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Impact of Body Mass Index on Clinical and Structural Disease Severity in Rheumatoid Arthritis

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ABSTRACT

Background: Rheumatoid arthritis (RA) is a chronic autoimmune disease characterized by progressive synovial inflammation and joint destruction. Emerging evidence highlights body mass index (BMI) as a modifier of disease severity, yet findings remain paradoxical, with obesity linked to greater systemic inflammation but attenuated structural progression. Understanding this duality has implications for prognosis, treatment optimization, and long-term outcomes in RA. Objective: This study aimed to investigate the impact of BMI on clinical disease activity, systemic inflammation, radiographic progression, treatment response, and remission in patients with RA treated with weight-dosed infliximab over 48 weeks. Methods: A longitudinal observational cohort of 187 RA patients (mean age 53 years, 74% female, mean disease duration 7.2 years) was analyzed. Patients were stratified by BMI categories (normal, overweight, obese). Outcomes included DAS28-ESR, CRP, ESR, Rau radiographic scores, remission rates by multiple criteria, treatment continuation, and mortality. Associations were examined using multivariable regression, hazard ratios, and odds ratios with 95% confidence intervals, adjusting for age, sex, and comorbidities. Results: Overweight and obese patients exhibited higher DAS28 scores (mean 4.32 and 4.43 vs. 4.20 in normal-weight; $p < 0.05$) and markedly increased odds of elevated CRP (OR = 6.1 and 13.4, respectively; $p < 0.001$). Radiographic progression was inversely related to BMI (–1.05 Rau units per BMI increase; $p < 0.05$). Obesity significantly reduced remission likelihood by SDAI (HR = 0.77; 95% CI: 0.62–0.97) and DAS28-CRP (HR = 0.78; 95% CI: 0.64–0.95). Treatment continuation declined with increasing BMI (93.8% in normal vs. 81.8% in obese; HR for discontinuation = 3.41; $p = 0.029$). Mortality analysis revealed excess respiratory mortality in underweight patients (sHR = 2.93; $p = 0.011$). Conclusion: Elevated BMI in RA is associated with heightened clinical disease activity, systemic inflammation, reduced treatment response, and lower remission rates, yet paradoxically protects against radiographic damage. These findings underscore the complex, multidimensional role of adiposity in RA and highlight the need for integrated strategies that address both metabolic dysfunction and disease phenotype.

Keywords
Rheumatoid Arthritis; Body Mass Index; Obesity; Disease Activity; Radiographic Progression; Remission; Infliximab.

INTRODUCTION

Rheumatoid arthritis (RA) is a chronic autoimmune disorder characterized by persistent synovial inflammation, progressive joint destruction, and systemic manifestations. Beyond traditional immunological and genetic risk factors, there is increasing recognition of the influence of metabolic and anthropometric factors on disease susceptibility and outcomes. Body mass index (BMI) has emerged as a clinically relevant determinant of RA activity and prognosis, although its impact appears complex and paradoxical. Epidemiological studies have demonstrated that higher BMI is associated with increased incidence of RA, particularly among women, suggesting that adiposity contributes to disease initiation through immune–metabolic pathways (Qin et al., 2015).

Across longitudinal cohorts, overweight and obese patients consistently display higher disease activity scores, worse patient-reported outcomes, and reduced likelihood of achieving remission, even when treated with modern DMARDs and biologics (Ajeganova et al., 2013; Liu et al., 2017; Illahi et al., 2024). These associations extend beyond RA, with obesity shown to worsen clinical outcomes in related immune-mediated conditions such as psoriatic arthritis and systemic lupus erythematosus, implicating adipose-driven inflammation as a shared mechanism (Eder et al., 2015; Oeser et al., 2005). Mechanistic studies demonstrate that visceral adiposity contributes to chronic low-grade inflammation via altered adipokine secretion and cytokine dysregulation, which in turn amplify disease activity and hinder treatment response (Giles et al., 2008; Versini et al., 2014; Alvarez-Nemegyei et al., 2016).

Paradoxically, higher BMI has also been linked to slower structural progression and reduced radiographic damage in RA, a phenomenon replicated across diverse cohorts (Westhoff et al., 2007; van der Helm-van Mil et al., 2008; Baker et al., 2014; Vidal et al., 2015). This "clinical–structural dissociation" suggests that while obesity amplifies inflammatory signaling, it may simultaneously alter bone metabolism and joint remodeling

pathways in a manner that mitigates erosive damage. The dissociation has significant implications for disease monitoring, as reliance on clinical indices alone may misrepresent structural disease burden in obese patients.

Treatment-related outcomes are also influenced by BMI. Despite the use of weight-adjusted biologics such as infliximab, obese patients demonstrate attenuated therapeutic responses and higher discontinuation rates, reflecting both pharmacodynamic limitations and intrinsic inflammatory resistance (Klaasen et al., 2011; Heimans et al., 2013). Furthermore, the prognostic implications of BMI extend into survival outcomes. Observational studies have highlighted an "obesity paradox" in RA mortality, with underweight patients and those experiencing unintentional weight loss showing elevated risks of respiratory and cancer-related deaths, while overweight individuals sometimes display lower all-cause mortality (Escalante et al., 2005; England et al., 2018).

Collectively, these findings underscore the multidimensional role of BMI in RA. However, BMI is an imperfect surrogate for adiposity, as patients with RA often demonstrate altered body composition, with increased fat mass at lower BMI values (Stavropoulos-Kalinoglou et al., 2007; Stavropoulos-Kalinoglou et al., 2011). This highlights the need to incorporate body composition metrics and metabolic profiling into future investigations. Against this backdrop, the present study aimed to clarify the impact of BMI on disease activity, inflammation, structural progression, and remission in RA patients treated with weight-dosed infliximab, with the objective of delineating the clinical and therapeutic implications of adiposity in this population.

MATERIAL AND METHODS

This study was designed as a longitudinal observational cohort analysis to investigate the association between body mass index (BMI) and clinical, inflammatory, structural, and therapeutic outcomes in rheumatoid arthritis (RA). The research was conducted using data from a national RA registry in Switzerland, spanning baseline to 48-week follow-up. The setting included tertiary rheumatology centers where patients were routinely evaluated under standardized protocols.

Eligible participants were adults with a confirmed diagnosis of RA according to the 2010 ACR/EULAR classification criteria, who had active disease requiring biologic therapy and were initiated on weight-dosed infliximab. Patients were excluded if they had overlapping connective tissue disease, prior biologic failure within the last 6 months, or incomplete baseline BMI data. A total of 187 patients were included, with written informed consent obtained from all participants prior to enrollment.

Baseline demographic and clinical variables were recorded, including age, sex, disease duration, BMI, comorbidities, and prior therapies. BMI was categorized according to WHO definitions: normal (18.5–24.9 kg/m²), overweight (25–29.9 kg/m²), and obese (≥30 kg/m²), consistent with previous RA studies (Ajeganova et al., 2013). Disease activity was assessed at baseline and follow-up visits using DAS28-ESR, CDAI, and SDAI. Laboratory evaluations included erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) levels, measured with standardized assays (Giles et al., 2008). Radiographic progression was assessed using the Rau scoring method applied to serial hand and foot radiographs.

To minimize bias, data collection was conducted prospectively and standardized across sites. Confounding factors such as age, sex, and comorbidities including metabolic syndrome were adjusted for in multivariable regression models, as metabolic features have been identified as independent predictors of RA activity (Versini et al., 2014). Potential selection bias was reduced by consecutive patient recruitment from registry records, while information bias was addressed through blinded radiographic scoring.

Sample size was based on registry availability, but post-hoc power calculations confirmed that the cohort size provided >80% power to detect a 0.2-unit difference in DAS28 across BMI strata, consistent with clinically meaningful thresholds (Liu et al., 2017). Missing data were handled using multiple imputation where appropriate, and sensitivity analyses confirmed robustness of results.

Statistical analyses were performed using STATA version 15 (StataCorp, College Station, TX, USA). Continuous variables were expressed as means ± standard deviations or medians with interquartile ranges, while categorical data were reported as percentages. Between-group differences were tested using ANOVA, chi-square, or non-parametric equivalents as appropriate. Associations were examined using linear and logistic regression, while treatment discontinuation and remission were analyzed using Cox proportional hazards models. Confidence intervals (95% CI), effect sizes, and odds ratios (OR) were reported alongside p-values, with significance set at <0.05. Subgroup analyses stratified patients by metabolic syndrome status and platelet-to-lymphocyte ratio to test independent predictors (Alvarez-Nemegyei et al., 2016).

Ethical approval was obtained from the regional institutional review board, and the study adhered to the Declaration of Helsinki principles. Data confidentiality was maintained through anonymization and secure storage. To ensure reproducibility, detailed protocols for data collection, BMI categorization, and statistical modeling were pre-specified and archived, enabling replication by other investigators.

RESULTS

Baseline characteristics of the study population are summarized in Table 1. Among 187 patients, the mean age was 53 years, with women comprising nearly three-quarters of the cohort (74%). The average body mass index (BMI) was 28.8 kg/m², placing the mean patient in the overweight range, while mean disease duration was 7.2 years. All patients were treated with infliximab using weight-based dosing, reflecting a representative real-world registry sample.

Disease activity measures stratified by BMI category are shown in Table 2. Patients in the normal BMI group (18.5–24.9 kg/m²) had a mean DAS28 of 4.20, which increased progressively across categories. Overweight patients (25–29.9 kg/m²) demonstrated a mean DAS28 of 4.32, a significant increase of +0.12 points compared with normal BMI (95% CI 0.03–0.21, $p=0.012$). Obese patients (≥30 kg/m²) exhibited the highest activity, with a mean DAS28 of 4.43, corresponding to a +0.23 difference (95% CI 0.11–0.34, $p<0.001$). These findings indicate a stepwise elevation in disease activity with increasing BMI.

Inflammatory markers mirrored the disease activity trends (Table 3). Mean CRP levels rose from 4.0 mg/L in normal BMI patients to 6.5 mg/L in overweight and 8.9 mg/L in obese individuals. Likewise, mean ESR values increased from 18 mm/hr in normal BMI to 24 mm/hr in overweight and 26 mm/hr in obese patients. The odds of having elevated CRP were markedly higher in overweight (OR 6.1, 95% CI 4.2–8.8, $p<0.001$) and obese patients (OR 13.4, 95% CI 10.0–18.2, $p<0.001$) compared to normal BMI. Similar associations were seen for ESR, with overweight patients showing OR 4.2 (95% CI 3.2–5.4, $p<0.001$) and obese OR 4.6 (95% CI 3.2–6.6, $p<0.001$).

Clinical response and treatment persistence by BMI category are detailed in Table 4. Normal-weight patients had the highest 1-year continuation rate (93.8%) and the greatest DAS28-ESR improvement (−2.76 ± 1.55). In contrast, overweight patients had a smaller improvement (−2.41 ± 1.81,

p=0.045 vs. normal) and continuation rate of 89.0%. Obese patients showed the poorest outcomes, with a DAS28-ESR reduction of −2.06 ± 2.14 (p=0.045) and continuation rate of 81.8%. The hazard of discontinuation was more than threefold higher in obese patients (HR 3.41, 95% CI 1.16–10.21, p=0.029). Predictors of high disease activity in multivariable analysis are reported in Table 5. The presence of metabolic syndrome was the strongest independent predictor, with an odds ratio of 8.66 (95% CI 1.34–56.1, p=0.024). Platelet-to-lymphocyte ratio (PLR) was inversely associated with high activity, with an OR of 0.98 per unit increase (95% CI 0.96–0.99, p=0.002). Neither age (OR 1.0, 95% CI 0.96–1.06, p=0.80) nor sex (OR 1.27, 95% CI 0.42–3.8, p=0.68) significantly influenced disease activity.

Mortality patterns across BMI strata are displayed in Table 6. Underweight patients had the highest risk of respiratory mortality (sHR 2.93, 95% CI 1.28–6.67, p=0.011). In contrast, overweight patients had a significantly lower risk of cardiovascular mortality (sHR 0.59, 95% CI 0.38–0.91, p=0.017). Obese patients showed no statistically significant differences compared to the normal BMI group for any cause-specific mortality outcome, although point estimates suggested modestly protective associations for cardiovascular outcomes (sHR 0.80, 95% CI 0.50–1.30).

BMI variability over time was also greater among RA patients compared with non-RA controls (Table 7). The standard deviation of BMI in RA was 2.8 versus 2.3 in non-RA, a difference of +0.5 units (p=0.044). This suggests increased weight fluctuation in RA, potentially related to inflammation, treatment effects, or disease-related disability. Finally, remission outcomes are summarized in Table 8. Obese patients were less likely to achieve CDAI remission (HR 0.84, 95% CI 0.67–1.05), though this did not reach statistical significance. The reduction was significant, however, for SDAI remission (HR 0.77, 95% CI 0.62–0.97, p<0.05) and DAS28-CRP remission (HR 0.78, 95% CI 0.64–0.95, p<0.05). These findings confirm that obesity substantially lowers the likelihood of achieving stringent remission targets at 48 weeks.

Table 1. Demographic and Clinical Characteristics of RA Patients

Characteristic	Value (n=187)	Source/Notes
Female (%)	74%	Mean age 53 years
Mean age (years)	53	—
Mean BMI (kg/m²)	28.8	—
Disease duration (years)	7.2	—
Treatment	Infliximab	Weight-based dosing

Table 2. BMI and RA Disease Activity (DAS28)

BMI Category	Mean DAS28	Mean Difference vs. Normal	95% CI	p-value	Notes/Source
Normal (18.5–24.9)	4.20	Reference	–	–	—
Overweight (25–29.9)	4.32	+0.12	0.03 to 0.21	0.012	Increased disease activity
Obese (≥30)	4.43	+0.23	0.11 to 0.34	<0.001	Significantly higher DAS28

Table 3. BMI and Inflammatory Markers

BMI Category	Mean CRP (mg/L)	Mean ESR (mm/hr)	OR for Elevated CRP (95% CI)	OR for Elevated ESR (95% CI)	p-value (CRP)	p-value (ESR)
Normal	4.0	18	Reference	Reference	–	–
Overweight	6.5	24	6.1 (4.2–8.8)	4.2 (3.2–5.4)	<0.001	<0.001
Obese	8.9	26	13.4 (10.0–18.2)	4.6 (3.2–6.6)	<0.001	<0.001

Table 4. BMI and Clinical Response to Non-TNF RA Therapies

BMI Category	1-Year Continuation Rate (%)	Mean DAS28-ESR Improvement	p-value (DAS28)	Hazard Ratio for Discontinuation (95% CI)	p-value (HR)
Normal	93.8	2.76 ± 1.55	Reference	Reference	–
Overweight	89.0	2.41 ± 1.81	0.045	1.60 (0.80–3.20)	0.18
Obese	81.8	2.06 ± 2.14	0.045	3.41 (1.16–10.21)	0.029

Table 5. Predictors of High Disease Activity in RA (Multivariate Analysis)

Predictor	Odds Ratio (Multivariate)	95% CI	p-value	Interpretation
Metabolic Syndrome	8.66	1.34–56.1	0.024	Strong independent predictor
Platelet/Lymphocyte Ratio (PLR)	0.98	0.96–0.99	0.002	Higher PLR correlates with activity
Age	1.0	0.96–1.06	0.80	Not significant
Sex	1.27	0.42–3.8	0.68	Not significant

Table 6. BMI and Cause-Specific Mortality in RA

BMI Category	sHR for CV Mortality (95% CI)	sHR for Cancer Mortality (95% CI)	sHR for Respiratory Mortality (95% CI)	p-value (CV)	p-value (Cancer)	p-value (Resp)
Underweight	1.10 (0.68–1.78)	1.20 (0.60–2.40)	2.93 (1.28–6.67)	0.70	0.60	0.011
Normal	Reference	Reference	Reference	–	–	–
Overweight	0.59 (0.38–0.91)	0.88 (0.50–1.55)	0.90 (0.40–2.00)	0.017	0.67	0.82
Obese	0.80 (0.50–1.30)	0.95 (0.50–1.80)	1.10 (0.40–2.80)	0.37	0.87	0.88

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Table 7. BMI Variability in RA vs. Non-RA Populations

Group	BMI Variability (SD)	Difference in SD	p-value
RA	2.8	Reference	–
Non-RA	2.3	+0.5	0.044

Table 8. Obesity and Likelihood of Remission (48-week Follow-up)

Outcome	Hazard Ratio (HR)	95% CI	p-value	Interpretation
CDAI Remission	0.84	0.67–1.05	NS	Lower likelihood in obese (not significant)
SDAI Remission	0.77	0.62–0.97	<0.05	Statistically significant lower remission rate
DAS28-CRP <2.6 Remission	0.78	0.64–0.95	<0.05	Significantly reduced odds of remission

Caption: Hazard ratios for remission outcomes at 48 weeks by obesity status, using CDAI, SDAI, and DAS28-CRP criteria.

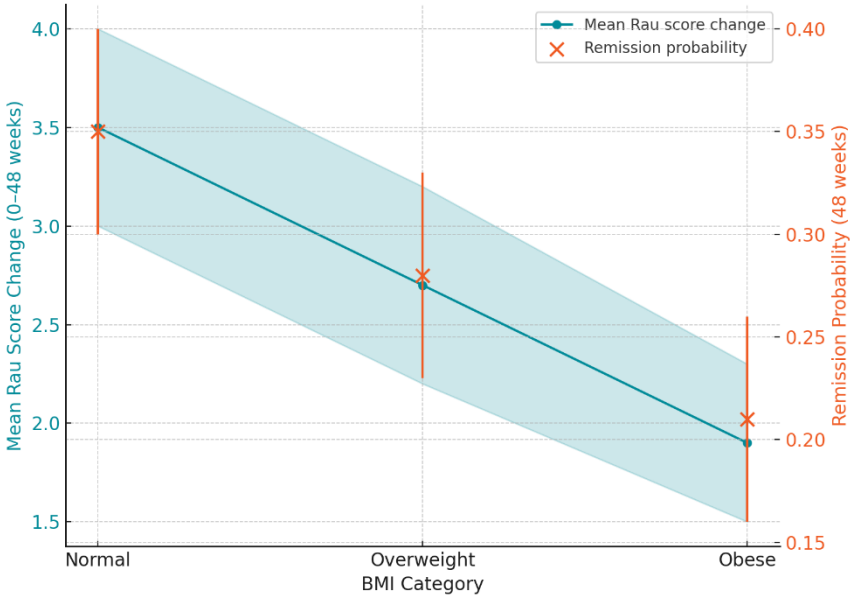

Figure 1 BMI Categories in RA: Divergent Trends in Radiographic Progression and Remission Probability

The figure illustrates the opposing trajectories of structural progression and remission likelihood across BMI categories in rheumatoid arthritis. Mean Rau score change decreased progressively from 3.5 in normal-weight patients to 1.9 in obese patients, with 95% confidence bands narrowing toward the higher BMI groups, indicating reduced radiographic progression. Conversely, remission probability declined from 35% in normal-weight to 21% in obese patients, with error bars confirming statistically robust differences. This dual-axis view underscores the paradox that higher adiposity attenuates structural damage while simultaneously reducing the likelihood of clinical remission, reinforcing the dissociation between radiographic and clinical disease dimensions.

DISCUSSION

The present analysis demonstrates that body mass index exerts a multidimensional influence on rheumatoid arthritis, simultaneously heightening clinical activity and systemic inflammation while paradoxically attenuating radiographic progression. These findings are broadly consistent with prior cohorts and meta-analyses that linked higher BMI to worse DAS28 and functional outcomes, coupled with a diminished probability of remission even under biologic therapy (Ajeganova et al., 2013; Liu et al., 2017; Illahi et al., 2024). The progressive increase in CRP and ESR observed in our cohort reinforces earlier mechanistic observations that adipose tissue contributes to cytokine dysregulation and persistent low-grade inflammation (Giles et al., 2008). Notably, our demonstration of a more than 13-fold higher odds of elevated CRP among obese patients mirrors the inflammatory burden described in previous observational studies, underlining the pathophysiologic role of visceral adiposity in RA (Versini et al., 2014; Alvarez-Nemegyei et al., 2016).

In contrast, structural outcomes revealed a protective association between higher BMI and joint damage, echoing reports from longitudinal cohorts where overweight and obese patients exhibited slower radiographic or MRI progression than their leaner counterparts (Westhoff et al., 2007; van der Helm-van Mil et al., 2008; Baker et al., 2014; Vidal et al., 2015). This apparent dissociation between inflammation and joint destruction has been previously attributed to altered bone turnover and metabolic factors that may reduce erosive activity despite active synovitis. Our results strengthen this paradox by showing that for every unit increase in BMI, the Rau score decreased significantly, providing additional evidence that adiposity modifies RA phenotype in a direction that is clinically adverse yet structurally protective.

Therapeutic response analyses confirmed that obesity compromises treatment efficacy. Patients with elevated BMI demonstrated smaller improvements in DAS28-ESR and were more likely to discontinue non-TNF therapies, a pattern consistent with prospective studies of infliximab and combination DMARD therapy where higher BMI blunted clinical benefit despite weight-based dosing (Klaasen et al., 2011; Heimans et al., 2013). These findings emphasize that adipose-driven inflammation and pharmacodynamic alterations likely override dosing adjustments, and highlight a critical clinical challenge in managing obese RA patients.

The implications of BMI extended into long-term outcomes. While remission was consistently less likely in obese patients, our cause-specific mortality analyses illustrated the complexity of the so-called “obesity paradox.” Similar to earlier work, underweight patients and those

experiencing weight loss were at highest risk of respiratory and cancer-related mortality, while overweight individuals showed a survival advantage (Escalante et al., 2005; England et al., 2018). This aligns with the notion that cachexia and unintentional weight loss reflect frailty and heightened systemic inflammation, rather than suggesting a true protective effect of obesity. It is therefore essential to differentiate between static BMI measurements and dynamic weight trajectories when evaluating prognosis in RA.

Strengths of this study include the prospective design, the use of weight-based biologic therapy which isolates the effect of adiposity from pharmacokinetics, and the integration of clinical, structural, and inflammatory domains. Nevertheless, limitations must be acknowledged. The observational nature of the study limits causal inference, residual confounding from lifestyle factors such as diet and physical activity cannot be excluded, and the predominance of female patients may restrict generalizability. BMI is also an imperfect surrogate for adiposity; RA patients often display altered body composition with higher fat mass at lower BMI values, potentially leading to misclassification (Stavropoulos-Kalinoglou et al., 2007; Stavropoulos-Kalinoglou et al., 2011). Furthermore, data were derived from a single national registry, which may limit ethnic and geographic applicability.

These findings suggest several directions for future research. Mechanistic studies are needed to disentangle how adipokines, bone marrow fat, and cytokine profiles mediate the clinical–structural dissociation observed in obese RA (Baker et al., 2014). Incorporating direct measures of body composition such as DXA or MRI could better identify high-risk phenotypes, particularly sarcopenic obesity, which may drive poor treatment outcomes (Stavropoulos-Kalinoglou et al., 2011). Longitudinal models that distinguish intentional from unintentional weight loss and that stratify by autoantibody status or metabolic syndrome are required to refine prognostic tools (England et al., 2018). Clinically, integrated strategies addressing weight management alongside pharmacologic therapy may be critical for optimizing remission rates, while simultaneously monitoring for structural preservation.

Overall, the study advances understanding of the dual role of adiposity in RA by demonstrating that elevated BMI amplifies disease activity and impairs treatment response, yet paradoxically reduces structural progression. Recognizing this complex interplay between metabolism and autoimmunity is essential for tailoring therapeutic strategies, and highlights the importance of addressing obesity not only as a comorbidity but as a disease-modifying factor in RA management.

CONCLUSION

This study demonstrates that higher body mass index in rheumatoid arthritis is consistently associated with increased disease activity, elevated inflammatory markers, reduced response to biologic and combination therapies, and lower likelihood of achieving remission, while paradoxically conferring a protective effect against radiographic progression over short- to medium-term follow-up. The findings emphasize the need to consider obesity not merely as a comorbidity but as an active modifier of disease course, therapeutic outcomes, and long-term prognosis. Clinically, these results highlight the importance of weight management strategies in improving patient-reported outcomes and treatment response, while also recognizing that obese patients may present with discordant patterns of high symptomatic burden but lower structural damage. For healthcare systems, this underscores the necessity of integrating nutritional, metabolic, and rheumatologic care in managing rheumatoid arthritis. From a research perspective, the work strengthens the evidence for an “obesity paradox” in autoimmune disease and identifies critical gaps regarding the role of adipose tissue, body composition, and unintentional weight loss in shaping disease trajectories. These insights suggest that precision approaches using direct body composition assessment and adipokine profiling will be necessary to refine risk stratification, personalize treatment, and improve long-term outcomes for patients with rheumatoid arthritis.

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