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The impact of obesity on the risk of developing, activity and treatment outcomes of autoimmune rheumatic diseases

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Abstract:

Background:

Obesity is becoming a new epidemic due to its high prevalence and rapid increase. It is associated with numerous serious complications, including insulin resistance, dyslipidemia, hypertension. Obesity is associated with chronic low-grade inflammation, oxidative stress, and increased expression of pro-inflammatory cytokines and adipokines. Autoimmune rheumatic diseases are driven by similar mediators and immunological dysregulations, which are disturbed in obesity. It suggests potential associations between obesity and these diseases.

Aim:

The aim of this review is to examine evidence on how obesity affects the risk of developing common rheumatic diseases, the activity/severity of these diseases, and the outcomes of treatment, with a focus on recent literature and adult patients.

Material and methods:

A literature search was conducted in the PubMed database up to February 2026 using the following terms: “obesity” and “BMI”, in combination with disease-specific keywords. Only articles published in English or with an available English abstract were included. Only studies on humans were included. Meta-analyses and cohort studies were prioritised; other types of studies were included only when higher-level evidence was not available. Additionally, two relevant conference abstracts [23, 27] that were not indexed as full records in PubMed were identified via manual screening of reference lists and included in the review. The literature selection process and the flow of information at each stage of the review were presented in accordance with the PRISMA 2020 guidelines (Figure 1).

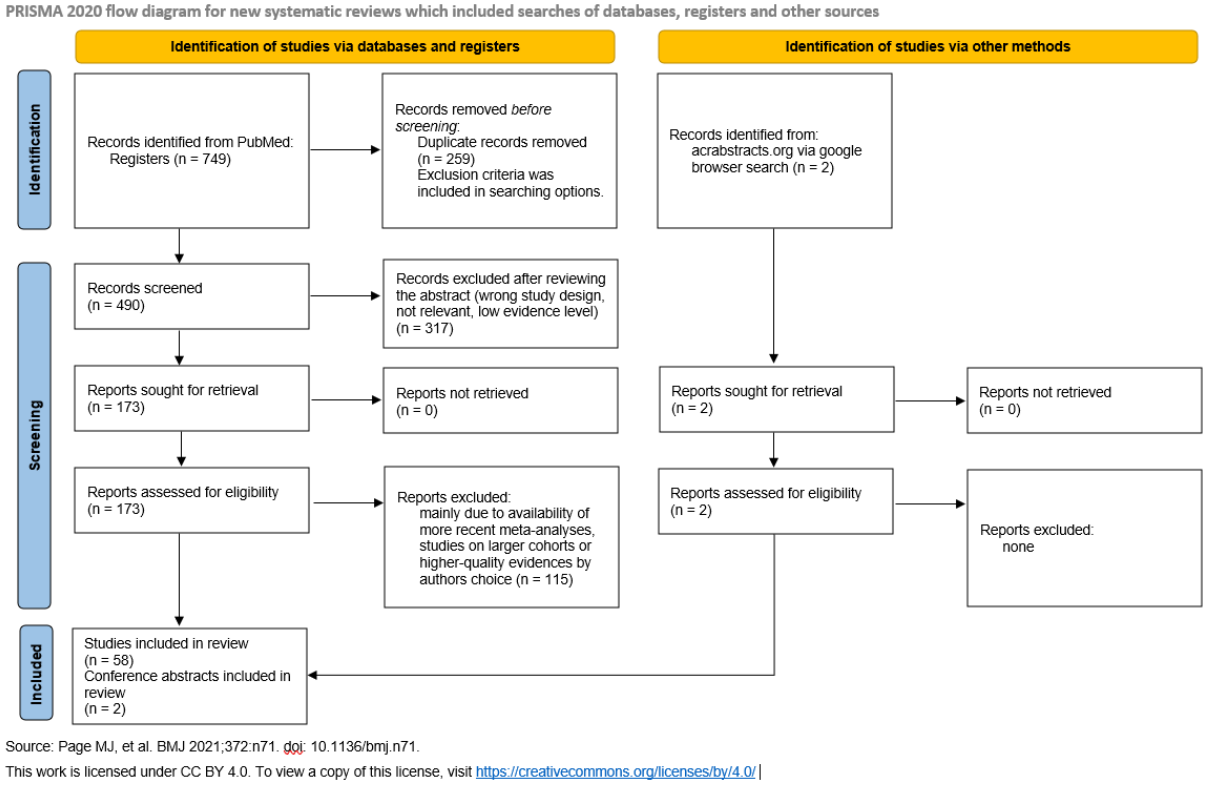

Figure 1. PRISMA flow chart

Conclusions:

Obesity increases the risk of development of rheumatoid arthritis and psoriasis and there is some evidence of common genetic backgrounds. Childhood obesity increases the risk of developing systemic lupus erythematosus, whereas the association with adult obesity is less consistent. There are positive correlations between body mass index and disease activity in rheumatoid arthritis, psoriasis and ankylosing spondylitis, as well as with certain clinical manifestations, including lupus nephritis and psoriatic arthritis. Obesity significantly reduces the likelihood of good treatment outcomes with anti-TNF agents, which suggests the need for treatment personalisation in patients with higher body mass.

Keywords:

obesity, rheumatoid arthritis, systemic lupus erythematosus, psoriasis, axial spondyloarthritis

1. Introduction: epidemic of obesity

Obesity is a medical condition characterized by excessive accumulation of body fat, which causes a negative impact on health and quality of life. It is defined as a Body Mass Index (BMI) of 30 or higher and is preceded by overweight, which is defined as BMI ≥ 25 kg/m². Obesity is becoming a new epidemic because of its high prevalence and rapid increase. According to Eurostat, in 2022, 50.6% of people aged 16 or older were overweight or obese. These conditions are more prevalent in men (58.7%) than in women (43.0%). The frequency is growing with age: 20.3% of people aged 16–24, 63.6% aged 65–74, but then it tends to decline to 57.7% in people older than 75 [1]. The problem is spreading with an increasing prevalence, and it is projected that this trend will continue in the foreseeable future. In 1990 in Poland, 40.2% of females aged 25 or more and 55.3% of males aged 25 or more were overweight and obese. In 2021, it was 52.5% of females (a relative increase of 31%) and 66.9% of males (+21%), and it is projected to be 60.1% of females (+14%) and 72.8% of males (+9%) in 2050 [2]. These data are presented in Figure 2.

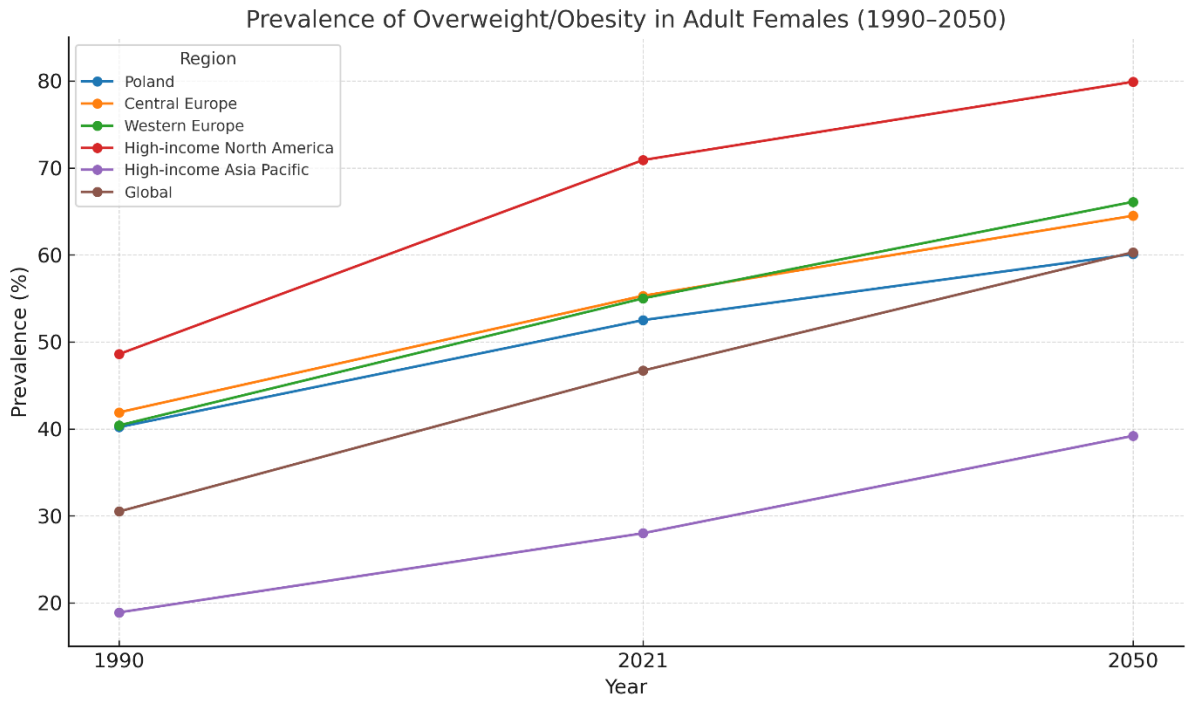

Figure 2.

Prevalence of obesity and overweight in adult females from 1990 to 2021, with projections to 2050 [2].

Obesity shortens life. A meta-analysis of over 10.6 million people who were never-smokers and had no chronic diseases from 239 cohort studies found that the lowest risk of death was observed at a BMI of 20–25 kg/m². Every 5-unit increase in BMI above 25 was associated with an approximately 31% higher risk of all-cause mortality. People with a BMI ≥40 kg/m² had more than double the risk of death and lived, on average, eight to ten years less than those with normal weight [3]. This is mainly due to adipose tissue's endocrine activity, which secretes pro-inflammatory adipokines. These molecules cause many health problems, such as insulin resistance, dyslipidemia, hypertension, chronic inflammation (so-called low-grade inflammatory state), and oxidative stress.

Autoimmune rheumatic diseases cause chronic joint and systemic inflammation. The rising prevalence of obesity may thus have important effects on the incidence, severity, and management of these diseases. This review will systematically examine evidence on how obesity affects the risk of developing common rheumatic diseases, the activity/severity of these diseases, and the outcomes of treatment, with a focus on recent literature and adult patients.

2. Rheumatoid Arthritis

Rheumatoid arthritis (RA) is an autoimmune disease characterized by synovial membrane inflammation, joint destruction, swelling, pain, morning stiffness, and extra-articular manifestations, including vasculitis and interstitial lung disease [4]. It also increases the risk of cardiovascular events [5]. It is one of the most common rheumatic diseases. A report by the Global Burden of Diseases, Injuries, and Risk Factors Study

(GBD) 2021 Rheumatoid Arthritis Collaborators estimated that in 2020, 17.6 million people worldwide (95% CI 15.8–20.3) had RA, with an age-standardized global prevalence rate of 208.8 (186.8–241.1) per 100,000 people, representing a 14.1% (12.7–15.4) increase since 1990, with an alarming forecast of 30 million RA patients worldwide by 2050. Prevalence was higher in females, with a female-to-male ratio of 2.45 (2.40–2.47), and peaked in the 75–79 years age group, with 828 cases (730–934) per 100,000 people. In high-income countries, there was a higher RA prevalence but a lower death ratio [6]. Table 1. presents studies on how obesity impacts this disease and its treatment.

Table 1. Clinical impact of obesity on rheumatoid arthritis

Study	**Key findings**
meta-analysis by T. Ohno et al., 2020; 473641 people and 4777 cases [7]	in middle-aged women, each 5 kg/m² increase in BMI was associated with RR = 1.15 (95% CI 1.08–1.21, I^2 = 17%), not observed in men; at age 18 years, each 5 kg/m² increase in BMI was associated with RR = 1.17 (95% CI 1.01–1.36, I^2 = 26%); each 10 cm increase in waist circumference was associated with RR = 1.13 (95% CI 1.02–1.25, I^2 = 44%).
meta-analysis by D. Flores-Alvarado et al., 2023; 4024 patients [8]	CRP was increased in female patients that have a higher BMI, not significant association in men
prospective cohort study by S. Ajeganova et al., 2025; 1813 patients [9]	lower BMI was associated with a markedly increased risk of radiographic joint damage progression compared with obese patients: underweight vs obese, OR = 4.85 (95% CI 1.76–9.05); normal weight vs obese, OR = 3.99 (95% CI 1.76–9.05); overweight vs obese, OR = 1.65 (95% CI 0.68–4.02, not significant); these associations were independent of DAS28-CRP, serostatus, glucocorticoid use, and DMARDs treatment
meta-analysis by C. Vidal et al., 2015; 3787 patients [10]	DAS28 was higher in obese (BMI > 30 kg/m^2) than non-obese (BMI ≤ 30 kg/m^2) patients MD = 0.14 (95% CI 0.01–0.27, p = 0.04, I^2 = 0%); HAQ score was also higher among obese patients, MD = 0.10, 95% CI 0.01–0.19, I^2 = 0%); radiographic joint damage was negatively associated with obesity, standardized MD = −0.15, 95% CI −0.29 to −0.02, I^2 = 38%)
meta-analysis by J. Shan et al., 2019;	obese patients (BMI >30 kg/m²) treated with anti-TNF agents (infliximab, adalimumab, etanercept and certolizumab were used) had significantly lower

10 studies on RA patients [11]	odds of good response (OR = 0.34, 95% CI 0.18–0.64) and remission (OR = 0.36, 95% CI 0.21–0.59) compared with non-obese patients; no significant differences between obese and non-obese were observed in RA patients treated with abatacept (good response OR = 0.75, 95% CI 0.42–1.36; remission OR = 0.84, 95% CI 0.65–1.09) or tocilizumab (good response OR = 1.08, 95% CI 0.44–2.63; remission OR = 0.91, 95% CI 0.50–1.66)
prospective cohort study by L. Tidblad et al., 2025; 1285 patients [12]	in early RA after 6 months of methotrexate treatment 64% (n=98/153) of obese, 52% (n=171/326) of overweight and 48% (n=210/433) of normal-weight patients failed to reach DAS28 remission with an RR = 1.33 (95% CI 1.14–1.55) for patients with obesity after adjustment for age and sex RR=1.27 (95% CI 1.08–1.50) and adjustment for seropositivity, educational level, smoking, alcohol use, physical activity, calendar period, glucocorticoid treatment and comorbidities
retrospective cohort study by S. Liu et al., 2025; 2564 patients [14]	after 102 months higher WWI was associated with increased all-cause mortality HR = 1.28 (95% CI 1.07–1.52) and cardiovascular disease mortality HR = 1.43 (95% CI 1.12–1.81) after full adjustment; BMI was less predictive, with HR = 1.15 (95% CI 0.94–1.42) for all-cause mortality and 1.24 (95% CI 0.97–1.59) for cardiovascular disease mortality

Abbreviations: RR – relative risk, HR – hazard ratio, BMI – body mass index, RA – rheumatoid arthritis, CRP – C-reactive protein, DAS28 – disease activity index in 28 joints, DMARDs – disease-modifying antirheumatic drugs, HAQ – health assessment questionnaire (functional disability in RA); WWI – weight-adjusted waist index, CVD – cardiovascular disease

The presented studies show that BMI and waist circumference are positively correlated with RA risk in women; however, early obesity (at age 18) increases the risk even more than obesity in older age [7]. Notably, this comorbidity appears to have a partially genetic background. A study by Tang et al. demonstrated that genetically predicted BMI, but not waist-to-hip ratio (WHR) or WHR adjusted for BMI (WHRadjBMI), was associated with an increased risk of RA (OR 1.22, 95% CI 1.09–1.37), similar for both sexes. Moreover, they identified significant local genetic correlations at three regions on chromosome 6 suggesting a shared genetic basis between obesity and RA [39]. Obesity is related to increased disease activity (DAS28, CRP) and functional disability, assessed by HAQ score [8,10], but data about radiographic progression are contradictory [9,10]. Obesity decreases the effectiveness of methotrexate and anti-TNF agents but does not appear to affect the efficacy of abatacept or tocilizumab, suggesting the need for therapy personalization [11,12]. It should be noted that a study by G. Vasileiadis showed that the chance of remission with methotrexate combined with prednisolone, certolizumab, abatacept, or tocilizumab is not correlated with baseline levels of adipokines,

including leptin, resistin, and visfatin [13]. The fact that the weight-adjusted waist index (WWI) is more correlated with mortality than BMI [14] suggests that RA is especially worsened by central obesity.

3. Systemic Lupus Erythematosus

Systemic lupus erythematosus (SLE) is an autoimmune disease characterized by chronic general symptoms such as weight loss, fatigue, and low-grade fever; arthritis with pain, morning stiffness, and joint swelling, but usually without deformities; a photosensitive rash, typically butterfly-shaped on the face, and also affecting the neck and limbs; cytopenia; and organ involvement, especially the kidneys (lupus nephritis) with haematuria, proteinuria, and nephrotic syndrome; the respiratory system involvement, including cough, dyspnoea, and haemoptysis; or the central nervous system involvement, manifesting as headaches, photophobia, or even seizures and psychosis. SLE is also associated with increased cardiovascular risk [15]. A meta-analysis of 112 studies by J. Tian et al. estimates that in 2020, there were 3.41 million people with SLE worldwide, with a prevalence rate of 43.7 per 100000 (95% CI 28.6–196.3). Prevalence was higher in females, with a female-to-male ratio of 8.50 (2.18–33.15) [16]. Table 2. presents studies on how obesity impacts this disease and its treatment.

Table 2. Clinical impact of obesity on systemic lupus erythematosus

Study	**Key findings**
retrospective cohort study by P. Thomas et al., 2020, 346627 people, 435 SLE cases [17]	children were observed and measured from 7-13 years; birthweight was not associated with SLE risk; childhood BMI and height were positively correlated and linearly associated with SLE risk; at age 7 for BMI HR = 1.11 (95% CI 1.01–1.23) per z-score and for height HR = 1.13 (95% CI 1.02–1.24) per z-score; estimates were similar in magnitude across all childhood ages for BMI and height; no significant differences by sex in any of associations
meta-analysis of two prospective (NHS I and II) cohort studies by S. Tedeschi et al., 2017, 138130 women,	obesity was associated with increased risk of SLE with HR = 1.46 (CI 95% 0.88–2.40), but not significantly; time-varying BMI and 4-year lagged BMI analyses yielded results consistent with the primary analysis; NHSII: 10-lb (~4.5 kg) weight gain from age 18 to enrolment was associated with a modest but increase in SLE risk HR = 1.09 (95% CI 1.02–1.18); NHS (secondary analysis with aligned calendar years): higher point estimate

268 SLE cases [18]	compared to the primary analysis but not statistically significant HR = 1.67 (95% CI 0.81–3.45)
prospective cohort study by Y. Cozier et al., 2020; 56443 black women, 127 SLE cases [19]	obesity in adulthood at ≥ 4 years prior to SLE diagnosis was not related to SLE risk with HR = 0.90 (95% CI 0.53–1.54); obesity at age 18 was associated with increased risk of SLE with HR = 2.38 (95% CI 1.26–4.51)
meta-analysis by A. Gomez et al., 2020; 1684 SLE patients [20]	almost identical SELENA-SLEDAI scores between overweight, obese and normal weight patients; overweight (0.82 ± 1.30) and obese (1.19 ± 1.54) patients had higher SDI scores compared to normal weight (0.63 ± 1.07); overweight and obesity were associated with worse HRQoL measured as: PCS (standardized coefficient $\beta = -0.10$, $p < 0.001$ and $\beta = -0.17$, $p < 0.001$, respectively), FACIT-Fatigue ($\beta = -0.11$, $P < 0.001$ and $\beta = -0.16$, $p < 0.001$) and EQ-5D ($\beta = -0.08$, $P = 0.001$ and $\beta = -0.12$, $p < 0.001$) scores, independently of demographic and disease-related factors
prospective cohort study by JH Kang et al., 2020; 393 SLE patients [21]	BMI ≥ 25 kg/m^2 (in comparison to BMI < 23 kg/m^2) was significantly associated with development of nephritis during 3-year follow-up with OR 26.63 (95% CI, 11.37–62.40) and cumulative organ damage with OR = 4.1 (95% CI, 2.12–7.89); obesity was also associated with increased risk of malar rash, photosensitivity, oral ulcers, and hematologic disorders development during follow-up
retrospective cohort study by F. Cuervo et al., 2025, 132 LN patients [22]	obesity significantly increased long-term CKD risk with OR = 4.23 (CI 95% 1.32–13.59) with LN patients
prospective cohort study by G. Stojan et al., 2012,	risk of cardiovascular events defined as stroke, myocardial infarction, incident angina, a coronary procedure (CABG or PCI), or claudication was higher in overweight (BMI 25-29.9 kg/m^2) patients with RR = 1.6 (95% CI

conference abstract; 2000 SLE patients [23]	1.0–2.4, p = 0.051), obese (BMI 30-34.9 kg/m^2) patients with RR = 1.8 (1.1–3.0, p = 0.015) and severely obese (BMI > 35 kg/m^2) patients with RR = 1.4 (0.8–2.4, p = 0.20), however after adjustment for various confounding factors (age, sex, race, complement, haematocrit, anti-dsDNA, immunosuppressant use), including related CV risk factors: hypertension, hypercholesterolemia, diabetes and smoking only result in obese patients was close to statistical significance with RR = 1.6 (0.9–2.7, p = 0.078)
cross-sectional study by T. Pedrosa et al., 2021, 108 LN patients [24]	despite lower prescribed HCQ dose per RBW in obese vs. non-obese patients [4.4 (2.9–5.4) vs. 4.9 (4.0–5.5) mg/kg/day, p < 0.001], HCQ blood levels were significantly higher in obese (1562 ± 548.6 vs. 1208 ± 448.9 ng/mL, p = 0.002) even with comparable dosing [4.8 (4.5–5.4) vs. 5.0 (4.5–5.5) mg/kg/day, p = 0.312], obese patients still had higher HCQ levels (1734 ± 457.3 vs. 1189 ± 449.4 ng/mL, p < 0.001).

Abbreviations: SLE – systemic lupus erythematosus, BMI – body mass index, NHS – Nurses' Health Study, LN – lupus nephritis, HCQ – hydroxychloroquine, RBW – real body weight, CKD – chronic kidney disease, SLEDAI – systemic lupus erythematosus disease activity index, SELENA-SLEDAI – Safety of Estrogen in Lupus National Assessment – SLEDAI, SDI – Systemic Lupus International Collaborating Clinics/American College of Rheumatology damage index, HRQoL – health-related quality of life (evaluated using SF-36), SF-36 – 36-item Short Form Health Survey, PCS – SF-36 physical component summary, FACIT – Functional Assessment of Chronic Illness Therapy, EQ-5D – European Quality of Life 5-dimension questionnaire

SLE appears to be more strongly associated with childhood overweight, obesity, and increased height than with obesity immediately preceding disease onset [16-19]. However, ultra-processed food (HR = 1.56, 95% CI 1.04–2.32, p = 0.03) and sugar-sweetened/artificially sweetened beverage intake (HR = 1.45, 95% CI 1.01–2.09), which often lead to obesity, are risk factors for SLE [81]. A meta-analysis by A. Gomez et al. showed that BMI does not correlate with SLEDAI, but it is associated with poorer quality of life, reduced physical function, impaired social functioning, and increased fatigue [20]. However, several studies have reported an inverse correlation between BMI and SLEDAI [26-28]. This inconsistency may reflect the "obesity paradox," in which overweight and mildly obese patients exhibit similar or even lower disease activity compared to normal-weight and underweight individuals. One potential explanation is confounding by treatment intensity, particularly glucocorticoid use, which may suppress disease activity while simultaneously promoting weight gain through increased appetite and adverse metabolic effects. Obesity in SLE patients increases the long-term risk of organ damage, particularly lupus nephritis and chronic kidney disease, as well as cardiovascular, dermatologic, and hematologic complications [20–23]. This may suggest that obesity is one factor that shortens life in SLE patients. There is also a lack of studies investigating the impact of obesity on SLE treatment. A study by T. Pedrosa et al. demonstrated that a lower hydroxychloroquine dose per real body weight (RBW) was

associated with higher blood concentrations. This may reflect altered pharmacokinetics of HCQ, probably due to lupus nephritis and chronic kidney disease (CKD) and is potentially concerning, as it increases the risk of retinopathy without improving therapeutic efficacy [24,29], indicating the necessity of therapy personalization.

4. Psoriasis and Psoriatic Arthritis

Psoriasis is primarily a dermatological disease, and its most common form – psoriasis vulgaris – is characterised by monomorphic, sharply demarcated erythematous plaques with characteristic silvery scales, often accompanied by pain and pruritus. The disease often also affects the nails, manifesting as pitting, onycholysis, and yellow–brown subungual discoloration [31]. Psoriasis can involve the joints, a condition referred to as psoriatic arthritis (PsA). This type is associated with the progression of atherosclerosis, increases cardiovascular risk by approximately 50%, and the risk correlates positively with disease activity. PsA is further associated with an increased risk of diabetes, hypertension, and a 40% higher prevalence of obesity [32]. According to the Global Burden of Disease (GBD) study in 2019, there were 4.62 million incident cases (95% UI 4.46–4.78 million) of psoriasis worldwide, and the age-standardised incidence rate was 57.8 (55.8–59.7) per 100,000 people, representing a decrease of 20.0% (95% UI −20.2 to −19.8) compared with 1990; the incidence was similar between sexes [33]. The prevalence of psoriasis varies geographically and is higher in high-income countries. A meta-analysis by R. Parisi (2020) showed that in Western Europe, it is 1.92% (95% CI 1.07–3.46), in North America 1.50% (0.63–3.60), while in East Asia, it is only 0.14% (0.05–0.40) [34]. According to a study by Z. Kang, the global prevalence of PsA in patients with psoriasis is estimated to be 17.58% (3.33%–43.69%), and it is slightly higher in females than males (19.14% vs 16.01%) [35]. Table 3. reviews studies exploring the impact of obesity on this disease, its treatment, and their shared genetic background.

Table 3. Clinical impact of obesity on psoriasis and psoriatic arthritis

Study	**Key findings**
meta-analysis by J. Wang et al., 2024 [36]	estimated a global comorbidity rate of 25% (95% CI 0.21–0.30), with 18% (95% CI 0.11–0.24) in children and adolescents and 35% (95% CI 0.30–0.39) in adults, 23% (95% CI 0.16–0.32) in men and 38% (95% CI 0.20–0.61) in women; obesity prevalence was higher in moderate psoriasis (36%, 95% CI 0.20–0.64) compared with severe psoriasis (30%, 95% CI 0.20–0.45) and mild psoriasis (27%, 95% CI 0.16–0.46)
meta-analysis by Y. Wu et al., 2024, 806834 genomes,	positive genetic correlation between BMI and psoriasis (r_g = 0.22, p = 2.44×10^{-18}), WHR and psoriasis (r_g = 0.19, p = 1.41×10^{-12}), and WHRadjBMI and psoriasis (r_g = 0.07, p = 0.0181);

9267 psoriasis cases [37]	there are 14 shared loci underlying psoriasis and these obesity-related traits and by mendelian randomization showed that BMI (IVW OR = 1.483, 95% CI 1.333–1.649), WHR (IVW OR = 1.393, 95% CI 1.207–1.608) and WHRadjBMI (IVW OR = 1.18, 95% CI 1.047–1.329) are positively correlated with risk of psoriasis; rather one-way dependency: genetic predisposition to obesity increases the risk of psoriasis, whereas genetic predisposition to psoriasis does not increase the risk of obesity
meta-analysis by C. Antonatos et al., 2025 [38]	genomes study showed significantly overlaps between up-regulated (n = 170, $p = 6.07\times10^{-65}$) and down-regulated (n = 49, $p = 7.1\times10^{-7}$) genes in both obesity and psoriasis, associated with increased T cell response and activated transcription factors; cWGCNA analysis identified 48 consensus modules related to either leukocyte differentiation (especially Th17) or metabolic pathways, with similar correlation signals in both conditions
meta-analysis by D. Aune et al., 2018; 695471 people, 17636 psoriasis cases [41]	each 5 kg/m² increase in BMI was associated with RR = 1.19 (95% CI 1.10–1.28, $I^2 = 83\%$, n = 7), but lower risk at BMI around 20; each 10 cm increase in WC with RR = 1.24 (95% CI 1.17–1.31), each 0.1 unit increase in WHR with RR = 1.37 (95% CI 1.23–1.53) and each 5kg of weight gain with RR = 1.11 (95% CI 1.07–1.16)
meta-analysis by H. Wang et al., 2025; 366776 genomes, 1979 psoriasis patients [42]	genomes study showed that genetic predisposition to increased BMI was associated with higher psoriasis risk, but it was one-way dependence only with OR = 2.28 (95% CI 1.33–3.92; $p = 0.003$); clinical studies showed that each 1 kg/m^2 increase in BMI (not standardised) was associated with an increase of 0.25 (95% CI 0.14, 0.36, $p < 0.001$) units and 0.06-0.10 units ($p < 0.01$) after standardisations in the mean PASI score, 0.34 units in the BSA score ($p = 0.001$, but only +0.20 units and $p = 0.072$ with sex adjustment), and 0.14 (95% CI 0.05–0.23; $p = 0.001$) units and 0.06-0.08 units ($p < 0.01$) in the DLQI score; each 1 kg/m^2 increase in BMI (unadjusted covariates) increase the risk of severe psoriasis (PASI ≥ 10, BSA ≥ 10%, DLQI ≥ 10) by 6%, 6%, and 3%, respectively

meta-analysis by W. Xie et al., 2021, 322967 psoriasis patients [43]	increased PsA risk in patients with psoriasis was associated with obesity with OR =1.75 (95% CI 1.42–2.16) and overweight with OR = 1.50 (95% CI 1.08–2.09); each 1 kg/m^2 increase in BMI was associated with 6% (95% CI 3%–10%) PsA risk increase; PsA risk was associated with history of physical trauma and fractures, but not with alcohol, smoking, psychological trauma, and female hormone exposure (adjusted by sex)
meta-analysis by S. Singh et al., 2018, 11873 psoriasis and PsA patients [44]	in 22 studies (12- and 16-week therapies with anti-TNF agents, mainly adalimumab, etanercept, and infliximab), obese patients had significantly higher odds of therapy failure (OR = 1.57, 95% CI 1.30–1.89); BMI was positively correlated with the risk of treatment failure
meta-analysis by R. Lupoli et al., 2016, 1562 PsA patients [45]	overweight (OR = 0.637, 95% CI 0.500–0.81, $p < 0.001$, I^2 = 60.2%) and obese (OR = 0.369, 95% CI 0.249–0.546, $p < 0.001$ I^2 = 63.1) PsA patients had lower chance of achieving MDA than normal weight patients with various DMARDs treatments
retrospective cohort study by E. Vallejo-Yagüe, 2022, 774 PsA patients [46]	obese patients had lower odds of achieving MDA at ≤12 months with OR = 0.45 (95% CI 0.24–0.82), DAPSA-remission with OR = 0.42 (95% CI 0.21–0.85), cDAPSA-remission with OR = 0.51 (95% CI 0.27–0.96) and DAS28-remission with OR = 0.51 (95% CI 0.32–0.81) compared to normal weight patients with DMARDs after adjustment for sex, age, educational level, smoking and type of treatment

Abbreviations: BMI – body mass index, WC – waist circumference, WHR – waist-to-hip ratio, WHRadjBMI – WHR adjusted to BMI, PASI – psoriasis area and severity index, BSA –body surface area, DLQI – dermatology life quality index, PsA – psoriatic arthritis, DMARDs – diseases-modifying antirheumatic drugs, DAPSA – disease activity for psoriatic arthritis, cDAPSA – clinical DAPSA, DAS28 – 28-joint disease activity score

The association between obesity and psoriasis appears to be complex. The prevalence of obesity among patients with psoriasis is much higher than in the general population, both in females (38% vs. 21%) and males (23% vs. 15%) [2,36]. Higher BMI and WHR are significantly correlated with an increased risk of psoriasis development [41]. Earlier studies and reviews have suggested that the association between these conditions might be bidirectional [48,49]. Attempts have also been made to link genetic predisposition to psoriasis with increased obesity risk. A Genome-Wide Association Study (GWAS) by A. Kisielnicka et al. identified seven SNPs (including variants in FTO and CALCRL) associated with higher BMI, and four SNPs (including ITLN2 and PAG1) associated with lower BMI [50]. However, GWAS demonstrates correlations rather than causality, which underscores the need for more robust approaches such as Mendelian randomisation (MR), currently the

most reliable tool for investigating genetic causation [51]. These studies showed that the genetic correlation between psoriasis and obesity is one-directional: a genetic predisposition to obesity causes an increased risk of psoriasis, but a genetic predisposition to psoriasis does not increase the risk of obesity [37,38,42]. The higher prevalence of obesity among psoriatic patients therefore appears to be explained by secondary mechanisms, including reduced physical activity due to depression [52], disability and joint pain in PsA [53], adverse metabolic effects of treatment with anti-TNF agents [54], and systemic inflammation, which leads to insulin resistance [55,56]. It is also worth mentioning that MR analysis showed that genetic predisposition to childhood obesity is independently associated with PsA risk, irrespective of adult body mass [57], which has been confirmed earlier in cohort studies [58]. Higher BMI is positively correlated with a higher risk of PsA and PsA development in patients with psoriasis without joint involvement at disease onset [43,59], nail psoriasis within PsA patients [60], and with more severe dermatological symptoms measured by the Psoriasis Area and Severity Index (PASI), Body Surface Area (BSA), and Dermatology Life Quality Index (DLQI) [42] Obesity is associated with higher odds of therapy failure and with lower likelihood of achieving or sustaining remission measured by MDA, DAPSA, and c-DAPSA, especially with anti-TNF agents [44-47]. However, a study by M. Mulder et al. on 855 PsA patients showed that BMI was associated with not reaching the Psoriatic Arthritis Disease Activity Score (PASDAS) low disease activity (LDA) only in females, with the strongest effect observed in those with BMI 25–30 with OR = 3.43 (95% CI 1.76–6.68, $p < 0.001$). These results suggest that overweight may reduce the chance of remission, in contrast to other reports; however, due to overlapping confidence intervals, they should be interpreted with caution [61]. It is worth noting that dietary interventions (calorie restriction and omega-3 supplementation with omega-6 intake reduction) and physical exercise reduce dermatological symptoms (PASI) and are significantly correlated with the chances of minimal PsA activity, with an OR = 4.20 (95% CI 1.82–9.66, $p < 0.001$) [62,63].

5. Ankylosing Spondylitis and Axial Spondyloarthritis

Ankylosing spondylitis (AS) is a radiographic subtype of axial spondyloarthritis (axSpA), which also includes non-radiographic axSpA. Together with psoriatic arthritis, reactive arthritis, and arthritis associated with inflammatory bowel disease, AS belongs to the broader family of spondyloarthritis [64]. AS, similar to PsA, is currently considered to be predominantly an autoinflammatory rather than a classical autoimmune condition, because of the lack of specific autoantibodies. It primarily affects the spine and sacroiliac joints, presenting with chronic back pain, morning stiffness, reduced spinal mobility, and extra-articular manifestations such as enthesitis (especially Achilles tendonitis), uveitis, dactylitis, osteoporosis, intestinal inflammation (including ileitis), pulmonary fibrosis, and aortic regurgitation [65]. Patients with AS are also at increased risk of cardiovascular complications, including myocardial infarction and stroke [66]. A meta-analysis by Dean et al. (2014) demonstrated marked geographic variation in prevalence, estimated prevalence rates were 23.8 per 10,000 in Europe, 16.7 in Asia, 31.9 in North America, and 7.4 in Africa [67]. AS is characterised by male

predominance (7:3 male-to-female ratio) and HLA-B27 carriage in approximately 80% of cases [68]. Approximately 14% of axSpA patients are obese [69]. Table 4. summarises studies evaluating the clinical impact of obesity on disease activity and treatment outcomes.

Table 4. Clinical impact of obesity on ankylosing spondylitis and axial spondyloarthritis

Study	**Key findings**
meta-analysis by A. Ortolan et al., 2021, 2504 axSpA patients [72]	in obese patients in comparison to normal weight control group disease activity measured as BASDAI with MD = 0.78 (95% CI 0.47–1.07) and as ASDAS with MD = 0.42 (95% CI 0.23–0.63) were higher there was no significant differences between overweight and normal weight patients
meta-analysis by J. Liew et al., 2020, 4054 axSpA patients [73]	in overweight and obese patients in comparison to normal weight control group disease activity measured as BASDAI with SMD = 0.38 (95% CI 0.21–0.55, I^2 =75.2%) and as ASDAS with SMD = 0.40 (95% CI 0.27–0.54)
meta-analysis by S. Singh et al., 2018, 966 axSpA cases [44]	in 6 studies 3-12 months therapies with anti-TNF agents, mainly adalimumab, etanercept and infliximab obese patients have higher risk of therapy failure (not reaching ASAS20 or BASDAI50 response) with OR = 3.36 (95% CI 1.33–8.51, I^2 = 81%); BMI was positively correlated with risk of therapy failure
meta-analysis by G. Jones et al., 2025, 8737 axSpA patients [76]	treated with anti-TNF agents overweight (OR = 0.75, 95% CI 0.64–0.88) and obese (OR = 0.55, 95% CI 0.46–0.68) patients had significantly lower chances of achieving BASDAI50 response at 3 months compared to normal weight and at 12 months (OR = 0.76, 95% CI 0.66–0.87; OR = 0.53 95% CI 0.45–0.63, respectively) after adjustment for age, sex, disease activity and baseline disease activity; results remained statistically significant after adjustment for other lifestyle factors, smoking and alcohol consumption; there were observed lower chances of various ASDAS and ASAS responses
prospective cohort study by B.	79/180 patients treated with anti-TNF agent (infliximab or adalimumab) received additionally csDMARDs (methotrexate and/or sulfasalazine)

Hernández-Breijo et al., 2019, 180 axSpA patients [77]	administration of concomitant csDMARDs was independently associated with serum anti-TNF-agents persistence with OR = 3.82 (95% CI 1.06–13.84); the use of concomitant csDMARDs contributed positively to achieve clinical response ($\Delta BASDAI \geq 2$) with OR = 7.86 (95% CI 2.39–25.78) and remission ($BASDAI < 2$ and $CRP \leq 5$ mg/L) with OR = 4.84 (95% CI 1.09–21.36) in overweight/obese patients, but no association was found for normal-weight patients

Abbreviations: axSpA – axial spondyloarthritis, AS – ankylosing spondylitis, BASDAI – bath ankylosing spondylitis disease activity index, ASDAS – ankylosing spondylitis disease activity score, ASAS20 – assessment of Spondyloarthritis International Society 20% improvement criteria, BASDAI50 – BASDAI 50% improvement criteria, csDMARDs – conventional synthetic disease-modifying antirheumatic drugs, CRP – C-reactive protein

There is no evidence that obesity increases the risk of developing AS/axSpA, and MR studies have also demonstrated that genetic predisposition to obesity does not correlate with an increased risk of these diseases [70,71], however, once the disease is established, obesity significantly affects its course and treatment outcomes. Obese patients, especially those with central adiposity, tend to have higher disease activity (BASDAI, ASDAS), elevated inflammatory markers (CRP, ESR), and reduced physical mobility, although some studies did not confirm this association in overweight people, suggesting a non-linear relationship. Moreover, obesity is correlated with more severe radiographic progression and a higher risk of syndesmophyte formation [72-75]. Obesity also decreases the likelihood of achieving a therapeutic response to anti-TNF agents, however the concomitant administration of conventional synthetic DMARDs (methotrexate and/or sulfasalazine) significantly improves drug persistence and increases the probability of achieving response or remission in overweight and obese patients [44,76,77]. Higher BMI does not significantly affect treatment outcomes with JAK inhibitors (upadacitinib) or IL-17 inhibitors (secukinumab, bimekizumab) [78-80]. These findings highlight the need for treatment individualisation, and monotherapy with anti-TNF agents may be less effective and should be used with caution in obese patients with axSpA.

6. Summary

Presented data show that obesity has a complex impact on autoimmune rheumatic diseases. It is associated with an increased risk of developing RA, SLE, and psoriasis. These associations appear to differ depending on the timing and phenotype of obesity, with stronger evidence for childhood and visceral obesity than for adult obesity per se. These observations suggest that the promotion of maintaining a healthy weight in children should be considered a priority in public health strategies. Modern methods of genome analysis have also shown that genetic predisposition to obesity may be associated with an increased risk of RA development, but do not confirm this for psoriasis. Obesity affects severity and clinical manifestations of autoimmune rheumatic diseases. Positive correlations between disease activity in RA, psoriasis, and AS and obesity suggest

that weight loss should be considered as a recommendation for patients with these diseases. The efficacy of weight loss as a treatment needs evaluation. Data also suggest that obesity is correlated with an increased risk of certain clinical manifestations of diseases, including LN in SLE patients and PsA in patients with psoriasis. Obesity also affects the chances of favourable treatment outcomes with anti-TNF agents; however, this effect seems to be reduced by co-administration of DMARDs. It indicates the necessity of an individualised therapeutic approach in this group of patients.

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