# Lung involvement in psoriatic arthritis patients: cross-sectional study on prevalence, clinical characteristics, and risk factors of a neglected comorbidity

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## Abstract

### Objective

*We aimed to assess lung involvement in patients with psoriatic arthritis (PsA), hypothesising that lung diseases (LDs) represent a distinct comorbidity of PsA and that the association between these two conditions is not merely coincidental.*

### Methods

*Cross-sectional study of 322 patients with a diagnosis of PsA from three Italian centres. Each patient underwent a chest high-resolution CT prior to the start of a biological DMARDs. After pneumologists' evaluation, patients were divided into three groups [chronic obstructive pulmonary disease (COPD), interstitial lung disease (ILD), and non-LD]. Univariate and multivariate statistical analysis were performed to analyse demographic and clinical characteristics of the total cohort and to compare disease characteristics between PsA patients with and without lung involvement.*

### Results

*COPD was diagnosed in 39 patients (12.1%) and ILD in 27 patients (8.4%). The inflammatory burden [DAPSA>14 (OR 4.3, p.adj=0.03), swollen joint count (SJC)>3 (OR 3.2, p.adj=0.02), PASI>10 (OR 3.6, p.adj=0.008), BMI ≥30 (OR 3, p.adj=0.04)] emerged as the most important risk factor for COPD development in PsA patients. This association was weaker in patients with ILD and PsA, since just SJC emerged as a risk factor for ILD development [SJC>3 (OR 3, p.adj=0.04)]. Old age (≥65 years) increased the risk of COPD by up to 9.6 times (p.adj=0.004) and ILD by up to 7.6 times (p.adj=0.04).*

### Conclusion

*Our data support the hypothesis of a correlation between PsA and LDs highlighting that non-traditional risk factors may contribute to the development of LDs in PsA patients, especially in cases of uncontrolled, active articular disease.*

**Key words**

psoriatic arthritis, lung disease, comorbidity, chronic obstructive pulmonary disease, interstitial lung disease.

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## Introduction

Psoriatic arthritis (PsA) is a chronic inflammatory systemic disease characterised by both articular and extra-articular manifestations associated with a high burden of comorbidities. Treatment recommendations from the Group for Research and Assessment of Psoriasis and Psoriatic Arthritis (GRAPPA) identify four main prevalent domains of the disease: peripheral arthritis, axial disease, enthesitis, and dactylitis, as well as extra-articular manifestations, such as onychopathy and psoriasis (PsO), and two related conditions, inflammatory bowel disease (IBD) and non-infectious uveitis (NIU) (1). The combination of these patterns defines the clinical phenotype and guides treatment management (2). Despite the potential to achieve disease remission with the introduction of biologic and targeted synthetic drugs, comorbidities such as lung involvement may still impact clinical outcomes. Respiratory assessment is not routinely performed in PsA patients, even though lung involvement, reported in 7% to 12% of cases, emerged in the literature years ago (3, 4). In this cross-sectional study, we aimed to assess lung involvement as a comorbidity in PsA patients and identify associated risk factors.

## Materials and methods

*Study design*

The study was a cross-sectional analysis of three cohorts of consecutive PsA patients attending the Rheumatology Units of the University of Rome Tor Vergata (Italy), the University of Bari (Italy), and the University of Florence (Italy) between January 1st 2023, and December 31st 2023. Data were collected in an electronic database.

Inclusion criteria were: 1. patients classified as affected by PsA according to the classification criteria for Psoriatic Arthritis (CASPAR) (5); 2. age ≥18 years; 3. a high-resolution computed tomography (HRCT) chest imaging prior to the initiation of biological disease-modifying anti-rheumatic drugs (bDMARDs) or targeted synthetic DMARDs (tsDMARDs).

Exclusion criteria were: 1. past and ongoing lung infections, including Covid 19 pneumonia; 2. primary or secondary lung diseases, including bronchial asthma; 3. concomitant presence of other rheumatological diseases, including fibromyalgia. Patients with missing major clinical data were excluded from the analysis.

The study was approved by the independent local ethics committee from the University of Rome Tor Vergata. Informed consent was obtained from all patients before they were included in the study. The study was conducted in accordance with the ethical principles of the Declaration of Helsinki and was consistent with good clinical practice guidelines.

*Definition of the cohorts*

Patients affected by PsA were assigned to one of the three different cohorts:

1. Patients with a diagnosis of chronic obstructive pulmonary disease (COPD) made by an expert pneumologist based on clinical manifestations (shortness of breath, cough, or sputum production) and spirometry values (post-bronchodilator ratio of forced expiratory volume in 1 second [FEV1] to forced vital capacity [FVC] <0.70) (6).

2. Patients with a diagnosis of interstitial lung disease (ILD), established through a multidisciplinary approach involving both a radiologist and a pulmonologist, based on high-resolution computed tomography (HRCT) features, and guided by the recommendations of the international guidelines of the American Thoracic Society (ATS) and European Respiratory Society (ERS) (7). HRCT features defined different ILD patterns (8). Usual interstitial Pneumonia (UIP) was characterised by areas of basal and subpleural fibrosis and typical radiographic signs such as honeycomb fibrosis and traction bronchiectasis. Probable UIP was characterised by the same distribution and features as UIP, but honeycombing was absent. Non-specific interstitial pneumonia (NSIP) was characterised by symmetrical ground-glass opacities, fine reticulation on basal and peripheral distribution and absence of honeycomb fibrosis. Cellular NSIP is typically characterised by minimal or absent reticulation, limited traction bronchiectasis, and relative subpleural sparing. In contrast,

fibrosing NSIP demonstrates more pronounced reticular abnormalities, traction bronchiectasis, and, in some cases, early honeycombing, reflecting irreversible fibrotic changes. Organising pneumonia (OP) was characterised by patchy periferal and peribronchial consolidation and migratory opacities (9).

3. Patients without clinical and/or radiographic signs of lung disease (LD).

Patients showing non-specific radiographic abnormalities, such as signs of bronchitis/emphysema, interstitial thickening, combined bronchovascular/interstitial abnormalities, and pulmonary nodules not associated with a defined lung condition, were excluded.

*Data collection*

Age, sex, smoking habits (considering both current and former smokers), Body Mass Index (BMI), age at PsA diagnosis, and clinical features of PsA were collected from medical records. PsA patients were classified as having peripheral or axial involvement based on the current joint manifestation and according to the most recent Assessment of Spondyloarthritis International Society (ASAS) classification criteria (10). Peripheral arthritis was defined as oligoarticular when fewer than five joints were involved and as polyarticular when five or more joints were involved (11). Dactylitis, characterised by the presence of diffuse swelling of an entire finger or toe (12), and enthesitis, defined as pain at the insertion sites of tendons and ligaments to the bone (13), were clinically assessed. Extra- articular manifestations were also recorded. Disease activity was assessed at the beginning of b- or tsDMARD therapy and included: laboratory values such as erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP), tender and swollen joint counts (TJC68/SJC66), Disease Activity Index for Psoriatic Arthritis (DAPSA), visual analogue scale (VAS) for pain (VAS pain) and disease activity (VAS activity). The Psoriasis Area Severity Index (PASI) was also assessed. Lastly, for patients with a diagnosis of COPD and ILD, age at LD diagnosis was collected.

*Statistical analysis*

Continuous variables were summarised as mean and standard deviation (SD) or median and interquartile range (IQR), as appropriate, after testing for Gaussian distribution. They were compared using the parametric unpaired t-test or non-parametric unpaired Mann-Whitney U-test, as appropriate. Categorical variables were presented as absolute frequencies and percentages and were compared using the Chi-squared test or Fisher's exact test, as appropriate. A *p*-value <0.05 was considered significant. A binary logistic regression analysis was used to evaluate the adjusted risk of LDs development by correcting for confounding factors and by the evaluation of variables that emerged as statistically significant in the univariate analysis, allowing calculation of the odds ratio (OR) for each risk factor. An adjusted *p*-value <0.05 was considered significant. All statistical analyses were made using IBM SPSS Statistics v. 27 (IBM SPSS Software, Armonk, NY, USA).

**Table I.** Demographics and clinical data of the entire cohort.

| Characteristics | PsA cohort |
|---|---|
| no. of patients | 322 |
| Smoke, n (%) | 115/261 (44.1) |
| BMI ≥30, n (%) | 62/224 (27.7) |
| Age (years), mean ± SD | 56.3 ± 13.4 |
| Age (years) at PsA diagnosis, median (Q1-Q3) | 47 (36.5-55) |
| **Disease domains** | |
| PsO, n (%) | 256/320 (80) |
| Onychopathy, n (%) | 109/320 (34.1) |
| Dactylitis, n (%) | 72/318 (22.6) |
| Enthesitis, n (%) | 127/321 (39.6) |
| Peripheral joint disease, n (%) | 256/320 (80) |
| Oligoarticular, n (%) | 134/320 (41.9) |
| Polyarticular, n (%) | 122/320 (38.1) |
| Axial disease, n (%) | 64/320 (20) |
| **Disease activity** | |
| TJC, median (Q1-Q3) | 5 (3-10) |
| SJC, median (Q1-Q3) | 1 (0-3) |
| DAPSA, median (Q1-Q3) | 14 (0-26) |
| VAS pain, median (Q1-Q3) | 60 (40-80) |
| VAS activity, median (Q1-Q3) | 50 (40-80) |
| PASI >10, n (%) | 76/281 (27) |
| **Lung disease** | |
| COPD, n (%) | 39/322 (12.1) |
| ILD, n (%) | 27/322 (8.4) |

BMI: Body Mass Index; SD: standard deviation; PsA: psoriatic arthritis; TJC: tender joint count; SJC: swollen joint count; DAPSA: Disease Activity in Psoriatic Arthritis; VAS: visual analogue scale; PASI: Psoriasis Activity and Severity Index; COPD: chronic obstructive pulmonary disease; PsO: psoriasis; ILD: interstitial lung disease.

## Results

*Demographic and clinical characteristics of the study population*

Demographic and clinical characteristics of the entire cohort are summarised in Table I. A total of 415 consecutive patients were identified, 93 of whom were excluded due to non-specific lung abnormalities (Fig. 1). Of the remaining patients, 115 (44.1%) were current or former smokers, and 62 (27.7%) had a BMI ≥30. A total of 256 patients (80%) had concomitant PsO, 109 (34.1%) had concomitant onychopathy, 72 (22.6%) had dactylitis and 127 (39.6%) had enthesitis. Peripheral arthritis was the most common articular involvement (80%), followed by axial involvement (20%). The median TJC and SJC were 5 (3–10) and 1 (0–3), respectively, while the median DAPSA was 14 (0–26). The median VAS pain was 60 (40–80) and the median VAS activity was 50 (40–80). A total of 76 patients (27%) had a PASI >10 (severe PsO). COPD was diagnosed in 39 patients (12.1%), ILD in 27 patients (8.4%) and 256 patients (79.5%) had normal chest imaging.

*Characteristics of PsA patients affected by COPD*

Demographic data and clinical characteristics of PsA patients affected by COPD are reported in Table II. A total of 39 patients (12.1%) were affected

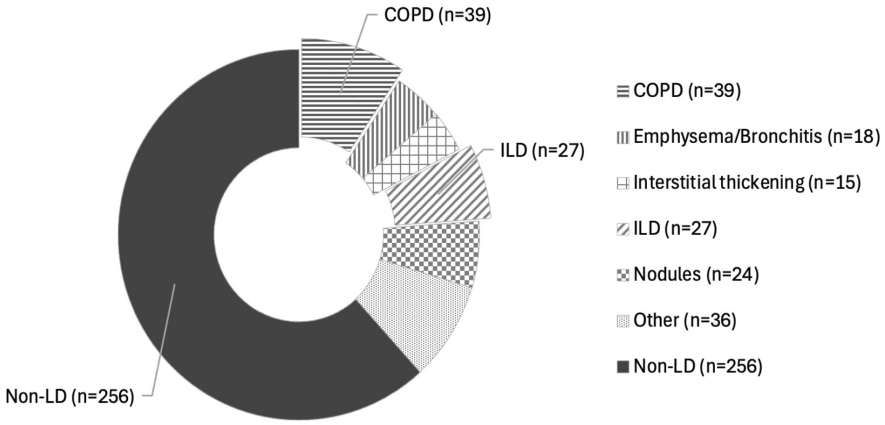


**Fig. 1.** Lung involvement in 415 patients with psoriatic arthritis.
COPD: chronic obstructive pulmonary disease; ILD: interstitial lung disease; LD: lung disease.

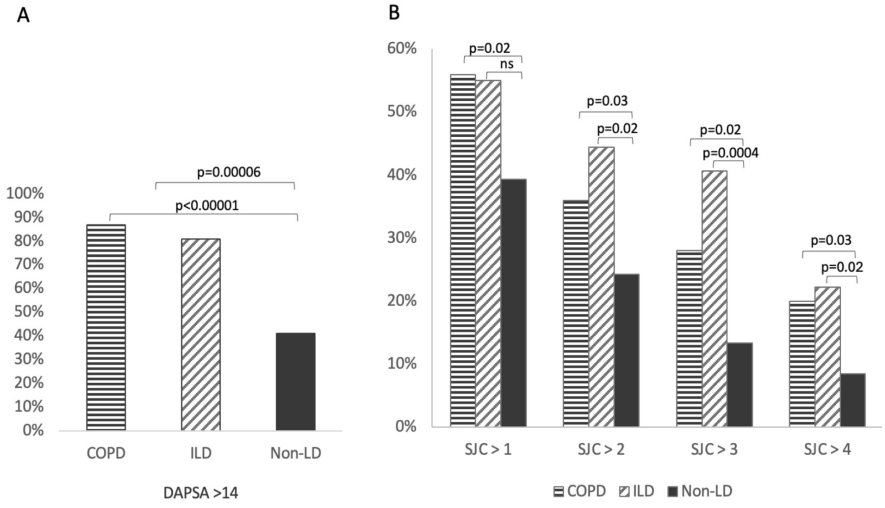


**Fig. 2 A**: DAPSA values in COPD, ILD and non-LD patients. 34 COPD (87.2%), 22 ILD (81.4%), and 105 non-LD patients (41%) had DAPSA >14.
**B**: Number of swollen joints in COPD, ILD and non-LD patients.
DAPSA: disease activity in psoriatic arthritis; COPD: chronic obstructive pulmonary disease; ILD: interstitial lung disease; LD: lung disease; SJC: swollen joint count.

by COPD, of whom 25 (64.1%) were female subjects and 14 (35.9%) were male subjects. The mean age ± SD was 65.1±7.8 years, and the median age at PsA and COPD diagnosis was 54 (48-59) and 61.5 (57–63.5) years, respectively. Eighteen patients (51.4%) were current and former smokers, and 15 patients (48.4%) had a BMI ≥30. PsO was reported in 29 patients (74.3%), onychopathy in 14 (35.9%), dactylitis in 7 (17.9%) and enthesitis in 17 (43.6%). Most patients had peripheral joint involvement (82.1%), with a predominance of polyarticular (48.7%) *vs.* oligoarticular arthritis (33.3%), while axial disease was detected in 7 patients (17.9%). The median DAPSA was 20 (0–32), with a median TJC of 6 (4–10), a median SJC of 2 (0–4), a median VAS pain of 62.5 (60–80) and a median VAS activity of 50 (40–75). A total of 20 patients (51.3%) had a PASI >10.

In the univariate analysis, the mean age of patients with concomitant COPD and PsA was higher than that of patients with PsA but without COPD (65.1±7.8 *vs.* 53.8±13.4, $p<0.00001$). COPD patients had a higher median age at PsA diagnosis compared to patients without LD [54 (48-59) *vs.* 44 (33.5-53), $p<0.00001$]. Smoking habits were not more prevalent among COPD patients compared to non-LD patients (51.4% *vs.* 42.6%, $p=0.3$). Obesity was statistically more common in COPD patients than non-LD patients (48.4% *vs.* 21.3%, $p=0.002$). Gastroesophageal reflux disease (GERD) was not prevalent among COPD patients compared to non-LD patients (46.1% *vs.* 43%, $p=0.7$). The prevalence of PsO, onychopathy, dactylitis and enthesitis was similar between COPD and non-LD patients. Among COPD patients, the prevalence of those with moderate to severe DAPSA was higher compared to patients without LD (87.2% *vs.* 41%, $p<0.00001$) (Fig. 2A). Additionally, the median DAPSA [20 (0–32) *vs.* 11 (0–24), $p=0.001$], median TJC [6 (4–10) *vs.* 5 (2–9.5), $p=0.04$], and median SJC [2 (0–4) *vs.* 1 (0-2), $p=0.04$] were higher in patients with COPD than in those without COPD (Table II).

As shown in Figure 2B, the number of swollen joints was higher in COPD patients compared to non-LD patients, regardless of the number of joints involved. A greater proportion of COPD patients also had a PASI score >10 compared to non-LD patients (51.3% *vs.* 17%, $p<0.00001$). Median VAS activity and VAS pain were similar between the two groups.

In the multivariate analysis (Fig. 3), adjusted binary logistic regression analysis showed that SJC >3 [OR 3.2 (95% CI 1.2 to 8.3), adjusted p=0.02], DAPSA >14 [OR 4.3 (95% CI 1.1 to 16.5), adjusted $p=0.03$], PASI >10 [OR 3.6 (95% CI 1.4 to 9.4), adjusted $p=0.008$], BMI ≥30 [OR 3 (95% CI 1.1 to 8.6), adjusted $p=0.04$], and age ≥65 years [OR 3.8 (95% CI 1.5 to 9.6), adjusted $p=0.004$] were associated with an increased risk of developing COPD. TJC did not maintain statistical significance in the multivariate analysis. Smoking habits and male sex were not found to be risk factors for COPD development in either the univariate or multivariate analysis (Table II and Fig. 3).

### *Characteristics of PsA patients affected by ILD*

Demographic data and clinical characteristics are summarised in Table II. ILD was diagnosed in 27 patients (8.4%), of whom 19 (70.4%) were female subjects and 8 (29.6%) were male subjects. The mean age ± SD was 66.9±9.6 years, while the median age at

**Table II.** Demographic data and clinical characteristics of COPD, ILD and non-LD patients.

| | COPD | ILD | Non-LD | P1 | P2 |
|---|---|---|---|---|---|
| n. of patients, n (%) | 39/322 (12.1) | 27/322 (8.4) | 256/322 (79.5) | | |
| Male, n (%) | 14/39 (35.9) | 8/27 (29.6) | 93/256 (36.3) | 0.9 | 0.5 |
| Smoke, n (%) | 18/35 (51.4) | 11/24 (45.8) | 86/202 (42.6) | 0.3 | 0.8 |
| BMI ≥30, n (%) | 15/31 (48.4) | 3/20 (15) | 40/188 (21.3) | **0.002** | 0.5 |
| Age (years), mean ± SD | 65.1 ± 7.8 | 66.9 ± 9.6 | 53.8 ± 13.4 | **<0.00001** | **<0.00001** |
| Age (years) at PsA diagnosis, median ($Q_1$-$Q_3$) | 54 (48-59) | 58 (53-64) | 44 (33.5-53) | **<0.00001** | **<0.00001** |
| Age (years) at LD diagnosis, median ($Q_1$-$Q_3$) | 61.5 (57-63.5) | 66 (58-72) | NA | - | - |
| **Disease domains** | | | | | |
| PsO, n (%) | 29/39 (74.3) | 18/26 (69.2) | 209/255 (82) | 0.3 | 0.1 |
| Onychopathy, n (%) | 14/39 (35.9) | 11/26 (42.3) | 84/255 (32.9) | 0.7 | 0.3 |
| Dactylitis, n (%) | 7/39 (17.9) | 5/26 (19.2) | 60/253 (23.7) | 0.4 | 0.6 |
| Enthesitis, n (%) | 17/39 (43.6) | 7/26 (26.9) | 103/256 (40.2) | 0.7 | 0.2 |
| Peripheral joint disease, n (%) | 32/39 (82.1) | 23/26 (88.5) | 201/255 (78.8) | 0.6 | 0.2 |
| Oligoarticular, n (%) | 13/39 (33.3) | 8/26 (30.8) | 114/255 (44.7) | 0.2 | 0.9 |
| Polyarticular, n (%) | 19/39 (48.7) | 15/26 (57.7) | 87/255 (34.1) | 0.08 | **0.02** |
| Axial disease, n (%) | 7/39 (17.9) | 3/26 (11.5) | 54/255 (21.2) | 0.6 | 0.2 |
| **Disease activity** | | | | | |
| TJC, median ($Q_1$-$Q_3$) | 6 (4-10) | 7 (5-10) | 5 (2-9.5) | **0.04** | 0.1 |
| SJC, median ($Q_1$-$Q_3$) | 2 (0-4) | 2 (0-4) | 1 (0-2) | **0.04** | 0.1 |
| DAPSA, median ($Q_1$-$Q_3$) | 20 (0-32) | 22 (0-29) | 11 (0-24) | **0.001** | **0.03** |
| VAS pain, median ($Q_1$-$Q_3$) | 62.5 (60-80) | 70 (50-90) | 60 (35-80) | 0.2 | **0.04** |
| VAS activity, median ($Q_1$-$Q_3$) | 50 (40-75) | 70 (50-80) | 50 (30-80) | 0.4 | **0.03** |
| PASI >10, n (%) | 20/39 (51.3) | 4/21 (19) | 28/165 (17) | **<0.00001** | 0.8 |

COPD: chronic obstructive pulmonary disease; ILD: interstitial lung disease; LD: lung disease; BMI: Body Mass Index; SD: standard deviation; PsA: psoriatic arthritis; NA: not applicable; PsO: psoriasis; TJC: tender joint count; SJC: swollen joint count; DAPSA: Disease Activity in Psoriatic Arthritis; VAS: visual analogue scale; PASI: Psoriasis Activity and Severity Index. P1: *p*=value between COPD and non-LD patients; P2: *p*=value between ILD and non-LD.

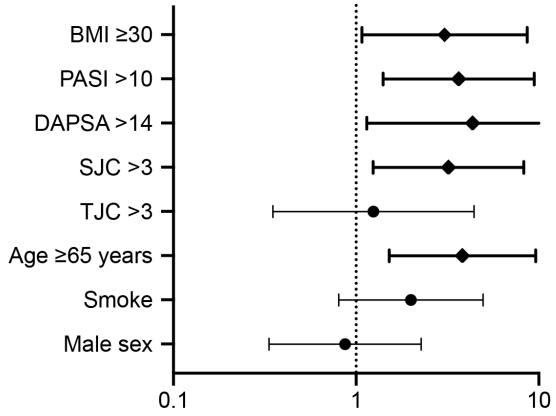


**Fig. 3.** Risk factors for COPD in PsA.
Forest Plot representing the OR of COPD in PsA patients, calculated by adjusted binary logistic regression analysis: male sex 0.9 (95% CI 0.3 to 2.3, adjusted *p*=0.8); smoke 2 (95% CI 0.8 to 5, adjusted *p*=0.1); age ≥65 years 3.8 (95% CI 1.5 to 9.6, adjusted *p*=0.004); TJC >3 1.2 (95% CI 0.3 to 4.4, adjusted *p*=0.7); SJC >3 3.2 (95% CI 1.2 to 8.3, adjusted *p*=0.02); DAPSA >14 4.3 (95% CI 1.1 to 16.5, adjusted *p*=0.03); PASI >10 3.6 (95% CI 1.4 to 9.4, adjusted *p*=0.008); BMI ≥30 3 (95% CI 1.1 to 8.6, adjusted *p*=0.04).
COPD: chronic obstructive pulmonary disease; PsA: psoriatic arthritis; OR: odds ratio; TJC: tender joint count; SJC: swollen joint count; DAPSA: disease activity of psoriatic arthritis; PASI: Psoriasis Activity and Severity Index; CVDs: cardiovascular diseases; BMI: Body Mass Index.

PsA and ILD diagnosis was 58 (53–64) and 66 (58–72) years, respectively. Eleven patients (45.8%) were current or former smokers, and 3 patients (15%) had a BMI ≥30. PsO was detected in 18 patients (69.2%) and onychopathy in 11 patients (42.3%), while dactylitis was diagnosed in 5 patients (19.2%) and enthesitis in 7 patients (26.9%). As with COPD, the majority of ILD patients had peripheral arthritis (88.5%), 15 patients (57.7%) had polyarticular involvement, and 8 patients (30.8%) had oligoarticular arthritis. Axial disease was detected in 3 patients (11.5%). Regarding disease activity, the median TJC was 7 (5–10) and the median SJC was 2 (0–4). The median DAPSA was 22 (0-29), while the median VAS pain and VAS activity scores were 70 (50–90) and 70 (50–80), respectively. Four patients (19%) had a PASI score >10. In Supplementary Table S1 we reported the articular phenotype stratified by cutaneous psoriasis, nail involvement, both, or neither. These data were excluded from the main manuscript due to a lack of significant differences.

In the univariate analysis, the mean age of patients with concomitant ILD and PsA was higher than that of patients with PsA but without ILD (66.9±9.6 *vs*. 53.8±13.4, *p*<0.00001). ILD patients had a higher median age at PsA diagnosis compared to patients without LD [58 (53–64) *vs*. 44 (33.5–53), *p*<0.00001]. Smoking habits were not more prevalent among ILD patients compared to non-LD patients (45.8% *vs*. 42.5%, *p*=0.8). The prevalence of PsO, onychopathy, dactylitis and enthesitis did not differ between ILD and non-LD patients. Patients with ILD had a higher prevalence of polyarticular arthritis compared to non-LD patients (57.7% *vs*. 34.1%, *p*=0.02). Among ILD patients, the prevalence of those with moderate-

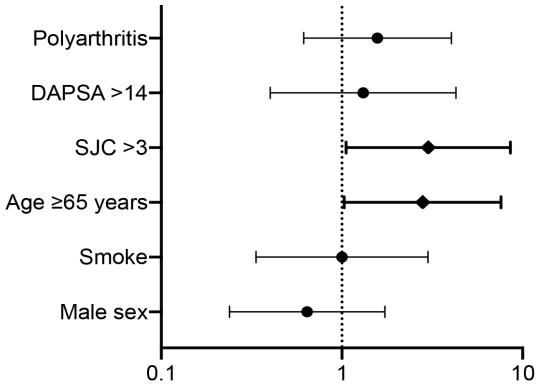


**Fig. 4.** Risk factors for ILD in PsA.
Forest Plot representing the OR of ILD in PsA patients, calculated by adjusted binary logistic regression analysis: male sex 0.6 (95% CI 0.2 to 1.7, adjusted $p$=0.4); smoke 1 (95% CI 0.3 to 3, adjusted $p$=0.9); age ≥65 years 2.8 (95% CI 1 to 7.6, adjusted $p$=0.04); SJC >3 3 (95% CI 1 to 8.5, adjusted $p$=0.04); DAPSA >14 1.3 (95% CI 0.4 to 4.3, adjusted $p$=0.6); polyarthritis 1.6 (95% CI 0.6 to 4, adjusted $p$=0.3).
ILD: interstitial lung disease; PsA: psoriatic arthritis; OR: odds ratio; SJC: swollen joint count; DAPSA: disease activity of psoriatic arthritis; CVDs: cardiovascular diseases.

severe DAPSA was higher compared to patients without LD (81.4% *vs*. 41%, $p$=0.00006) (Fig. 2A). This prevalence also emerged when comparing median DAPSA values between ILD and non-LD patients [22 (0–29) *vs*. 11 (0–24), $p$=0.03] (Table II). Median VAS pain [70 (50–90) *vs*. 60 (35–80), $p$=0.04] and VAS activity [70 (50–80) *vs*. 50 (30–80), $p$=0.03] was higher in the ILD group rather than the non-LD group. In contrast, the median number of TJC and SJC was similar between ILD and non-LD patients (Table II). However, as shown in Figure 2B, the prevalence of SJC was higher in ILD patients than in non-LD patients when more than two joints were involved.

In the multivariate analysis (Fig. 4), adjusted binary logistic regression analysis revealed that age ≥65 years [OR 2.8 (95% CI 1 to 7.6), adjusted $p$=0.04] and SJC >3 [OR 3 (95% CI 1 to 8.5), adjusted $p$=0.04] were associated with an increased risk of developing ILD. DAPSA>14 and polyarticular arthritis failed to maintain statistical significance in the multivariate analysis. Smoking habits and male sex did not emerge as risk factors for ILD development in either the univariate or multivariate analysis (Table II and Fig. 4).

## Discussion

Here we described demographic data, clinical characteristics, and comorbidities of 322 patients from three rheumatological centres. The median age at PsA diagnosis was 47 years (36.5–55), consistent with previously reported data in the literature (14). Nearly half of the patients were smokers, and almost one-third were obese. Cigarette smoking and obesity have been associated with worse prognosis and reduced treatment response in PsA patients (15-17), which may have contributed to the high articular disease activity observed in our cohort [median DAPSA of 14 (0–26)].

As expected, peripheral arthritis was the most common articular involvement, affecting 79% of patients, followed by axial disease in 21% (18, 19). The prevalence of clinical dactylitis was lower than reported in the literature (20, 21), highlighting the importance of musculoskeletal ultrasound, which is more sensitive in detecting dactylitis (22). Regarding enthesitis, it was present in approximately one-third of PsA patients (39.6%) similar to the 35% frequency reported by Polachek *et al*. (23). In line with previous studies, up to 80% of PsA patients in our cohort had psoriasis (20). However, comparing the prevalence of onychopathy to other studies is challenging due to the high variability of this clinical manifestation, as revealed in a previous review (24).

Regarding COPD, the link between this disease and PsA has garnered increasing interest, although their association remains largely hypothesised (25). Advanced age is a well- established independent risk factor for COPD, with its prevalence increasing five-fold in the general population aged over 65 years (OR up to 9.6 times in patients aged ≥65 years in our cohort) (26). Undoubtedly older age *per se* increases the risk of COPD, however inflammation along with airways remodeling have been identified as potential mechanisms for the pathogenesis of COPD in never-smokers subjects, since about half of COPD diagnosis are due to non-tobacco-related risk factors (27). This evidence may explain the intriguing observation in our study that smoking, a known risk factor for COPD, did not reach statistical significance when comparing COPD and non-COPD patients. However, in our study, smoking status was typically recorded in categorical terms (current, former, or never smoker) without precise quantification on cigarette smoking exposure, such as pack/years. Previous studies (28) in patients with psoriasis have demonstrated a significantly increased risk of COPD among individuals with ≥20 pack-years of exposure. Therefore, the absence of this information may have led to an underestimation of the association between smoking and pulmonary comorbidities in PsA. Future studies should aim to systematically collect pack-year data to better evaluate the impact of smoking and how non-traditional risk factors, such as chronic inflammatory diseases, may increase the risk of COPD development.

After evaluating the main environmental pneumotoxic agents (such as mold or high levels of air pollution due to proximity to high-traffic roads), no risk factors for COPD were identified. Since the data were collected from medical records in the rheumatological setting, passive exposure to smoking was not investigated.

Obesity also emerged as an important factor in our analysis, with PsA patients with concomitant COPD exhibiting higher BMI levels compared to those without lung involvement. Obesity is a recognised risk factor for COPD, as it induces chronic low-grade inflammation (29). Notably, in our study PsA pa-

tients with a BMI ≥30 had an 8.6-fold increased risk of developing COPD compared to non-obese patients. On the other hand, the association between PsO and COPD has been more extensively studied. We observed that nearly half of PsA patients with COPD had more severe PsO than those without lung involvement. Moreover, PsA patients with PASI >10 had a 9.4-fold higher risk of developing COPD, higher than what is reported in an extensive systematic review, which nevertheless only included patients with PsO (30). This suggests that PsO severity, known to be positively associated with the risk of developing PsA (31), may also elevate the risk of COPD in PsA patients. Furthermore, our findings indicate that PsA patients with concomitant COPD had higher articular disease activity than PsA patients without COPD. In the multivariate analysis, DAPSA >14 emerged as the most significant predictor of COPD in PsA patients, with an OR of up to 16.5. While the number of tender and swollen joints was associated with that increased risk, only swollen joints maintained statistical significance in the multivariate analysis (OR 2.9). This distinction is important, as tender joints may reflect residual chronic pain caused by structural joint damage or mechanisms such as central sensitisation, rather than active inflammation (32). Interestingly, COPD diagnosis was generally made 7.5 years after PsA diagnosis, highlighting the potential for systemic inflammation from the articular disease to target airways of these patients over time. Additionally, PsA patients with concomitant COPD were likely to exhibit polyarticular arthritis than those without LD (48.7% *vs*. 34.1%, respectively). Although these differences did not reach statistically significance, they support our hypothesis that systemic inflammation may play a role in COPD development. About 46% of PsA patients with COPD patients had GERD in a similar percentage of PsA patients without LD (43%). GERD is a highly prevalent comorbidity in patients with COPD, with reported prevalence rates ranging from 17% to as high as 78%, depending on the diagnostic criteria used (clinical symptoms, instrumental testing, or questionnaire-based assessments). This wide variability reflects differences in diagnostic approaches. Several studies have shown that the presence of GERD in patients with COPD is significantly associated with increased exacerbation frequency, more severe respiratory symptoms, and poorer quality of life. (33). In our study, GERD did not appear to be associated with COPD. This is because, being a retrospective study, the presence of GERD was self-reported by the patients during clinical history-taking, and we had no information on the diagnostic criteria (clinical, instrumental, or endoscopic) used and on the type and severity of symptoms presented. Regarding ILD, recent studies and case reports have explored its prevalence among patients with PsO (34-36). However, limited research has focused on ILD in patients with PsA (37-40). In patients with rheumatoid arthritis (RA), advanced age is closely linked to the development of lung fibrosis (41). Similarly, in PsA patients, the risk of ILD increases significantly with age, reaching an OR of 7.6 in patients aged ≥65 years in our cohort. In our cohort, no cases of familial interstitial lung disease were reported. While this finding does not exclude a potential genetic predisposition, it contrasts with evidence from studies on rheumatoid arthritis-associated ILD (RA-ILD), in which familial clustering and specific genetic variants, such as mutations in TERT, RTEL1 (42), and the MUC5B promoter polymorphism (43) is associated with a high risk of ILD in RA. The absence of a reported family history in our PsA-ILD population may reflect a true lower prevalence of genetically driven ILD in psoriatic arthritis or could be related to the retrospective nature of the data collection, which may limit the availability of detailed familial histories.

ILD patients had not reported environmental exposure to major known pneumotoxic agents such as asbestos, silica or use of drugs known to induce pulmonary fibrosis.

We also investigated smoking, a well-known risk factor for the development of ILD (44). Interestingly, in our cohort of PsA patients, smoking prevalence was similar between those with ILD and those without lung involvement. Moreover, smoking habits did not emerge as a risk factor for ILD disease in PsA patients, suggesting that other mechanisms may play a role in its pathogenesis, such as the underlying inflammatory process due to the chronic articular disease (45). As specified in PsA patients with COPD, cumulative smoking exposure – measured in pack-years – was not available for patients with ILD either. This represents a limitation, as existing literature in RA has demonstrated a clear dose-dependent relationship between smoking exposure and the development of fibrotic lung changes, with significantly higher incidence observed in individuals with more than 20 pack-years compared to those with lower exposure (46). Consequently, the lack of an observed association between smoking and ILD in our cohort may be attributable either to an underestimation resulting from insufficient data, or to the possibility that underlying systemic inflammation plays a predominant role in the pathogenesis of ILD in PsA. In fact, PsA patients with ILD had higher articular disease activity than patients without LD, although DAPSA failed to maintain statistical significance in the multivariate analysis. However, DAPSA is a composite index partially influenced by the subjective perception of the disease and in some cases, it may not reflect the real inflammatory burden. Conversely, a high purely objective inflammatory index such as SJC was associated with an 8.5-fold increased risk of developing ILD.

In our cohort, the median time to ILD diagnosis was approximately 8 years after PsA onset. As for COPD, this temporal relationship supports the hypothesis that ILD occurrence may be related to the cumulative inflammatory burden of PsA, akin to the well-documented relationship in RA (47). Unlike COPD, patients with ILD did not exhibit more severe PsO compared to those without ILD. While this finding may be influenced by the small sample size of the PsA-ILD group, it aligns with observations from a Japanese cohort, where

PASI scores did not differ between PsO patients with and without ILD (34).

The diagnosis of ILD in our study was based on clinical features and high-resolution computed tomography (HRCT) imaging. Within our total cohort, 2 patients (7.4%) exhibited radiological features consistent with a typical UIP pattern, 3 with probable UIP (11.1%), 14 with cellular NSIP (51.9%), 1 with fibrosing NSIP (3.7%) and 2 with OP (7.4%). 5 patients (18.5%) presented HRCT findings as limited areas of reticulation and minimal architectural distortion insufficient to meet the criteria for either probable UIP or to suggest an alternative diagnosis. This predominance of NSIP suggests that immune-mediated inflammation, rather than fibrotic changes, may be the primary pulmonary manifestation of PsA. Notably, our findings align with those of a recent study, where 55% of PsA patients with ILD exhibited an NSIP pattern (48).

The high incidence of LDs observed in our cohort suggests a potential association between PsA and LDs. This relationship may be explained by shared immune and molecular mechanisms linked to chronic inflammation, common risk factors such as smoking and obesity, and other pathways still to be identified. The prevalence of COPD in our cohort aligns with existing literature (49), while the incidence of ILD appears higher than previously reported (36). Notably, a subset of patients excluded from our analysis presented radiographic anomalies without respiratory symptoms suggestive of LDs. Emerging evidence of new conditions such as pre-COPD that identifies individuals based on symptoms, physiological or radiographic abnormalities not meeting COPD diagnostic criteria but are clearly at risk, suggests more thorough evaluation of PsA patients with chest CT abnormalities to detect subclinical lung diseases at an early stage (50).

The limitations of this study include: 1. its retrospective design; 2. exclusion of a substantial number of patients due to missing data, including lung function test results; 3. lack of investigation into other potential lung injury factors (*e.g.* passive smoking, unknown occupational and environment exposures); 4. absence of a healthy control group for comparison.

In summary, our findings suggest a potential association between psoriatic arthritis (PsA) and lung diseases (LDs), particularly in patients with active or prolonged articular disease. This aligns with recent evidence highlighting the clinical heterogeneity of PsA and the emergence of complex phenotypes, including difficult-to-treat cases, which are often associated with comorbidities and suboptimal treatment response (50, 51). Systemic inflammation, in addition to traditional risk factors such as smoking and obesity, may play a key role in the pathogenesis of pulmonary involvement. Given the potential impact on morbidity and quality of life, clinicians should maintain a high index of suspicion for LDs in patients with PsA, especially in those with more severe disease trajectories.

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