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Letter to the Editor

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Letter to the Editor (Other)

The influence of deprivation in the outcomes of psoriatic arthritis within the UK—utilizing Outcomes of Treatment in Psoriatic Arthritis Study Syndicate (OUTPASS) data

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Key message

- Deprivation does not influence the time to initiate biologics or their efficacy in psoriatic arthritis.

Dear Editor, Psoriatic arthritis (PsA) affects up to 30% of patients with psoriasis and is associated with a high degree of morbidity, including decreased work capacity, social participation and limitations in physical function [1]. The introduction of the biologic DMARDs (bDMARDs) revolutionized treatment strategies for patients with PsA, however, none are universally effective [2, 3]. Although the reasons for this are numerous and complex, one potential factor is that of deprivation and its associated environmental factors that may affect treatment response. Interestingly, it has been reported that higher levels of education, a key determinant of deprivation, is associated with a lower incidence of PsA [4, 5], but the association with treatment response has not yet been explored. We aimed to assess the impact of deprivation on access to biologic treatment and the clinical response to these medications.

Data were used from the Outcomes of Treatment in Psoriatic Arthritis Study Syndicate (OUTPASS REC ref: 13/NW/0068) study, a national UK prospective, observational study of PsA patients commencing a targeted synthetic or biologic DMARD [6]. Patients are seen at baseline (pre-bDMARD) and 3, 6 and 12 months. Clinical data recorded include 28 and 66/68 swollen and tender joint count, patient global visual analogue scale (0–100 mm), physician and patient global assessment (0–5) and CRP/ESR. Deprivation was measured by the Index of Multiple Deprivation (IMD), the official measure of relative deprivation in England utilizing seven measures. It ranks every small area in England from 1 (most deprived area) to 32 844 (least deprived area). Deprivation was categorized into three groups to better represent the degree of deprivation (20% most deprived, 40% middle deprived, 40% least deprived). Response to treatment was measured using changes in the 28-joint DAS (DAS28) or PsA response criteria (PsARC) at 3 months. The change in DAS28 between the IMD groups was assessed using multivariable linear regression, adjusted for baseline DAS28, while PsARC changes over time were analysed using multivariable logistic regression.

Of a cohort size of 635, 538 (85%) were eligible for analysis after excluding those with missing baseline, IMD or 3-month response data. Baseline characteristics and clinical response are presented in Table 1. There was no difference in the distribution of patients between the three deprivation groups (Table 1). Those in the 20% most deprived group had an increased risk of depression with an odds ratio (OR) of 1.63 (95% CI 1.07, 2.52; $P = 0.02$).

There was no statistically significant difference in the time from diagnosis to starting biologics between the most deprived 20% and the 40% middle deprived [$\beta = -0.52$ (95% CI −2.60, 1.55), $P = 0.62$] or the 40% least deprived [$\beta = 0.86$ (−1.23, 2.94), $P = 0.42$]. There was no statistically significant difference in treatment responses between the deprivation groups. The DAS28 change at 3 months remained similar between the most deprived 20%, the 40% middle deprived [$\beta = -0.091$ (95% CI −0.48, 0.29), $P = 0.64$] and least deprived 40% groups [$\beta = -0.048$ (95% CI −0.43, 0.33), $P = 0.80$].

The PsARC change over 3 months was also analysed and demonstrated no statistically significant difference between the most deprived 20%, the 40% middle [OR 1.86 (95% CI

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Table 1. Baseline characteristics, treatment response and time to bDMARD compared across groups of socio-economic deprivation in the OUTPASS cohort

n	All participants 538	20% most deprived 138	40% middle deprivation 207	40% least deprived 193	Missingness, *n* (%)
Age started biologic, mean (S.D.)	51.58 (1.86)	51.71 (1.79)	51.42 (1.85)	51.39 (1.88)	95 (17.66)
Female, *n* (%)	306 (56.89)	87 (63.04)	109 (52.66))	110 (56.99)	10 (1.86)
White ethnicity, *n* (%)	411(98.33)	104 (99.05)	159 (98.15)	148 (98.01)	120 (22.30)
Baseline DAS28, mean (S.D.)	4.94 (1.03)	4.93 (0.09)	4.97 (0.08)	4.92 (0.09)	42 (7.81)
CASPAR criteria met, *n* (%)	393 (79.72)	99 (82.5)	150 (78.13)	144 (79.56)	59 (10.12)
bDMARD, %					28 (5.20)
Etanercept	33.33	32.28	32.99	34.4	
Adalimumab	34.51	31.50	36.55	34.4	
Golimumab	6.27	7.09	8.12	3.76	
Certolizumab	4.12	4.72	4.57	3.23	
Ustekinumab	6.86	8.66	4.56	8.06	
On methotrexate at baseline, %	46.20	42.52	52.76	41.71	25 (4.65)
No comorbidities, %	166 (32.49)	37 (29.37)	55 (27.64)	74 (39.78)	67[a]
1 comorbidity, %	162 (31.70)	37 (29.37)	74 (37.19)	51 (27.42)	
2 comorbidities, %	101 (19.77)	29 (23.02)	37 (18.59)	35 (18.82)	
≥3 comorbidities, %	79 (15.46)	23 (18.25)	33 (16.58)	26 (13.98)	
DAS28 response at 3 months, mean (S.D.)	1.67 (1.32)	1.74 (1.35)	1.66 (1.27)	1.69 (1.38)	240 (44.60)
PsARC improvement at 3 months, %[b]	76.40	68.75	80.34	77.12	239 (44.42)
Months from diagnosis to biologic, mean (S.D.)	8.09 (8.95)	8.03 (9.60)	7.51 (8.00)	8.89 (9.64)	52 (9.66)

[a] All comorbidity data missing.
[b] PsARC was measured as a binary outcome of improved or non-responder. To achieve response, a patient has to achieve two of the following, one of which has to be a tender (68) and swollen (66) joint count, and no worsening of any measure: tender or swollen joint count improvement of ≥30% and/or patient global or physician global improvement of at least 1 point on a 5-point Likert scale.

0.92, 3.73), $P = 0.082$] and least deprived 40% [OR 1.53 (95% CI 0.77, 3.02), $P = 0.22$].

This study demonstrated that in this cohort there is equal inclusion of patients across all degrees of deprivation, demonstrating no significant recruitment bias. There was no difference in time from diagnosis to starting bDMARD therapy or response to treatment across the deprivation spectrum.

There are very little data exploring the effect of deprivation on disease response to biologics in PsA, but we have found it not to be a significant factor in determining baseline disease, accessing biologic therapies or the response to them in a cohort of 538 patients.

These findings suggest that the National Health Service in England is performing its intended role of providing care according to need and regardless of circumstance. However, it is important to note that there is a complex relationship between low socio-economic background and disease outcomes. This study assesses the impact of deprivation in the context of a health service that provides care free at the point of delivery and based on need, and so it cannot be applied to other healthcare systems where deprivation may impact access to healthcare. A further limitation is that the sample size was relatively small and lacked power in certain variables with missing data. Further research is required to explore the relationship between deprivation and disease outcomes in PsA.

Supplementary material

Supplementary material is available at *Rheumatology Advances in Practice* online.

Data availability

The data that support the findings of this study are available upon reasonable request to the corresponding author. The data are not publicly available due to privacy/ethical restrictions.

Authors' contributions

OUTPASS collaborators were involved in participant recruitment and follow-up visits. J.B., A.B. and M.L. were involved in study conception. J.B., S.S.Z. and M.L. were involved in study analysis and drafting the work. All authors critically revised the article for intellectual content, read and approved the article.

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References

1. Ogdie A, Coates LC, Gladman DD. Treatment guidelines in psoriatic arthritis. Rheumatology (Oxford) 2021;59(Suppl 1):i37–46.
2. Makos A, Kuiper JH, Kehoe O *et al*. Psoriatic arthritis: review of potential biomarkers predicting response to TNF inhibitors. Inflammopharmacology 2023;31:77–87.
3. Magee C, Jethwa H, FitzGerald OM *et al*. Biomarkers predictive of treatment response in psoriasis and psoriatic arthritis: a systematic review. Ther Adv Musculoskelet Dis 2021;13:1759720X211014010.
4. Kerola AM, Sexton J, Wibetoe G *et al*. Incidence, sociodemographic factors and treatment penetration of rheumatoid arthritis and psoriatic arthritis in Norway. Semin Arthritis Rheum 2021;51:1081–8.
5. Eder L, Haddad A, Rosen CF *et al*. The incidence and risk factors for psoriatic arthritis in patients with psoriasis: a prospective cohort study. Arthritis Rheumatol 2016;68:915–23. doi:10.1002/art.39494
6. Jani M, Chinoy H, Barton A; for OUTPASS. Association of pharmacological biomarkers with treatment response and longterm disability in patients with psoriatic arthritis: results from OUTPASS. J Rheumatol 2020;47:1204–8.