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ORIGINAL ARTICLE

Body mass index and treatment response to subcutaneous abatacept in patients with psoriatic arthritis: a *post hoc* analysis of a phase III trial

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ABSTRACT

Objective This *post hoc* analysis of the phase III Active PSoriaTic Arthritis RAndomizEd TriAl (ASTRAEA) evaluated the effect of baseline body mass index (BMI) on subsequent response to subcutaneous (SC) abatacept in patients with psoriatic arthritis (PsA).

Methods In ASTRAEA, patients with active PsA were randomised (1:1) to receive blinded weekly SC abatacept 125 mg or placebo for 24 weeks. Treatment response at week 24 was assessed by the proportions of patients achieving American College of Rheumatology 20% improvement response, Disease Activity Score in 28 joints (DAS28 (C reactive protein (CRP))) ≤3.6 and <2.6, Health Assessment Questionnaire-Disability Index reduction from baseline ≥0.35 and radiographic non-progression (defined as change from baseline ≤0 in PsA-modified total Sharp/van der Heijde score). Responses were stratified by baseline BMI (underweight/normal, <25 kg/m^2; overweight, 25–30 kg/m^2; obese, >30 kg/m^2) and compared in univariate and multivariate models.

Results Of 212/213 and 210/211 patients with baseline BMI data in the abatacept and placebo groups, respectively, 15% and 19% were underweight/normal, 36% and 27% were overweight, and 49% and 54% were obese. After adjusting for baseline characteristics, there were no significant differences for any outcome measure at week 24 with abatacept in the overweight or obese versus underweight/normal subgroup. In the placebo group, patients in the obese versus underweight/normal subgroup were significantly less likely to achieve DAS28 (CRP) <2.6 at week 24 (OR 0.26; 95% CI 0.08 to 0.87; p=0.03).

Conclusion BMI does not impact clinical or radiographic response to SC abatacept in patients with PsA.

Trial registration number NCT01860976.

Key messages

What is already known about this subject?

- Patients with psoriatic arthritis (PsA) tend to have a higher body mass index (BMI) than the general population. A high BMI can be associated with less favourable clinical outcomes with biologics, such as tumour necrosis factor inhibitors.

What does this study add?

- This analysis evaluated the relationship between BMI and treatment response to subcutaneous (SC) abatacept in patients with PsA.
- Obese and overweight patients with PsA had a statistically similar treatment response to SC abatacept compared with patients who were underweight or had normal BMI.

How might this impact on clinical practice or future developments?

- These findings suggest that SC abatacept could be considered for patients with PsA irrespective of BMI status.

INTRODUCTION

Patients with psoriatic arthritis (PsA) are twice as likely as the general population to be obese, as defined by body mass index (BMI) ≥30 kg/m^2, with a prevalence of obesity among patients with PsA of 37%.[1] Tumour necrosis factor inhibitors (TNFis), a class of biologic (b) disease-modifying antirheumatic drugs (DMARDs), are an effective treatment option for PsA; however, a meta-analysis of 20 randomised clinical trials and 34 observational studies in patients with rheumatic diseases, including PsA, concluded that obesity is associated with reduced treatment response to TNFis in these patients.[2] The effect of obesity on the efficacy of bDMARDs with other mechanisms of action is unclear. An understanding of the potential impact of BMI on treatment-related outcomes in PsA is important for informing clinical decision-making in practice; therefore, information on the effect of obesity on the efficacy of non-TNFi bDMARDs for PsA is needed.

Abatacept is a cytotoxic T lymphocyte-associated antigen-4-immunoglobulin fusion molecule that selectively modulates T-cell co-stimulation and activation through the inhibition of the CD80/CD86:CD28 co-stimulatory signal. T-cell-driven pathways are implicated in the pathogenesis of immunological diseases including PsA, making abatacept a plausible therapeutic candidate.[3] Indeed, abatacept, available as an intravenous or subcutaneous (SC) formulation, is approved for the treatment of moderate-to-severe rheumatoid arthritis (RA), juvenile idiopathic arthritis and PsA.[4] Evidence from interventional trials and real-world studies has shown that BMI does not affect treatment response to abatacept in patients with RA.[5 6] However, there are limited data on the impact of BMI on response to abatacept in patients with PsA.

In the randomised, double-blind, placebo-controlled, international phase III Active PSoriaTic Arthritis RAndomizEd TriAl (ASTRAEA), the American College of Rheumatology 20% improvement (ACR20) response rate in patients with PsA at week 24 was significantly higher with abatacept versus placebo.[7] Here, we present findings from an analysis of ASTRAEA to explore the impact of patient BMI at baseline on the response to SC abatacept.

METHODS

Study design and treatment

A *post hoc* analysis of the ASTRAEA study was conducted to evaluate the effect of baseline BMI on the response to SC abatacept in patients with PsA. The study design, ethics approvals, study population, patient eligibility criteria and main endpoints of ASTRAEA have been reported previously.[7] The trial is registered at www.clinicaltrials.gov.

Briefly, patients with active PsA and an inadequate response or intolerance to ≥1 non-bDMARD were randomised (1:1) to receive blinded weekly SC abatacept 125 mg or placebo for 24 weeks. Concomitant treatment with methotrexate, leflunomide, sulfasalazine or hydroxychloroquine; non-steroidal anti-inflammatory drugs (NSAIDs) and oral corticosteroids (<10 mg/day); and use of low-potency topical corticosteroids in sensitive areas were permitted. Patients without ≥20% improvement from baseline in swollen or tender joint counts at week 16 were switched to open-label abatacept (early escape) for 28 weeks. Patients designated as an early escape or with missing data were imputed as non-responders.

Study assessments

For patients with available baseline BMI data, all analyses were conducted by baseline BMI subgroup: underweight/normal (<25 kg/m^2), overweight (25–30 kg/m^2) and obese (>30 kg/m^2). Patient demographics and disease characteristics were also reported by BMI subgroup. Treatment response by BMI subgroup at week 24 was assessed by determining the proportion of patients with ACR20 response, Disease Activity Score in 28 joints (DAS28) (C reactive protein (CRP)) ≤3.6 or <2.6, and Health Assessment Questionnaire-Disability Index response (reduction from baseline ≥0.35). Structural damage was assessed by determining the rates of radiographic non-progression (defined as change from baseline ≤0 in PsA-modified total Sharp/van der Heijde score).

Statistical analyses

Baseline patient demographics and disease characteristics were analysed descriptively according to the baseline BMI subgroup (percentage for categorical variables and mean (SD) for continuous variables). Rates for treatment response and radiographic non-progression in each BMI subgroup were determined at week 24 and compared between subgroups using univariable and multivariable analyses with the underweight/normal BMI subgroup as the reference. Key potential confounding factors for treatment efficacy were included in the multivariable model. Results are presented as ORs with corresponding 95% CIs; p values were calculated for each treatment outcome by BMI subgroup, based on a logistic regression model. The ORs were statistically significant when the 95% CIs did not cross 1. An additional stratified multivariable analysis was performed to evaluate treatment responses by baseline BMI subgroup at week 24, (i) in patients who received abatacept without any concomitant non-bDMARD (defined as not receiving any of actarit, apremilast, auranofin, aurothioglucose, aurotioprol, aurotioprol gold salt, azathioprine, bucillamine, chlorambucil, chloroquine, cyclosporine, gold salts, gold sodium thiomalate, hydroxychloroquine, leflunomide, lobenzarit, methotrexate, mizoribine, penicillamine, sulfasalazine, tiopronin or tofacitinib); concomitant NSAIDs, oral corticosteroids or topical corticosteroids were permitted, and (ii) in those who received abatacept in combination with a non-bDMARD.

RESULTS

Analysis population

Baseline BMI data were available for 212/213 and 210/211 patients randomised to abatacept or placebo, respectively. In the abatacept group, 31/212 (14.6%) patients were underweight/normal, 77/212 (36.3%) patients were overweight and 104/212 (49.1%) patients were obese. In the placebo group, 39/210 (18.6%) patients were underweight/normal, 57/210 (27.1%) were overweight and 114/210 (54.3%) were obese. Patient demographic and disease characteristics at baseline by BMI subgroup are presented in table 1.

Treatment response by BMI—univariate analysis

In the abatacept group, there were no significant differences in the likelihood of achieving treatment outcomes at week 24 in the obese or overweight versus underweight/normal subgroup (p≥0.17 for all measurements) (figure 1A). In the placebo group, patients in the obese versus underweight/normal BMI subgroup were significantly less likely to achieve an ACR20 response (OR 0.40; 95% CI 0.18 to 0.90; p=0.03) or DAS28 (CRP) <2.6 (OR 0.34; 95% CI 0.13 to 0.87; p=0.02) at week 24 (figure 1B).

Table 1 Baseline patient characteristics by BMI

	Abatacept (n=212)			Placebo (n=210)		
	Underweight/ normal (n=31)	Overweight (n=77)	Obese (n=104)	Underweight/ normal (n=39)	Overweight (n=57)	Obese (n=114)
Age, years	52.4 (11.3)	49.8 (10.5)	51.5 (10.7)	46.1 (11.8)	48.6 (10.7)	51.8 (11.0)
Female (%)	67.7	42.9	63.5	41.0	40.4	64.0
BMI	22.6 (1.7)	27.5 (1.5)	35.4 (5.5)	22.9 (1.6)	27.5 (1.4)	36.0 (5.4)
Weight, kg	61.9 (9.4)	77.6 (10.4)	97.2 (18.8)	64.0 (9.7)	79.1 (9.6)	98.0 (19.3)
Prior TNFi, n (%)	20 (64.5)	51 (66.2)	57 (54.8)	22 (53.9)	31 (54.4)	75 (65.8)
Concomitant MTX, n (%)	23 (74.2)	43 (55.8)	62 (59.6)	28 (71.8)	36 (63.2)	61 (53.5)
Concomitant oral corticosteroids, n (%)	10 (32.3)	21 (27.3)	22 (21.2)	11 (28.2)	11 (19.3)	26 (22.8)
PsA duration, years	9.0 (8.0)	7.6 (7.5)	8.6 (8.7)	9.1 (8.8)	9.4 (8.7)	8.4 (8.0)
TJC(68)	18.6 (14.6)	20.1 (14.1)	22.3 (12.4)	15.8 (11.2)	17.9 (11.9)	20.9 (13.7)
SJC(66)	10.6 (8.6)	10.9 (6.4)	13.4 (8.4)	10.1 (5.5)	11.1 (7.2)	11.5 (7.6)
HAQ-DI	n=30 1.3 (0.6)	1.3 (0.7)	1.3 (0.8)	1.1 (0.8)	1.2 (0.7)	1.4 (0.7)
CRP, mg/L	13.1 (23.7)	n=76 14.6 (25.7)	13.9 (15.6)	19.7 (54.6)	11.1 (20.0)	n=112 14.1 (21.9)
DAS28 (CRP)	4.8 (1.1)	n=75 5.0 (1.1)	n=103 5.1 (1.0)	4.5 (1.2)	4.8 (0.9)	n=111 5.1 (1.0)
Erosion score	n=29 9.1 (16.3)	n=75 13.5 (28.9)	n=101 14.0 (31.3)	20.2 (27.8)	n=55 11.8 (29.4)	n=112 8.1 (20.0)
Dactylitis, n (%)	13 (41.9)	36 (46.8)	43 (41.4)	10 (25.6)	26 (45.6)	36 (31.6)
Enthesitis*	2.1 (2.0)	1.9 (1.8)	2.2 (2.1)	1.7 (2.0)	2.1 (2.1)	1.8 (1.9)
PASI†	n=23 5.6 (4.9)	n=54 6.3 (6.5)	n=69 8.7 (9.5)	n=30 5.0 (4.5)	n=43 7.0 (7.0)	n=75 8.2 (9.0)
PsA-modified total SHS	n=29 16.9 (32.7)	n=75 22.4 (50.0)	n=101 25.2 (56.2)	n=34 34.6 (47.8)	n=55 22.7 (54.9)	n=112 15.2 (39.7)
BASDAI	n=30 6.1 (2.0)	n=75 6.4 (2.1)	n=103 6.2 (2.1)	5.9 (2.2)	n=56 6.1 (2.2)	n=113 6.3 (2.0)

Data are expressed as mean (SD) unless otherwise noted.
Underweight/normal, BMI <25 kg/m^2; overweight, BMI 25–30 kg/m^2; obese, BMI >30 kg/m^2.
*Measured using the Leeds Enthesitis Index.
†For patients with baseline BSA ≥3%.
BASDAI, Bath Ankylosing Spondylitis Disease Activity Index; BMI, body mass index; BSA, body surface area affected; CRP, C reactive protein; DAS28, Disease Activity Score in 28 joints; HAQ-DI, Health Assessment Questionnaire-Disability Index; MTX, methotrexate; PASI, Psoriasis Area and Severity Index; PsA, psoriatic arthritis; SHS, Sharp/van der Heijde score; SJC, swollen joint count; TJC, tender joint count; TNFi, tumour necrosis factor inhibitor.

There were no significant differences in the likelihood of achieving other treatment outcomes between the obese or overweight versus underweight/normal subgroups (p≥0.10).

Treatment response by BMI—multivariable analyses

In the abatacept group, there were no significant differences in the likelihood of achieving treatment outcomes at week 24 in the obese or overweight versus underweight/normal BMI subgroups (figure 2). In the placebo group, patients in the obese versus underweight/normal BMI subgroup were significantly less likely to achieve DAS28 (CRP) <2.6 (OR 0.26; 95% CI 0.08 to 0.87; p=0.03; figure 2).

In the stratified multivariable analysis of patients receiving abatacept without any non-bDMARD and of those receiving abatacept in combination with a non-bDMARD, there were no significant differences in the likelihood of achieving treatment outcomes between the obese or overweight versus underweight/normal BMI subgroups in either cohort (figure 3).

DISCUSSION

In this *post hoc* analysis of the ASTRAEA study, baseline BMI did not impact treatment response to SC abatacept in patients with active PsA across a range of measures. This

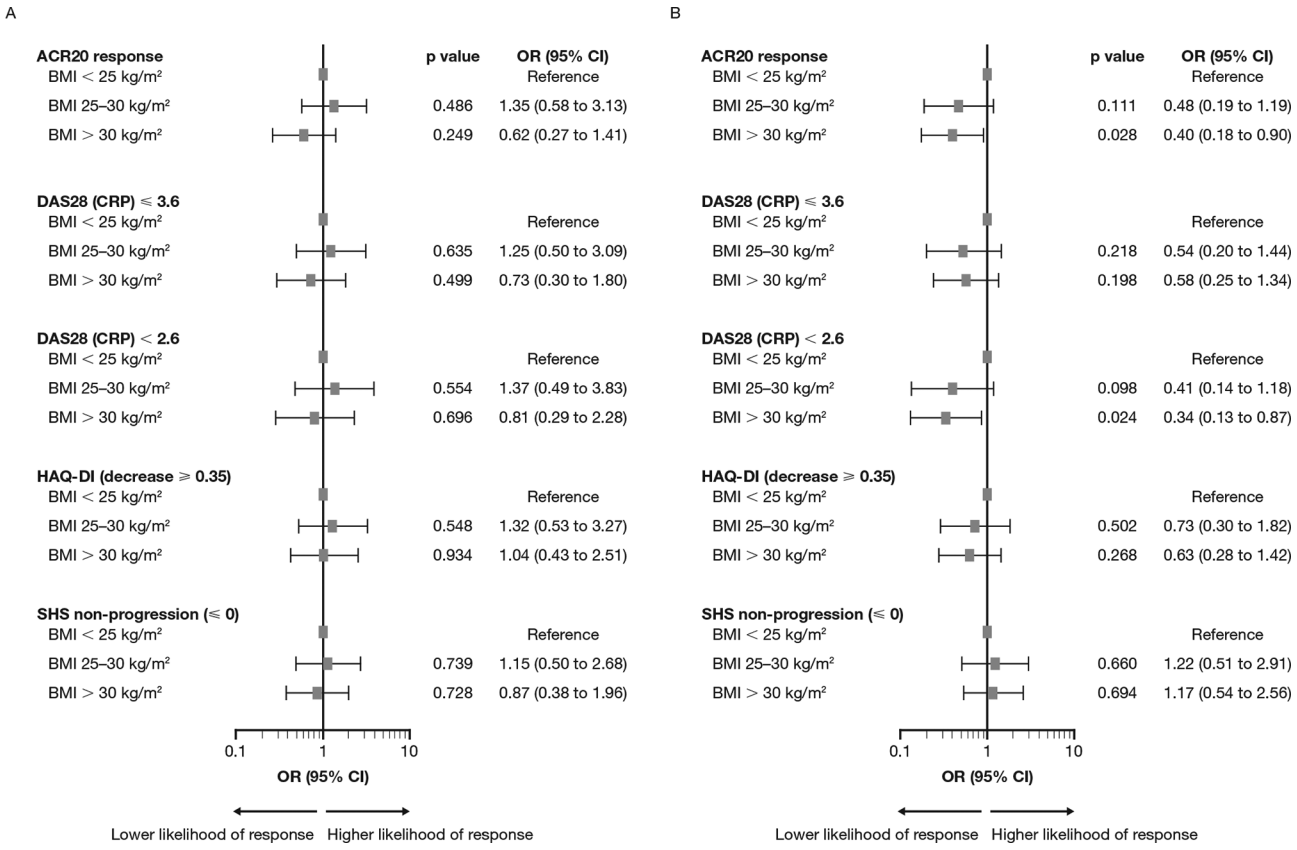

Figure 1 Univariate model of adjusted* comparisons of treatment response at week 24 between BMI subgroups in patients receiving (A) abatacept and (B) placebo. An OR >1 indicates a higher likelihood of response, while an OR <1 indicates a lower likelihood of response. ORs are significant when 95% CIs do not overlap 1. *The model was adjusted for the following baseline factors: treatment, BMI, MTX use, prior TNFis, CRP, erosion, enthesitis, dactylitis, BASDAI and DAS28 (CRP). ACR20, American College of Rheumatology 20% improvement; BASDAI, Bath Ankylosing Spondylitis Disease Activity Index; BMI, body mass index; CRP, C reactive protein; DAS28, Disease Activity Score in 28 joints; HAQ-DI, Health Assessment Questionnaire-Disability Index; MTX, methotrexate; SHS, Sharp/van der Heijde score; TNFis, tumour necrosis factor inhibitors.

lack of impact remained regardless of whether abatacept was administered alone or in combination with a non-bDMARD.

In contrast to the results reported here for abatacept, higher BMI negatively impacts the treatment response to TNFi.[2 8 9] In a study of patients with psoriasis and concomitant PsA receiving adalimumab, a significantly lower Psoriasis Area and Severity Index (PASI) 50 response was achieved in patients with BMI ≥30 kg/m^2 (obese) compared with those with a BMI <30 kg/m^2 (non-obese; 58% vs 79%; p=0.02).[8] Similarly, a 24-month observational study of patients with active PsA receiving TNFi found that patients with a BMI ≥30 kg/m^2 (obese) had a significantly higher risk of not achieving a combination of seven selected outcome measures versus patients with a BMI <30 kg/m^2 (non-obese; HR 4.90; 95% CI 3.04 to 7.87).[9]

The difference in the impact of BMI on response to abatacept versus TNF is seen in PsA has also been observed in RA. Previous findings from interventional trials and real-world studies showed that, as reported here for PsA, BMI does not affect retention or the response to abatacept in patients with RA.[5 6 10–12] In contrast, obesity was significantly associated with reduced treatment response to TNFis in a meta-analysis of patients with rheumatic diseases, including RA and PsA, based on multiple outcome measures including European League Against Rheumatism response and a 75% reduction in the PASI score (PASI 75).[2] Indeed, a recent systematic review and meta-analysis of patients with inflammatory diseases concluded that obesity reduced the efficacy of TNFis, but not of abatacept and tocilizumab.[12]

The reasons for the differences in the effects of obesity on response to different bDMARDs are unclear. While factors, such as half-life and volume of distribution, may partly explain these observations, it is also possible that the unique mechanism of action of abatacept might contribute to the lack of impact of BMI on accrued responses. Adipose tissue is a source of specific adipocytokines, such as TNF, macrophage chemoattractant protein-1, plasminogen activator inhibitor-1, interleukin-6, leptin and adiponectin, which are associated with a proinflammatory status in patients with obesity.[13] It could be speculated that the increased levels of proinflammatory adipocytokines, particularly TNF, may affect the treatment response to TNFis,[14 15] whereas the upstream selective modulation of T-cell co-stimulation by abatacept may be more independent and less affected by adipocytokines and, consequently, BMI.

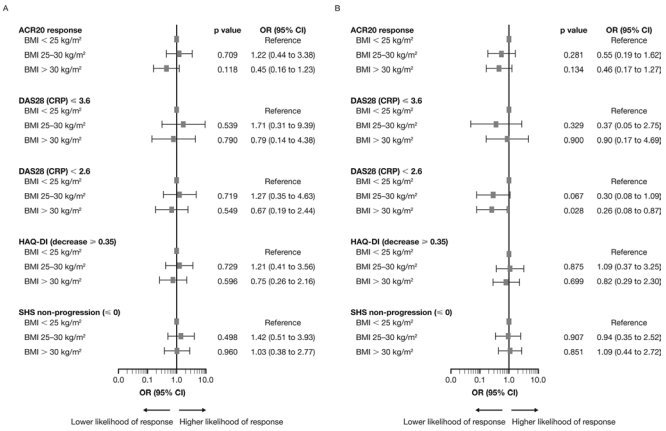

Figure 2 Multivariable model of adjusted* comparisons of treatment response at week 24 between BMI subgroups in patients receiving (A) abatacept and (B) placebo. An OR >1 indicates a higher likelihood of response, while an OR <1 indicates a lower likelihood of response. ORs are significant when 95% CIs do not overlap 1. *The model was adjusted for the following baseline factors: treatment, BMI, MTX use, prior TNFis, CRP, erosion, enthesitis, dactylitis, BASDAI and DAS28 (CRP). ACR20, American College of Rheumatology 20% improvement; BASDAI, Bath Ankylosing Spondylitis Disease Activity Index; BMI, body mass index; CRP, C reactive protein; DAS28, Disease Activity Score in 28 joints; HAQ-DI, Health Assessment Questionnaire-Disability Index; MTX, methotrexate; SHS, Sharp/van der Heijde score; TNFis, tumour necrosis factor inhibitors.

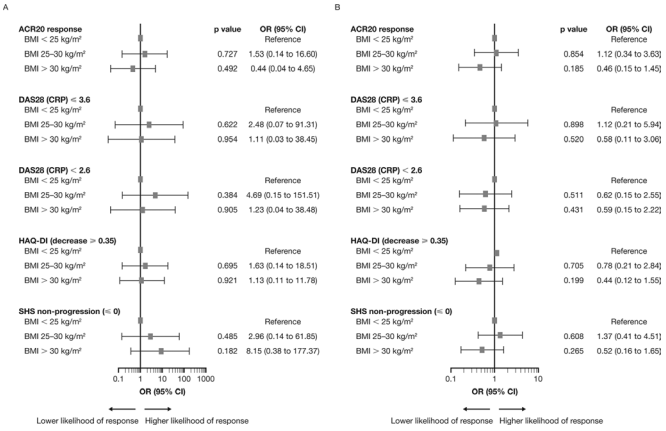

Figure 3 Multivariable model of adjusted* comparisons of treatment response at week 24 between BMI subgroups in patients receiving abatacept (A) without any non-biologic DMARD and (B) in combination with a non-biologic DMARD. An OR >1 indicates a higher likelihood of response, while an OR <1 indicates a lower likelihood of response. ORs are significant when 95% CIs do not overlap 1. *The model was adjusted for the following baseline factors: treatment, BMI, MTX use, prior TNFis, CRP, erosion, enthesitis, dactylitis, BASDAI and DAS28 (CRP). ACR20, American College of Rheumatology 20% improvement; BASDAI, Bath Ankylosing Spondylitis Disease Activity Index; BMI, body mass index; CRP, C reactive protein; DAS28, Disease Activity Score in 28 joints; DMARD, disease-modifying antirheumatic drug; HAQ-DI, Health Assessment Questionnaire-Disability Index; MTX, methotrexate; SHS, Sharp/van der Heijde score; TNFis, tumour necrosis factor inhibitors.

Interestingly, we found that, among patients receiving placebo, obesity was associated with less favourable outcomes for some measures in the obese versus underweight/normal BMI subgroups. This finding is consistent with previous reports showing that obesity is associated with a lower likelihood of achieving sustained minimal disease activity state in patients with PsA receiving none, any or combinations of non-bDMARDs, bDMARDs and NSAIDs.[16] These findings are aligned with the recommendation from the National Psoriasis Foundation for dietary weight reduction among patients with PsA who are overweight or obese based on evidence from a systematic review of 55 studies.[17]

The results reported here should be interpreted after considering the limitations of the current analysis. In addition to the inherent limitations of a *post hoc* analysis, this study included short-term data only—further research is needed to evaluate the long-term impact of BMI on treatment response to abatacept in patients with PsA. The early escape design of the ASTRAEA study represents an additional limitation, as the week 24 data for the placebo arm are from a mixed patient population of early escape patients who received abatacept between weeks 16 and 24 and non-early escape patients who continued to receive placebo.

In summary, the results reported here indicate that BMI does not appear to impact the response to abatacept administered at the approved dose of 125 mg SC weekly in patients with PsA across multiple outcome measures, similar to reported findings in RA.[5 6 10] Given that obesity is common in patients with PsA and negatively impacts treatment response to TNFis,[1 2 8 9] these data suggest that abatacept can be considered as an important treatment option for patients with PsA irrespective of BMI status.

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Contributors All authors contributed to the study conception and design, and interpretation of data. PJM and HAA contributed to the acquisition of data. All authors contributed to drafting the article or revising it critically for important intellectual content. All authors approved the version of the article to be published.

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Competing interests IBM has received grant/research support from Bristol-Myers Squibb, Celgene, Janssen and UCB, and has acted as a consultant to Bristol-Myers Squibb (<US$10 000), AbbVie (<US$10 000), Celgene (<US$10 000), Janssen (<US$10 000), Lilly (<US$10 000), Novartis (<US$10 000), Pfizer (<US$10 000) and UCB (<US$10 000). GF has received grant/research support from Roche, Bristol-Myers Squibb and Pfizer, and has received speaking fees from MSD (<US$10 000), UCB (<US$10 000), Pfizer (<US$10 000), AbbVie

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Patient consent for publication Not required.

Ethics approval The ASTRAEA study was conducted in accordance with the Declaration of Helsinki and was consistent with the International Conference on Harmonisation and Good Clinical Practice. The study protocol, patient informed consent form and any other written materials supplied to the patient received ethics approval from the Schulman Associates Institutional Review Board (SAIRB). IRB organization number: 0000635; IRB registration number: 00000971. SAIRB did not provide approval numbers for each site.

Provenance and peer review Not commissioned; externally peer reviewed.

Data availability statement Data are available on reasonable request.

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