

Objectives: To address this need and our hypothesis that age-related immune effector and regulatory cell subsets changes are present, we employ a multi-dimensional approach using mass cytometry to identify the cell subsets that shape the developing immune landscape from birth to adulthood.

Methods: We interrogated the peripheral blood mononuclear cells from 126 healthy individuals (cord blood, newborn to adult) with mass cytometry using two extensive antibody panels embracing the most important cell lineages and their function. Quality control and batch effect correction were performed with outliers excluded before dimensional reduction and clustering to identify the unique immune cell subsets. Their frequencies across the age categories were presented as 3-D frequency histograms. Subsequent manual gating was done to further describe these immune cell subsets changes with age using Pearson correlation coefficient.

Results: Distinct developmental gradients involving multiple effector and regulatory cell subsets shaped the maturing immune landscape. The naïve TNF α +CD4+T cells were enriched in the early childhood period and declined with age (Pearson correlation coefficient, $r=-0.3503$, $p<0.001$). In contrast, the memory TNF α +CD4+T cells increased with age ($r=0.4149$, $p<0.0001$). A transitional milestone from naïve to memory TNF α +CD4+T cells was observed after 2 year old. This indicates that an intact CD4+T cells TNF α effector mechanism is present at birth which progressively mature to involve the memory CD4+T cells. For another cytokine IL17A, it was secreted solely by the memory CD4+T cells with its population increasing with age ($r=0.3753$, $p<0.001$). Another distinct maturation milestone was observed in the naïve CD8+T cell subset where a transition from IL8 to IFN γ secretion after 10 year old was observed. The IL8+and IFN γ +naïve CD8+T cell subsets correlated negatively and positively with increasing age respectively ($r=-0.2661$, $p<0.01$ and $r=0.2835$, $p<0.01$).

For the T regulatory cells, the age related changes were more gradual. The early thymic T regulatory cells (CD3+, CD4+, CD31+, CD45RA+, CD25+, Foxp3+, CD152+) demonstrated a gradual decline with age ($r=-0.2966$, $p<0.01$) while the memory T regulatory cells gradually increased with age (CD3+, CD4+, CD45RO+, CD25+, Foxp3+, CD152+) ($r=0.2444$, $p<0.05$).

Conclusions: Key developmental milestones in the T cell compartment were identified and with the other subsets identified, a holistic description of the developing immune landscape was obtained. This atlas has the potential dual translational role of defining the stage of immune maturity and distilling the pathological cell subset in both paediatric and adult immune mediated diseases. The database and the related pipeline to analyse it will be provided as a free reference.

Disclosure of Interest: None declared

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AB1194 ARE THERE NEW PARAMETERS TO BE CONSIDERED IN SPECTRAL DOPPLER TO EVALUATE THE NAIL BED IN PSORIASIS AND PSORIASIS ARTHRITIS?

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Background: Other spectral Doppler parameters can assess joint impairment caused by psoriatic arthritis and psoriasis¹.

Objectives: To detect and compare Doppler velocimetric indexes changes in 3 groups of patients.

Methods: Thirty – eight patients were evaluated: 8 in the control group – healthy (52 nail beds); 15 in the psoriasis group (134 nail beds); and 15 in the group with psoriatic arthritis (147 nail beds). "CASPAR" criteria were used to classify the patients. The ultrasound (US) was performed in all patients using Esaote MyLab 50, with high resolution linear probe with a frequency of 18 MHz, it was positioned longitudinally to the nail bed.

Results: The psoriasis group of patients included 7 males (46.6%) and 8 females (53.4%); 66.6% were white, 33.4% black.

Abstract AB1194 – Table 1. Mean and standard deviations of US, clinical and laboratory variables by clinical group:

	Psoriatic Arthritis	Psoriasis	Control
RI (Resistance Index)	0.58 (0.09)	0.59 (0.10)	0.60 (0.07)
PI (Pulsatility Index)	1.05 (0.31)	1.06 (0.45)	1.12 (0.25)
Acceleration (A =m/s ² ;	0.37 (0.29)	0.39 (0.47)	0.51 (0.44)
Acceleration Time (AT =ms)	107.30 (37.10)	114.64 (41.14)	113.10 (40.10)
Angle of Insonation ($\alpha=^{\circ}$)	18.21 (25.8)	14.76 (24.71)	27.28 (30.10)
Nail bed thickness (T =mm)	1.60 (0.78)	1.68 (0.50)	1.44 (0.40)
VHS	27.5 (24.9)	20.4 (14.9)	-
PCR	0.75 (1.1)	0.54 (0.7)	-

Abstract AB1194 – Table 2. Distribution and percentage of individuals according to clinical group and to the PASI, NAPS variables:

	Psoriatic Arthritis N(%)	Psoriasis N(%)	Control N (%)
NAPSI			
Normal (=0)	0 (0)	0	8 (100)
Mild (<20)	4 (28,6)	10(71.4)	0
Severe (>20)	11(68.7)	5 (31,3)	0
PASI			
Mild (<15)	14(51.8)	13(48.2)	0
Moderate ¹⁵⁻²⁵	1 (50,0)	1 (50,0)	0
Severe (>25)	0	1 (100,0)	0

Statistically significant: Mean age and SD of the psoriasis, psoriatic arthritis and control groups, respectively: 57.13 \pm 14.3 years; 54.66 \pm 10.6 years; 24.87 \pm 2.03 years ($p<0.05$). A statistically significant difference was found in the **NAPSI** variable among all groups ($p<0.05$); in the variable **PASI**, a difference was only found in the control group ($p<0.05$) (Kruskall-Wallis) (table 2); the **A** variable between the psoriasis group and control ($p<0.001$); between the psoriatic arthritis group and control ($p=0.001$), the variable **T** between the psoriasis and psoriatic arthritis groups ($p=0.006$) and between the psoriasis and control groups ($p=0.001$) and the α variable between control and psoriasis groups ($p=0.017$) (table 1). **Spearman and Pearson correlations** between US variables per group, psoriasis, psoriatic arthritis and control were: **RlxPI** $r=0.744$ ($p<0.001$), **PD (power Doppler) xT** $r=0.301$ ($p<0.001$), **RlxPI** $r=0.914$ ($p<0.001$), **PlxA** $r=0.46$ ($p<0.001$); **RlxPI** $r=0.889$ ($p<0.001$), **PDxT** $r=0.490$ ($p<0.001$) respectively.

Conclusions: There are other parameters in spectral Doppler to be validated in order to characterise changes in nail beds.

REFERENCE:

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AB1195 DEVELOPMENT AND VALIDATION OF AN ULTRASONOGRAPHIC ACTIVITY SCORE (USAS) FOR RHEUMATOID ARTHRITIS

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Background: Composite scores developed in Rheumatoid arthritis (RA) not include all dimensions of disease activity. An index based on essential clinical plus a ultrasound (US) measures, focused on simplicity, with appropriate validation, would allow a better classification at different levels of disease activity than a clinical only or US only index.

Objectives: To develop and validate a mixed clinical-US inflammation score in RA for use in clinical practice.

Methods: Mixed methods. Experts elicited items reflecting inflammation which were prioritised by Delphi. Patients with RA with various grades of activity underwent clinical [28 swollen and tender joints counts, patient and physician global assessment (PhGA), erythrocyte sedimentation rate, and C-reactive protein (CRP)] and US assessments [synovitis or tenosynovitis by grey-scale (GS) and Power Doppler (PD) of 42 structures], blinded to the clinical assessment. An index was created after supported selection of US structures and scoring method. Construct validity was tested by correlation with DAS28, SDAI, CDAI, and PhGA. Reliability was evaluated in a subgroup of patients with the intraclass correlation coefficient (ICC).

Results: US of joints and tendons, CRP, and swollen joints were the items that passed the prioritisation phase. Then, 281 patients were randomly divided into design (n=141) and validation analysis (n=140). The combination of US sites chosen detected the maximum proportion of GS and PD present. Were elected