(methylprednisolone and/or DMARDs), and the changes in the patients' condition and lymphocyte subsets were observed. The t-test of two independent samples was used when the measurement data conformed to the normal distribution and the variance was homogeneous, and Manil-Whitney rank sum test was used when the measurement data did not conform to the normal distribution.

Results: Among 108 patients, 58 were males and 50 were females, with an average age of 41 + 14 years. Compared with the normal control group, total T cells, total B cells, Th cells, Ts cells, Th1 cells, Th17 cells, Th1/Th2, Th17/Treg in patients with autoimmune uveitis were higher than those in healthy control group ($P < 0.05$), while Th1 cells and Treg cells were lower than those in healthy control group ($P < 0.05$). After IL-2 treatment, the number of Treg cells increased from 21.90±15.29 /ul to 51.54±41.86 /ul ($P < 0.05$), the Th17/Treg ratio decreased back from 0.44±0.27 to 0.33±0.23 ($P < 0.05$), and both serum sedimentation rate and CRP decreased compared with before treatment ($P < 0.05$).

Conclusion: Treg cells are involved in the pathogenesis of autoimmune uveitis. Low dose of IL-2 selectively elevates Treg cells, regulates Th17/Treg balance and improves the condition of the disease.

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AB0049 IMMUNE DYSFUNCTION IN ANKYLOSING SPONDYLITIS (AS) AND THE POTENTIAL OF TUMOR NECROSIS FACTOR-A (TNF-α) INHIBITOR ANBAINUO AS AN EFFECTIVE TREATMENT

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Background: Studies into ankylosing spondylitis (AS) and its relationship with immune function are controversial, and the correlation between the efficacy of TNF-α inhibitor and changes in immune function is unclear.

Objectives: We conducted a prospective study of T-cell and B-cell subset distribution and analyzed lymphocyte function in AS patients to further clarify changes to the immune system caused by AS and to explore resistance that could contribute to relapse after treatment.

Methods: A total of 40 immune cells were tested with flow cytometry, and the results of 105 HC (healthy control) subjects, 177 active-stage AS patients, and 23 AS cases before and after 12 weeks of Anbainuo therapy were analyzed.

Results: Compared with the HC group, the proportion of immune cells, such as naïve and central memory CD4+T cells, in AS increased ($p<0.0001$), but effector memory and terminally differentiated CD4+T cells were decreased ($p<0.01$ and 0.0001, respectively). Naïve, central memory, and effector memory CD8+T cells were increased ($p<0.0001$, 0.001, and 0.01, respectively), but terminally differentiated CD8+T cells were decreased ($p<0.0001$). Th1 cells (helper T cells-1), Tfh1 cells (follicular helper T cells-1), Tc1 cells (cytotoxic T cells-1), and Tregs (regulatory T cells) were lower ($p<0.01$, 0.05, 0.0001, and 0.001, respectively), but Th17 cells, Tfh17 cells, and Tc cells were higher ($p<0.001$, 0.0001 and 0.001, respectively). The proportions of total B cells and class-switched B cells were increased ($p<0.05$), but non-switched B cells, plasma cells, memory B cells, and immature Bregs (regulatory B cells) were lower ($p<0.01$, 0.0001, 0.0001, and 0.0001, respectively). After Anbainuo therapy, the percentage of Tregs and B10 cells (IL-10-producing regulatory B cells) had increased ($p<0.01$and 0.05, respectively), and the increase in Tregs was positively correlated with the decrease in CRP (C-reactive protein) ($r= 0.489$, $p=0.018$).

Conclusion: We found that, in terms of both innate and acquired immunity, active-stage AS patients have an immunity imbalance involving multiple types of immune cells, including CD4+T cells, CD8+T cells, Th cells, Tfh cells, Tc cells, Tregs, Bregs, and B cells. Anbainuo can not only help to inhibit disease activity and partial immune function imbalance in AS but can also increase the number of negative regulatory cells in inflammation.

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AB0050 EXTENDED POLYDIMENSIONAL IMMUNOME CHARACTERISATION (EPIC) PLATFORM AS A TOOL FOR TRANSLATIONAL RESEARCH

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Background: We created a high dimensionality healthy human Immunome atlas by interrogating the peripheral blood mononuclear cells (PBMC) of >200 healthy subjects (cord blood to adult) with 63 unique mechanistic and phenotypic markers per cell by mass cytometry (CyTOF). This database is built with an open source, web-based bioinformatics toolkit, enabling its mining and uploading of datasets for comparison with the EPIC healthy database.

Objectives: Here, we demonstrate the platform's ability to identify the immunological differences of mechanistically important cell subsets in the uploaded data in comparison with EPIC.

Methods: CyTOF data from 37 healthy elderly (>60 years old) was uploaded onto the EPIC Discovery tool where down-sampling, normalising and FlowSOM (Flow analysis with Self-Organising Maps) clustering were done with the EPIC database for comparison. Online visualisation outputs include cluster frequency boxplots, correspondence analysis (CA) plot and markers expression heat-map. The CA 2-dimensional plot depicts the global differences in immune cells composition between subjects with proximity between points (subjects) denoting similarity. Kruskal-Wallis test was done to identify age groups differences.

Results: Increasing distances on the CA plot with age were observed with the elderly being farthest from the new-borns. Notably, we observed significant changes in naive $CD4^+$ $IL8^+$ T cells ($p<1\times10^{-20}$), memory $CD4^+$ $IL17A^+$ T cells ($p<1\times10^{-20}$) and type 2 innate lymphoid cells (ILC2) (Lin^- $CD7^+$ $CD25^+$ $CD127^+$ $CD161^+$, $p<1\times10^{-17}$) with increasing age. The naive $CD4^+$ $IL8^+$ T cells (median: 0.68%, interquartile range: 0.415 to 1.055% of CD45+ PBMC) and ILC2 (0.09%, 0.065 to 0.12%) were lowest and memory $IL17A^+$ T cells (0.58%, 0.41 to 0.905%) highest in the elderly. Significantly, the memory $IL17A^+$ T cells and ILC2 have been implicated in the pathogenesis of auto-immune conditions[1,2].

Conclusion: With EPIC, we have created an online tool enabling data uploading for comparison to a healthy database, allowing the holistic characterisation of immunological changes in different clinical scenarios. Using it, we were able to identify mechanistically important differences in immune cells composition in a distinct clinical cohort (elderly) compared to the younger ages. Translationally, the EPIC platform can be utilised similarly to catalyse the discovery process in auto-immune diseases interrogated with the EPIC antibody panels.

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