among ILCs. Multivariate linear regression and Receiver-Operating Characteris-
tic (ROC) Curve analysis was performed using the IBM SPSS Statistics software.
Different in vivo models were used to assess functional implications of ILCs at
different time points of the disease. Joint inflammation was assessed through
MRI and H&E staining of ankle areas. Peripheral blood was obtained from mice
of each group and flow cytometry analysis was performed. High dimensional
analyses including RNA-seq was performed to identify phenotypic characteris-
tics of ILCs implemented into the pathogenesis of the disease.

Results: Total number of circulating ILCs were increased in PsA patients compared
to PsO and healthy controls (p<0.001). Linear regression analyses of the relation-
ship between disease activity and circulating ILCs showed strongest corre-
lation between ILC3 counts and DAPSA score. ILC3 counts also correlated with
imaging signs of inflammation such as enthesitis, synovitis, erosions and/or
osteoporosis as assessed by MRI and HPI-pQCT. Musculoskeletal inflammation in
mice was predominantly associated with p19 expression and IL-23R-signaling as
assessed by RNA-seq. These effects were also accompanied by a strong upregula-
tion of IL-17 -producing lymphocytes within the inflamed joint niche with a dominant
presence of ILC3s. Multi-channel immunofluorescence and confocal laser scanning
microscopy revealed not only upregulation of ILC3 induced IL-17 production within
the synovial membrane but also in peri-articular areas of the inflamed joints.

Conclusion: ILC3s not only correlate with various facets of PsA manifestations
but also functionally contribute to synovitis and enthesitis suggesting them as
an interesting target for upcoming treatment strategies in the near future.

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Background: Vascular calcification is highly correlated with arteriosclerosis.
Ankylosing spondylitis (AS) is associated with a process of accelerated athero-
sclerosis. Wnt signaling plays an important role in the pathogenesis of vascular
calcification. However, there has been no study of the role of Wnt signaling in
vascular calcification in patients with AS.

Objectives: We investigated the relationship between vascular calcification and
Wnt signaling in patients with AS.

Methods: Sixteen male patients aged over 20 years with AS were enrolled. They
fulfilled the modified New York criteria and each of their ankylosing spondylitis
disease activity score was more than 2.1. Sex and age matched nineteen healthy
controls were also recruited.

Mouse MOVAS vascular smooth muscle cell line (American Type Culture Col-
lection, ATCC® CRL-2797™) were stabilized in maintain media for 24 hours.
Then media were exchanged for the 10% serum of patients with AS or controls in
maintain media. Cells were stimulated for another 72hours. We exchanged this
medium with calcification medium. Cells were cultured until 2 weeks then stained
with Alizarin Red S and the optical density (OD) was measured.

For Western blotting and RT-PCR, cells were stabilized for 24 hours and stim-
ulated for another 72 hours through the same procedure as that of Alizarin Red S
staining. After cell stimulation, the level of mRNA and protein were measured by
RT-PCR and western blot, respectively. We measure the level of Low-den-
sity lipoprotein receptor-related protein (LRP5), LRP6, Dickkopf-related protein 1,
Wnt3a, matrix metalloproteinase-7 (MMP-7), beta-catenin for canonical Wnt sig-
naling; Receptor Tyrosine Kinase Like Orphan Receptor 2, Wnt5a, Runt-related