Comparative Analysis of Disease Activity Measures, Use of Biologic Agents, Body Mass Index, Radiographic Features, and Bone Density in Psoriatic Arthritis and Rheumatoid Arthritis Patients Followed in a Large U.S. Disease Registry

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ABSTRACT. ***Objective.*** To compare disease activity, radiographic features, and bone density in psoriatic arthritis (PsA) and rheumatoid arthritis (RA) matched cohorts.

Methods. Disease activity and radiographic data in the Consortium of Rheumatology Researchers of North America database from 2001 to 2008 were compared for 2481 patients with PsA and 17,107 patients with RA subsequently matched for age, gender, and disease duration. Radiographic outcomes included presence of erosions, and joint deformity. In addition, bone mineral density (BMD) scores for lumbar spine (L-spine) and femoral neck were compared using the same matching criteria plus weight and smoking status.

Results. Tender (4.5 vs 3.4, $p < 0.001$) and swollen (4.4 vs 2.9, $p < 0.012$) joint counts, and modified Health Assessment Questionnaire scores were significantly higher (0.4 vs 0.3, $p < 0.001$) in patients with RA compared with patients with PsA. Patient general health and pain scores were also higher in patients with RA vs patients with PsA. Joint erosions (47.4% vs 37.6%, $p = 0.020$) and deformity (25.2% vs 21.6%, $p = 0.021$) were more prevalent in RA than PsA. In multivariate analysis, a reduced prevalence of erosions in PsA vs RA was noted (OR 0.609, $p < 0.001$). After matching, T-scores for L-spine (–0.54 vs –0.36, $p = 0.077$) and femoral neck (–0.88 vs –0.93, $p = 0.643$) were similar in patients with RA and patients with PsA, although body weight was a major confounder.

Conclusion. The level of disease activity and radiographic damage was significantly higher for RA vs PsA subjects, although the magnitude of differences was relatively small. BMD levels were comparable between cohorts. Outcomes in patients with PsA and patients with RA may be more similar than previously reported. (J Rheumatol First Release Sept 15 2010; doi:3899/jrheum.100483)

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Rheumatoid arthritis (RA) and psoriatic arthritis (PsA) are inflammatory arthritides with distinct clinical features, but destruction of bone and cartilage is observed in both disorders. PsA has been considered a milder disease compared to RA and this perception was associated with less aggressive treatment of this condition in the past[1]. Recent studies, however, have shown that up to half of patients with PsA manifest radiographic joint erosions 2 years following the initial diagnosis, which indicates that joint damage in PsA can be quite severe, even early in the disease course[2,3].

In RA, 3 major forms of bone loss have been identified: focal bone loss that presents as erosions, periarticular bone loss, and generalized bone loss that presents as osteopenia or osteoporosis (OP)[4,5]. Bone erosions are a prominent feature in PsA as well, but the perception is that in PsA, bone ero-

sions are not as prevalent. Psoriatic erosions tend to be asymmetric, large, and eccentric compared to RA[6]. The presence of periarticular bone loss was not thought to be a prominent finding in PsA although one study reported periarticular osteopenia in PsA radiographs[7]. Systemic bone loss, manifest as generalized osteopenia or OP, has not been well studied in PsA but 2 reports found decreased bone mineral density (BMD) in PsA subjects[8,9]. In contrast, Nolla, *et al* found no evidence of BMD loss in patients with PsA compared to control patients[10].

Two studies compared radiographic joint changes in RA and PsA with conflicting results. Rahman, *et al* examined joint radiographs in a small cohort of 42 RA and PsA patients matched for age, sex, and disease duration[11]. No significant difference in the modified Steinbrocker scores was observed in the PsA and RA subjects. In addition, the number of joints with significant radiographic damage was comparable in the 2 groups. Sokoll, *et al* evaluated 47 PsA and RA patients matched for disease duration and secondarily for age and gender and showed that the RA cohort had significantly more damage by Larsen scores on radiographs compared to the PsA cohort (median hand score 39 vs 8; foot score 11 vs 4)[12]. The Larsen score, used in the Sokoll study, does not record distal interphalangeal (DIP) involvement, a feature more common in the PsA cohort, and the omission of DIP joints may have contributed to the differences in these 2 studies.

While generalized and periarticular bone loss have been studied extensively in RA, there is a paucity of data on patients with PsA. One study of early PsA and RA patients found no differences in periarticular hand bone loss as measured by dual energy x-ray absorptiometry (DEXA)[7]. A small study of BMD in patients with severe chronic plaque psoriasis compared to controls matched for age and body mass index (BMI) found no difference in mean hip and lumbar spine (L-spine) Z-scores[13]. When patients with PsA were compared to patients with psoriasis who did not have arthritis, significantly lower L-spine Z-scores were noted in those subjects with joint disease[13]. In another study, Frediani, *et al* compared 186 patients with PsA to 100 healthy controls and found that BMD was significantly lower at the L-spine, femur, and total body in the patients with PsA compared to controls[8]. To date, no studies comparing generalized BMD between patients with RA and patients with PsA have been published.

Taken together, the evidence suggests that both focal bone loss (erosions) and generalized bone loss can be extensive in PsA. Thus, we hypothesized that the extent of erosive joint damage and systemic bone loss would not be significantly different between patients with RA and patients with PsA. To test this hypothesis, we compared disease activity, radiographic features, and BMD in patients with PsA and patients with RA matched for age, gender, and disease duration enrolled in the Consortium of Rheumatology Researchers of North America (CORRONA) database.

MATERIALS AND METHODS

Data sources and collection. A total of 17,107 patients with RA and 2481 patients with PsA identified in the CORRONA registry from October 2001 to May 2008 were included in the study. These patients were treated by 160 rheumatologists at 82 private and 20 academic rheumatology practices in the United States. The CORRONA registry is a prospective disease registry of patients with RA and PsA confirmed by the physician. Patients are followed longitudinally, and patient and physician-derived information is recorded approximately every 3 to 6 months at routine followup visits, and the data are entered into a secure website. The characteristics of the CORRONA registry have been described[14,15]. The CORRONA registry was approved for use by the respective institutional review boards for academic sites and a central review board for private practice sites.

Study population. Patient demographics and clinical characteristics were assessed among the entire study population at the entry of the CORRONA registry. Data evaluated include age, gender, duration of disease, rheumatoid factor status, BMI, smoking status, college education status, and medication use [disease-modifying antirheumatic drugs (DMARD), prednisone, biologics, estrogen].

Disease activity study populations. The study population for disease activity measures included all available patients with RA and PsA. Disease activity measures evaluated included tender and swollen joint counts, erythrocyte sedimentation rate (ESR), and C-reactive protein (CRP) levels, physician-evaluated disease activity by visual analog scale (VAS), patient general health by VAS, patient pain scores by VAS, and modified Health Assessment Questionnaire (MHAQ) disability index. Subsequently the patients with RA and the patients with PsA were matched on gender, age, and disease duration for each individual disease activity measure to create its corresponding matched cohorts. Matching was carried out without replacement. Multivariate analysis for disease activity measures was performed on 1646 patients from 823 matched pairs that had all variables available.

Radiograph study population. The study population for radiographic assessments of joint erosions and deformities included 11,734 patients with RA and 1574 patients with PsA who had any radiographic information available. The patients' first visit recorded in the database with radiographic information was used in the analyses. Radiographic outcomes were obtained from the physician questionnaire and included presence or absence of erosions and radiographic deformity as determined by the physician's reading of the radiographic films or reports. Subsequently these patients were matched on gender, age, and disease duration for every radiographic assessment to create the corresponding matched cohorts. The number of available patients for each radiographic assessment varied because of incomplete data. Matching was carried out without replacement. Multivariate analysis for radiographic evaluations was performed on 2940 patients from 1470 matched cohorts (matched patients) that had all variables available.

Bone density study population. The study population for bone density assessment consisted of patients with RA and PsA identified in the database who had DEXA information with at least 1 T score available. T scores for L-spine and femoral neck were obtained from the physician questionnaires and BMD (g/cm^2) when available. DEXA data from the first visit when the measures became available were used in the analyses. Patients were then matched for age, gender, duration of disease, BMI, and smoking status, for each of the areas. Matching was carried out without replacement. Multivariate generalized linear regression analyses were performed for L-spine and femoral neck on matched cohorts adjusting for prednisone and estrogen use. Because of incomplete DEXA information, the numbers of patients available for assessments of each analysis varied.

Statistical analysis. Categorical variables were summarized as frequencies and compared for the difference among PsA and RA patients using the chi-square test. Means and SD were reported for the continuous variables. The equality of variances of these variables among patients with PsA and patients with RA was examined. If the test results showed that the assump-

tion of equal variances was reasonable, the group means were compared using pooled t tests. Otherwise Satterthwaite tests were implemented. Cohorts were matched without replacement for independent variables. Indicators of erosive disease, joint space narrowing, and joint deformity were modeled using conditional logistic regressions to assess the difference among patients with PsA and patients with RA. The generalized estimating equation (GEE) approach was used to examine the difference in the continuous outcome variables between the 2 cohorts. A stepwise matching procedure was adapted to investigate a set of potential confounders differentiating the outcome variables among patients with PsA and patients with RA. All analyses were carried out using SAS 9.1 (SAS Institute Inc., Cary, NC, USA).

RESULTS

Clinical characteristics. The demographic and clinical characteristics of the entire unmatched study populations are shown in Table 1. Several differences between the 2 unmatched groups were noted. Patients with PsA were, on average, 7 years younger (51.9 vs 58.5, $p < 0.001$), and were less likely to be female (52.0% vs 75.5%, $p < 0.001$) than patients with RA. A notable finding was that patients with PsA had a significantly higher BMI (32.1 vs 29.8, $p < 0.001$) than patients with RA. Patients with PsA were also less likely to be current smokers compared to patients with RA (14.0 vs 16.3%, $p = 0.004$) and more likely to have a negative rheumatoid factor (11.6 vs 71%, $p < 0.001$). Patients with PsA were more likely to have a college education (63.0% vs 51.0%, $p < 0.001$). Rates of DMARD and prednisone use were also higher in patients with RA compared to those with PsA. Biologic drug use, predominantly in the form of tumor necrosis factor (TNF) inhibitors, was higher in patients with PsA compared to patients with RA (51.3% vs 43.6%, $p < 0.001$).

Disease activity measures. In the radiograph assessment study cohort, matched for age, gender, and disease duration (Table 2A), there were more tender (4.5 vs 3.4, $p < 0.001$), more swollen joints (4.5 vs 2.9, $p < 0.001$) using 28-joint count, higher physician VAS disease activity score (36 mm vs 31 mm, $p < 0.001$), and worse MHAQ disability scores (0.4 vs 0.3, $p < 0.001$) in patients with RA compared to patients with PsA. Patient-reported outcomes of general health assessment and pain were also higher in patients with RA compared to patients with PsA.

In the GEE analysis of the study population (n = 1646) matched for age, gender, and disease duration (Table 2B), a reduced risk was found only for the prevalence of higher swollen joint count (OR 0.925, $p < 0.001$), and ESR (0. 981, $p < 0.001$) in PsA compared to RA.

Radiographic assessment. Patients with RA had a higher prevalence of joint erosions than PsA-matched cohorts (47.4% vs 37.6%, $p < 0.001$; Table 3A) and a higher prevalence of radiographic deformity (25.2% vs 21.6%, $p = 0.021$), but the absolute differences were not large (10.0% and 4%, respectively).

In the conditional logistic regression analysis of the entire study population matched for age, gender, and disease duration (Table 3B), a reduced risk was found only for the prevalence of erosions in PsA compared to RA (OR 0.609, $p < 0.001$). No significant differences were noted in the risk of radiographic deformity in multivariate analyses.

Bone density assessments. L-spine T scores and femoral neck T scores were significantly lower for patients with RA in the unmatched cohorts (Table 4). The same trends remained for cohorts matched for age, gender, disease duration, and smoking status. When cohorts were matched for the additional variable BMI, the differences in L-spine T scores and femoral neck T scores were no longer significant, after adjusting for prednisone and estrogen use.

DISCUSSION

In our study of patients with PsA and RA enrolled in a longitudinal database, we identified 4 major findings. First, disease activity measures and radiographic outcomes were of comparable severity in PsA and RA. The RA cohort demonstrated a statistically significantly greater number of swollen and tender joints, but the magnitude of these clinical differences was quite small. The prevalence of erosions and radiographic deformity was significantly higher in patients with

Table 1. Clinical characteristics of the unmatched study population.

Characteristics	Psoriatic Arthritis, n = 2481	Rheumatoid Arthritis, n = 17,107	p
Age, yrs, mean (SD)	51.9 (12.8), n = 2470	58.5 (13.6), n = 17,009	< 0.001
Disease duration, yrs, mean (SD)	8.5 (8.9), n = 2451	9.6 (9.8), n = 16,963	< 0.001
Body mass index, mean (SD)	32.1 (8.3), n = 2346	29.8 (7.9), n = 15,827	< 0.001
Female, %	52.0, n = 2477	75.5, n = 17,081	< 0.001
RF positive, %	11.6, n = 773	71.0, n = 9431	< 0.001
Smoking status, %	14.0, n = 2418	16.3, n = 16,603	0.004
College education, %	63.0, n = 2354	51.0, n = 16,097	< 0.001
Any DMARD use, %	86.7, n = 2481	90.4, n = 17,107	< 0.001
Prednisone use, %	14.6, n = 2470	36.9, n = 16,917	< 0.001
Biologic use, %	51.3, n = 2481	43.6, n = 17,107	< 0.001
Estrogen use, %	4.2, n = 2481	5.9, n = 17,107	< 0.001

DMARD: disease-modifying antirheumatic drug; RF: rheumatoid factor.

Table 2A. Disease activity measures in psoriatic arthritis and rheumatoid arthritis in unmatched and matched cohorts for age, gender, and duration of disease. Values are mean (SD).

Measures	Unmatched Cohorts			Matched Cohorts		
	PsA	RA	p	PsA	RA	p
28 Tender joint count	3.3 (5.0), n = 2446	4.3 (5.9), n = 16,918	< 0.001	3.4 (5.0), n = 2357	4.5 (6.1), n = 2357	< 0.001
28 Swollen joint count	2.9 (4.4), n = 2444	4.8 (5.9), n = 16,931	< 0.001	2.9 (4.2), n = 2355	4.5 (5.2), n = 2355	< 0.001
ESR, mm/h	16.9 (16.3), n = 964	25.6 (22.7), n = 7514	< 0.001	17.0 (16.3), n = 925	22.4 (21.9), n = 925	< 0.001
CRP	2.1 (2.6), n = 365	2.3 (4.1), n = 2512	0.123	2.1 (2.5), n = 334	2.4 (3.0), n = 334	0.162
Physician VAS disease activity, 0–100 mm	31 (15), n = 880	37 (16), n = 6943	< 0.001	31 (15), n = 845	36 (17), n = 845	< 0.001
Patient VAS general health, 0–100 mm	29.3 (25.1), n = 2312	31.0 (26.2), n = 15,905	0.002	28.3 (29.3), n = 2227	32.2 (31.3), n = 2227	0.010
Patient VAS pain, 0–100 mm	31.4 (25.6), n = 2304	33.3 (26.6), n = 15,998	< 0.001	31.4 (25.8), n = 2223	33.0 (27.1), n = 2223	0.041
MHAQ Disability Index, 0–3	0.3 (0.4), n = 2360	0.4 (0.5), n = 16,440	< 0.001	0.3 (0.4), n = 2273	0.4 (0.5), n = 2273	< 0.001

PsA: psoriatic arthritis; RA: rheumatoid arthritis; ESR: erythrocyte sedimentation rate; CRP: C-reactive protein; VAS: visual analog scale; MHAQ: Modified Health Assessment Questionnaire.

Table 2B. Multivariate logistic regression for psoriatic arthritis vs rheumatoid arthritis in 823 matched cohorts (n = 1646 total patients).

Measures	OR	Confidence Limits		Probability chi-square
28 Tender joint count	0.985	0.950	1.021	0.415
28 Swollen joint count	0.925	0.897	0.954	< 0.001
Erythrocyte sedimentation rate, mm/h	0.981	0.972	0.989	< 0.001
Physician VAS disease activity, 0–100 mm	1.147	0.945	1.391	0.164
Patient VAS general health, 0–100 mm	1.001	0.993	1.009	0.813
Patient VAS pain, 0–100 mm	1.002	0.995	1.009	0.581
MHAQ Disability Index, 0–3	0.767	0.556	1.058	0.106

VAS: visual analog scale; MHAQ: Modified Health Assessment Questionnaire.

Table 3A. Radiographic outcomes in psoriatic arthritis (PsA) and rheumatoid arthritis (RA) in unmatched and matched cohorts for age, gender, and duration of disease. Values are percentages.

Outcomes	Unmatched Cohorts			Matched Cohorts		
	PsA	RA	p	PsA	RA	p
Presence of erosions	37.7 (n = 1574)	49.2 (n = 11,582)	< 0.001	37.6 (n = 1520)	47.4 (n = 1520)	< 0.001
Presence of radiographic deformity	21.8 (n = 1532)	26.8 (n = 11,199)	< 0.001	21.6 (n = 1476)	25.2 (n = 1474)	0.021

Table 3B. Multivariate logistic regression for psoriatic arthritis (PsA) vs rheumatoid arthritis (RA) in 1470 matched cohorts (2940 total patients).

Outcomes	OR	Confidence Limits		Probability chi-square
Erosions	0.609	0.511	0.725	< 0.001
Radiographic deformity	1.192	0.967	1.469	0.101

Table 4. Bone density measured by T scores in psoriatic arthritis (PsA) and rheumatoid arthritis (RA) in unmatched and matched cohorts for age, gender, duration of disease, smoking status, and body mass index, after adjusting for prednisone and estrogen use. Values are percentages.

Areas Measured	Unmatched Cohorts			Matched Cohorts		
	PsA	RA	p	PsA	RA	p
Lumbar spine	–0.37 (1.52), n = 530	–0.76 (1.52), n = 7166	< 0.001	–0.36 (1.53), n = 457	–0.54 (1.56), n = 457	0.182
Femoral neck	–0.91 (1.21), n = 336	–1.25 (1.28), n = 4096	< 0.001	–0.93 (1.21), n = 273	–0.88 (1.28), n = 273	0.388

RA compared to patients with PsA, but again the absolute differences were small. Second, bone density was similar in patients with PsA and patients with RA who were matched for weight and smoking status. These findings support the concept that inflammation and bone loss in PsA and RA are of similar magnitude and extent.

The third major finding was that the overall incidence of the use of biologic agents in this large U.S. cohort was 51.3% and 43.6%, respectively, in patients with PsA and RA. We believe that this is the first report derived from a large U.S. population that allows us to compare the prevalence of the use of biologic agents in these 2 different rheumatologic inflammatory diseases. Finally, patients with PsA displayed a significantly higher BMI than matched cohorts with RA. To our knowledge, this is the first report to describe these very significant differences in a very large population of U.S. patients.

Patients from both cohorts were quite similar with respect to reported symptoms. For example, the 2 groups had similar numerical although statistically different results in the patient-derived outcome measures of global health and pain as measured by VAS, previously well-validated techniques for assessment of disease activity. The minimal clinically important difference (MCID) for patient pain measured by VAS in RA has been reported to be between 0.5 and 1.1 on a 0–10 scale[16]. The absolute difference between the RA and PsA-matched patients was only 0.16 on a converted 0–10 scale (equivalent to a 1.6 absolute difference on a 0–100 scale as reported in Table 2A)[16]. The MHAQ (range 0–3) difference of 0.1 was statistically higher in patients with RA than in patients with PsA, but this finding is unlikely to be clinically significant because psychometric assessment of the HAQ in PsA revealed that the MCID of this measure is 0.3[17,18]. The similarities in patients' disease assessment, particularly in regards to patient global health assessment, despite higher joint counts in RA, may reflect a contribution from other disease domains in PsA, such as enthesitis, dactylitis, axial disease, or skin lesions of psoriasis.

The finding of higher tender and swollen joint counts in RA subjects may have been in part related to the instrument applied to assess these domains. The 28-joint count used in this database may underestimate the extent of joint involvement in PsA given its exclusion of hand DIP joints and all the joints of the feet, which are more commonly affected in patients with PsA than in patients with RA. The low tender and swollen joint scores in both groups were not surprising given the very high prevalence of the use of biologic agents in the U.S. compared to other European registries[19]. As has been described, registry patients display a lower number of tender and swollen joints vs those enrolled in clinical trials with tight inclusion and exclusion criteria[20]. As noted, the rate of DMARD use was slightly higher in the RA group, while biologic use was higher in the PsA group. The similarities in acute-phase reactants (no difference in CRP levels and slightly higher ESR levels in RA compared to PsA groups) observed in the 2 cohorts may reflect similar treatment responses, although it is difficult to quantify the effect of therapy in a retrospective manner for these comparisons.

The radiographic data provide further support for the similarities between the 2 diseases. The prevalence of erosions was 9.8% higher and radiographic deformity was 3.6% higher in RA subjects. High rates of DMARD and biologic use in these cohorts may explain the similarities observed in the 2 groups. In addition, radiographs were obtained at the discretion of the treating physician, which may have favored selection of radiographs of patients with more severe RA and PsA. Patients with PsA also have other radiographic features, such as periostitis and productive bone formation, which may contribute to overall disease severity and disability, but which are not captured with this method.

The BMD data also shed light on the similarities between the 2 groups. On initial assessment, the patients with RA had lower BMD scores, even after adjusting for prednisone and estrogen use, but these differences were no longer present when the subjects were matched for weight. These findings strongly suggest that both diseases have a similar impact on BMD, and that aggressive screening for generalized OP should occur in both groups. The relationship between obesity and high BMI is well established as a negative predictor of OP risk[21,22]. Future studies of BMD in PsA should adjust for BMI differences among comparator groups. Our current study was limited by the small number of matched cohorts because of the relatively small number of patients with PsA who had BMD information available compared to patients with RA.

We were again struck by the very significant differences in BMI observed in the 2 cohorts. This finding is consistent with emerging evidence that psoriasis patients tend to be obese but this is the first study to compare weight from matched RA and PsA patients from a large registry. The role of obesity in psoriasis remains controversial although it has been suggested that inflammation plays a role in both conditions[23]. A recent study identified obesity as a major risk factor for incident psoriasis[24]. In addition, case reports recorded remission of psoriasis after gastric bypass surgery, suggesting that obesity plays a role in persistence of skin inflammation[25,26]. The links between obesity and psoriasis have not been elucidated, but inflammatory macrophage subsets, stimulation of T cell subsets, and leptin activation are all under active investigation[23].

The strengths of this registry study include a well-characterized large population of patients with PsA and patients with RA matched for important variables. The study used a large cohort with the ability to match using standardized data collection across 2 disease states.

The major limitation of this registry study is the lack of numeric scoring of radiographs to determine the extent of radiographic involvement and the inability to compare the severity of erosive change across the 2 groups. As is usually the case in registry-derived data as opposed to randomized controlled trials, only data on the presence or absence of erosions and radiographic deformity were available as determined by the physician's reading of the radiographs or

reports. It is not possible to account for the effect of radiographic features of PsA that are not captured with this method or for differences in the severity of erosions or deformity among the 2 groups. Interobserver variability cannot be controlled for in this registry study. In addition, patients were taking different therapeutic agents and entered the database at various stages of disease and treatment. The CORRONA database is not an inception cohort, and as a result clinical information of disease status prior to initiating their present DMARD or biologic agent was not available on many patients who entered the registry with established treatment regimens (prevalent cases). A potential bias of our study is that the lack of differences seen in the 2 cohorts may reflect selection bias for patients with more severe disease and treatment effects, and not differences in the disease processes themselves. It is useful to note that the same sites were enrolling both patients with RA and patients with PsA, but a bias toward patients with more severe disease in both groups is possible. Of note, we observed a different prevalence of use of biologic agents in the 2 populations of 51.3% vs 43.6% for PsA and RA, respectively. It is possible that this difference could have contributed to the overall lesser degree of disease activity observed.

Osteoclasts mediate both focal bone erosion and OP in RA[27]. TNF-α and interleukin 1 are key mediators in the pathogenesis of synovitis in both RA and PsA and play a key role in the regulation of bone resorption through potentiation of the osteoclastogenic effects of receptor activator of nuclear factor-κB (RANK)[27,28,29]. Similarities in the 2 groups in regard to bone erosions and OP may reflect both systemic and local activation of the RANK ligand pathway. Some investigators have suggested that distinct osteoclast-related mechanisms mediate generalized vs periarticular bone loss. The existence of these 2 pathways stemmed from studies that showed TNF-α blockade halted bone loss at the hip but not periarticular bone loss at the hand[30]. Lastly, the diffuse bone marrow edema observed in some patients with PsA supports osteitis as a dominant feature rather than synovial inflammation — the predominant finding in the rheumatoid joint[31]. Thus, it seems likely that RA and PsA share key inflammatory and osteoclastogenic pathways leading to OP, but subtle differences in these pathways likely determine disease-specific patterns of bone erosions, generalized OP, and periarticular osteopenia.

Our study demonstrates that the severity of PsA is more similar to RA than previously believed, especially with respect to BMD. A fascinating finding was the large difference in BMI noted between patients with PsA and patients with RA, even after correction for differences in gender. While it is clear that obesity contributes significant comorbidity and must be adjusted for when comparing other variables, the possibility that adipose tissue may exert antiinflammatory and antiosteoclastogenic actions must also be considered. Finally, this is the first study to compare the overall incidence of biologic use in patients in the United States with RA and PsA. We were struck by the very high prevalence of use of biologic agents in these cohorts. Additional prospective studies are needed to further delineate the similarities and differences between these 2 groups and to increase our understanding of both systemic and localized bone loss in PsA and RA.

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