1184 Scientific Abstracts

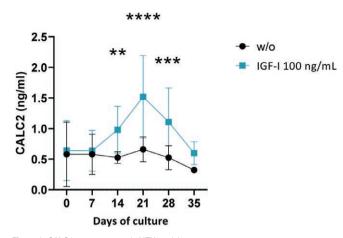


Figure 1. CALC2 measurements in HEX model.

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AB0106 THE PATTERN OF JOINT INFLAMMATION IN THE CAIA MOUSE MODEL OF ARTHRITIS

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Background: Animal models of inflammatory arthritis such as the collagen-antibody induced arthritis (CAIA) have a variable disease incidence in hind paw arthritis, complicating experimental design.

Objectives: To investigate the use of 18FDG μ PET/CT as a guide for inflammation in joints otherwise inaccessible for clinical scoring, enabling the use of additional joints for histological analysis.

Methods: CAIA was induced in 8 male DBA/1 mice using the ArthritoMab Antibody cocktail (4 mg/mouse on day 0), followed by 100 μ g LPS at day 3. Body weight, clinical signs of arthritis such as paw swelling and grip strength loss were recorded 3 times per week in both front and hind paws. Whole body 18FDG μ PET/CT was performed at day 11, the estimated time of peak inflammation, and inflammation was scored visually on images scaled to the same standardised uptake value. At day 28, animals were euthanized and peripheral joints were collected for histological analysis.

Results: Cumulative disease incidence based on paw swelling dropped from 100% to 87.5% when looking only at hind paws, and to 75% when taking into account only the region suitable for histological analysis, namely the ankle and midfoot of the hind paws. Symmetrical, bilateral hind paw arthritis was not observed. While hind paw arthritis showed a tendency to be less severe in comparison to front paw arthritis, grip strength was equally affected, indicating possible involvement of other hind limb joints. μPET/CT images detected inflammation in the hind paws (at least unilateral involvement in 62.5% of the mice, bilateral involvement in 0%), knee joints (at least unilateral in 50%, hip joints (at least unilateral in 50%, bilateral in 50%, bilateral in 37.5%) and shoulder joints (at least unilateral in 25%, bilateral in 25%). Histology could confirm this inflammation with the presence of inflammatory infiltrates and bone erosions. Grip strength loss in hind limbs without paw swelling correlated only weakly with knee inflammation detected by μPET/CT.

Conclusion: In the CAIA model, inflammatory arthritis can develop in all peripheral joints, in particular with a high incidence in knee joints, which are highly suitable for subsequent histological analysis. Since clinical scoring seems insufficient for detecting these affected joints, implementation of in vivo imaging modalities such as $\mu PET/CT$, offers a substantial benefit in disease monitoring and assessment.

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AB0107

IMPACT OF SARS-COV-2 INFECTION ON THE DISEASE ACTIVITY OF PATIENTS WITH PSORIATIC ARTHRITIS UNDER BDMARDS: REAL LIFE DATA

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Background: SARS-CoV-2 infection can lead to severe inflammation and has been suggested to induce Psoriatic Arthritis (PsA) flares. However, the impact on disease activity and response to biological disease modifying anti-rheumatic drugs DMARDs (bDMARDs) remains unknown.

Objectives: To evaluate the effect of SARS-CoV-2 infection on disease activity and bDMARDs responses in patients with PsA.

Methods: We performed a retrospective analysis including all the patients with PsA, meeting the CASPAR criteria and under biologic therapy, followed in the Rheumatology department of a tertiary university hospital. Demographic and clinical data, including occurrence of SARS-CoV-2 infection, were collected from our national database (reuma.pt). Disease activity (CDAI, SDAI, DAS28 4v, BASDAI, ASDAS) and bDMARDs responses (EULAR, ASDAS, ASAS, ACR and PsARC responses) were evaluated before and after SARS-Cov-2 infection. Statistical analysis was performed with SPSS. Continuous variables were compared through paired samples t-test.

Results: A total of 102 patients with PsA were included. Fifty-two were females (51%). The mean age was 53 ± 11.09 years and the median disease duration was 15 years [min 2, max 47]. Overall, 54 (53%) patients had predominant axial involvement, 26 (26%) peripheric and 36 (37%) enthesopathic. The most used bDMARD was etanercept (n=28, 27.5%) followed by adalimumab (n=22, 21.6%) and secukinumab (n=18, 17.6%).

The prevalence of SARS-CoV-2 infection was 15.7% (n=16). Sixty-three per cent received the BNT162b2 (Pfizer/BioNtech) vaccine, 31% received mRNA-1273 (Moderna), 13% received AZD1222 (AstraZeneca) and 13% received AD26. COV2.S (Janssen/Johnson & Johnson). Sixty-three percent were infected before any vaccination, 13% after the first dose and 25% after the second. The most common symptoms were anosmia (65%), dysgeusia (56%) and cough (56%). All patients fully recovered from the infection, with no need for hospitalization. Regardless of the score used, the difference between the mean disease activity

after SARS-CoV-2 infection and that at baseline did not reach statistical significance. At baseline and after infection, mean (SD) disease activity parameters were, respectively: CDAI 8.6±5.7 vs 8.6±5.7, p=0.997; SDAI 9.3±6.6 vs 9.2±6.1, p=0,928; DAS 28 4v 2.9±1.2 vs 2.9±1.2, p= 0.818; BASDAI 3.6 ±2.6 vs 3.2±2.7, p=0.506; ASDAS 2.2±1.2 vs 2.2±1, p=0.721.

The number of patients unresponsive to bDMARDs (according EULAR, ASDAS, ASAS, ACR and PsARC) before the infection wasn't different from post-infection. **Conclusion:** Our study suggests that SARS-CoV2 infection has no negative impact on PsA disease activity and bDMARD responses. However, more studies are still needed to better understand the long-term effects of SARS-CoV2 infection.

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[1] Zhou Q et al. SARS-CoV-2 Infection Induces Psoriatic Arthritis Flares and Enthesis Resident Plasmacytoid Dendritic Cell Type-1 Interferon Inhibition by JAK Antagonism Offer Novel Spondyloarthritis Pathogenesis Insights. Front Immunol. 2021 Apr 15; 12:635018

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AB0108

INFLAMMATION IN AXSPA AS A DISRUPTOR OF BONE METABOLISM – THE EFFECT OF PATIENTS' SERA ON AN IN VITRO BONE MODEL

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Background: In axial spondyloarthritis (axSpA), two opposing processes of bone resorption and neogenesis are closely related, with a common denominator – chronic inflammation. This pathological condition affects osteoblasts, among other cells, residing in sites of inflamed tissue, but it can also activate mononuclear precursors in the blood to form osteoclasts [1,2]. To this day, it is not