking program, consisting of three weekly sessions. Each session included ten high-intensity intervals (80–90% VO_2reserve) interspersed with low-intensity periods (50–60% VO_2reserve). At the end of the intervention, a significant reduction in disease activity, measured by the DAS28 score, was observed, along with an improvement in cardiorespiratory fitness. Regarding immunological parameters, a decrease in the expression of TLR2, TLR4, and HLA-DR on the surface of monocytes was reported ($p < 0.05$), together with a significant increase in monocyte phagocytic capacity against *Escherichia coli* ($p = 0.02$) Inflammatory markers — including IL-1β, IL-6, CXCL-8, IL-10, CRP, and TNF-α — did not show significant changes. These findings suggest that improvements in immune function of peripheral mononuclear cells may occur independently of cytokine modulation. The intervention was well tolerated, with no serious adverse events, supporting the feasibility of HIIT as a safe adjunctive strategy for improving function and disease outcomes in older RA patients.[22]

4.5. Prodromal Phase

In seropositive individuals, in whom the presence of rheumatoid factor (RF) and/or anti-citrullinated peptide antibodies (anti-CCP/ACPA) has been detected despite the absence of clinical symptoms, a higher level of physical activity (PA) was correlated with lower levels of inflammatory markers such as CRP, which translated into a slower progression of symptom onset [23]. This group may be categorized as having *systemic autoimmunity associated with RA*, as reflected in the EULAR recommendations concerning the phases of RA development [23]. According to these guidelines, individuals considered"at-risk, " i.e., those in the preclinical stage, can be characterized by the presence of autoimmune biomarkers such as RF or ACPA, without imaging abnormalities or joint pain. The proposed EULAR terminology emphasizes the need to establish precise

definitions of intermediate states. Such classification would facilitate comparability across studies and enable the design of future preventive interventions. Preventive strategies — such as the promotion of physical activity in this specific phase — may substantially contribute to delaying or preventing the onset of symptomatic RA. This approach aligns with public health priorities and the concept of a"therapeutic window" in autoimmune diseases [23].

4.6. Immunological Biomarkers

Numerous studies have confirmed that exercise increases the levels of anti-inflammatory cytokines such as IL-10 and decreases pro-inflammatory cytokines including TNF-α and IL-6 at rest [10, 11, 18, 24]. According to Petersen and Pedersen [10], this effect is partly explained by the so-called"*skeletal muscle anti-inflammatory hypothesis."* During contraction, skeletal muscles release myokines, including IL-6, which exert both metabolic and immunomodulatory effects. Exercise-induced IL-6, in turn, stimulates the production of IL-10 and IL-1 receptor antagonist (IL-1ra), thereby shifting the balance toward an anti-inflammatory phenotype [10, 18].

Gleeson *et al.* [11] emphasize that regular physical activity modulates the number and function of monocytes and lymphocytes by reducing the expression of Toll-like receptors (TLRs), which are responsible for initiating the inflammatory cascade. This may explain why physically active individuals demonstrate lower levels of pro-inflammatory cytokines at rest, thereby protecting against chronic low-grade inflammation typical of autoimmune diseases, including RA.

Petersen *et al.* [18] demonstrated that the acute rise in IL-6 following exercise is not associated with muscle damage but represents a physiological response dependent on exercise intensity and duration. In this context, IL-6 functions as an anti-inflammatory mediator, in contrast to macrophage-derived IL-6 observed in chronic inflammation.

Furthermore, Lancaster and Febbraio [24] reported that regular exercise reduces insulin resistance and adipose tissue mass. Adipocytes represent a major source of pro-inflammatory cytokines; thus, a reduction in visceral fat mass leads to diminished chronic immune system activation, which may be particularly relevant in preventing the progression of early RA stages.

5. Discussion

Based on the available findings, substantial evidence indicates that regular recreational physical activity (PA) reduces the risk of developing rheumatoid arthritis (RA) in genetically or environmentally predisposed individuals by approximately 20–35% [5–7], and additionally attenuates disease severity once RA has developed [8]. It is important to emphasize that this protective effect pertains to recreational rather than occupational PA [9]. The underlying protective mechanisms include modulation of the immune response [10–12], improvement in metabolic function [25], and preservation of musculoskeletal integrity [26]. However, several limitations of existing studies should be acknowledged. These include imprecise assessment of PA, the absence of intervention trials during the prodromal phase of RA, and insufficient adjustment for potential confounders [27]. Furthermore, many analyses rely on self-reported PA questionnaires, which may introduce bias due to subjective misclassification of exercise intensity. The heterogeneity of study populations also complicates the generalizability of findings, particularly across groups differing in genetic background, lifestyle factors, and access to healthcare services. Future research should therefore incorporate objective PA measurements (e.g., accelerometry), extended follow-up periods, and stratified analyses in subgroups with varying risk profiles [28–30]. Importantly, interventional studies in high-risk populations, such as asymptomatic seropositive individuals, are needed to evaluate the potential of PA as a component of primary prevention strategies. Moreover, it is essential to investigate the specific impact of different exercise modalities—including aerobic, resistance, and high-intensity interval training—on immune system components and imaging parameters of joint pathology. Such an approach may contribute to the development of precise, personalized PA recommendations for the prevention and management of RA.

6. Conclusions

Rheumatoid arthritis (RA) is a chronic systemic connective tissue disease that leads to progressive joint destruction, disability, and premature mortality. The development and clinical course of RA are influenced by genetic, immunological, and environmental factors, including lifestyle and physical activity (PA). In this review, both cohort and cross-sectional studies consistently demonstrate a beneficial effect of pre-disease physical activity, showing an association with milder disease severity at diagnosis and reduced risk of RA

onset. These findings support the promotion of PA as an important element in RA prevention strategies. Evidence from the literature suggests that the protective mechanisms underlying these associations include modulation of immune responses, reduction of chronic systemic inflammation, improvement of metabolic profile, and preservation of musculoskeletal function. These effects contribute to enhanced joint stability and lower symptom severity in the clinical phase of the disease. The benefits appear to be particularly pronounced in the case of regular recreational PA of moderate to vigorous intensity, maintained consistently over many years prior to the onset of symptoms. Nevertheless, significant research gaps remain. There is a lack of prospective studies with objective measurements of PA. Most available analyses rely on self-reported questionnaires, which are prone to bias due to subjective misclassification of activity levels. Future studies should therefore focus on larger and more diverse populations, encompassing individuals with different genetic and environmental risk profiles, and utilize standardized methods of PA assessment (e.g., accelerometry). Such an approach would allow for the formulation of precise, evidence-based preventive recommendations for RA.

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Authors' Contribution

- Conceptualization: Katarzyna Maria Turek, Katarzyna Adrianna Tryniecka
- Methodology: Weronika Worosz, Diana Wisz, Alicja Zań, Filip Maciej Huzarski
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- Supervision: Katarzyna Maria Turek, Katarzyna Adrianna Tryniecka

All authors have read and approved the final manuscript.

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