from TNF
treatment than patients with an active T/NK-cell component. This proof-
of-concept study establishes a new framework to explore patient stratification in chronic inflammatory diseases according to the patients’ immune phenotype.

REFERENCE:

Disclosure of Interests: None declared

SP0056 CLINICAL MULTI-OMICS
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It is becoming clear that the field of medicine needs to change to stay affordable. One way of doing that is by optimizing the patients journey with true personalized medicine approaches. In other words, how to give every patient the right drug, at the right moment for the optimal duration. Recent advances in molecular techniques have truly revolutionized research into molecular fingerprinting – classification of clinical phenomena on the basis of a molecular basis rather than a diagnosis based on organ, symptoms or clinical criteria. This makes personalized medicine reachable within the coming years.

In my presentation I will dive in to the various molecular technologies (multi-omics) to achieve molecular fingerprinting of patients. Next, I will explain the computational techniques necessary to process the big data originating from the molecular processes enabling researchers to visualize big data from clinicians and patients.

Finally, I will provide several examples of how these exercises might improve care for patients covering the space of rheumatic and musculoskeletal diseases (RMDs).

At the end of my presentation I will show how big data can be used for drug discovery, immune monitoring in clinical trials and/or daily clinical practice as well as drug repurposing. Together, I will provide a glimpse of the future on personalized healthcare within the field of RMDs.

Disclosure of Interests: None declared

SP0058 GROWING UP CHANGES EVERYTHING!
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Background: Many disease susceptibility variants have been recently identified by genome wide association study (GWAS). Germline genetic variations exist before the disease onset and provide us with evidence into the causal relationship of the observed phenomenon and their pathogenesis. In this regards, the majority of GWAS risk variants have been found to function as an expression-quantitative trait locus (e-QTL).

Objectives: We need functional studies aiming at determining the causal genetic variants uncovered by GWAS and finding biological mechanisms underlying the observed statistical associations.

Methods: We established a system to evaluate various subtypes of leukocytes from peripheral blood mononuclear cells (PBMC) from healthy individuals. PBMC are separated with a fluorescence activated cell sorter and are analyzed in a steady state or activated conditions to capture the dynamic responses of gene regulation. Genotyping, RNA-seq, assay for transposase-accessible chromatin using sequencing (ATAC-seq) are performed on each subset. To further investigate the genetic determinants of promoter and enhancer activities, we also perform Cap Analysis of Gene Expression (CAGE) on the corresponding samples. CAGE is a technology developed in our research center RIKEN in Japan. It relies on random priming and cap-trapping of CDNAAs to capture the 5'ends of both polyadenylated and non-polyadenylated RNA transcripts. It thus enables genome-wide identification of transcription start sites (TSSs), which could be used as a portal to annotate and measure the activity of promoters and enhancers and IncRNAs active in each leukocyte subtype and correlate their activities with genetic variants.

Results: I will present our ongoing data.

REFERENCES: