from TNFi 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:

- [1] Duffy, D., et al. Functional Analysis via Standardized Whole-Blood Stimulation Systems Defines the Boundaries of a Healthy Immune Response to Complex Stimuli. *Immunity* 40, 436-450 (2014).

Disclosure of Interests: None declared

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SP0055

CLINICAL MULTI-OMICS

Timothy R. Radstake. *University Medical Center Utrecht, Netherlands*

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

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SP0056

MAPPING GENES TO IMMUNE CELLS

Kazuhiko Yamamoto. *RIKEN Center for Integrative Medical Sciences, Laboratory for Autoimmune Diseases, Yokohama, Japan*

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 promotor 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 cDNAs 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 (Carninci P. et al Science 2005, Forrest ARR. et al Nature 2014 and other publications of the FANTOM project). Using CAGE, we can map the promoters, enhancers and lncRNAs active in each leukocyte subtypes and correlate their activities with genetic variants.

Results: I will present our ongoing data.

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- [1] Okada Y, et al. Genetics of rheumatoid arthritis contributes to biology and drug discovery. *Nature* 2014;506:376-381.
[2] Ishigaki K, et al. Polygenic burdens on cell-specific pathways underlie the risk of rheumatoid arthritis. *Nat Genet* 2017;49:1120-1125.

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THURSDAY, 13 JUNE 2019

13:30:00 – 15:00:00

Adults are just grown up children! Discuss

SP0057

EVERYTHING STARTS IN CHILDHOOD!

Berent J. Prakken. *University Medical Center Utrecht, Biomedical education, Utrecht, Netherlands*

Background: While part of our future may be determined by our genes, the interaction of our immune system with the environment is crucial for the balance between health and disease. Most of these crucial interaction take place in the early years of life during which the developing immune system settles between self and non-self; danger and non danger or whatever paradigm you may want to apply. This early flexibility and early interactions between developing immunity and the environment hold the key for a health future; and ultimately will be the most effective way for true (very!) early prevention of chronic inflammatory disorders.

Disclosure of Interests: None declared

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SP0058

GROWING UP CHANGES EVERYTHING!

Iain McInnes. *University of Glasgow, Institute of Infection, Immunity, and Inflammation, Glasgow, United Kingdom*

Background: There is increasing interest in understanding the impact of age upon the emergence and clinical impact of immune mediated inflammatory diseases. In this lecture I will consider the similarities and distinctions between the presentation of inflammatory arthritis in children and adults.

Disclosure of Interests: Iain McInnes Grant/research support from: AstraZeneca, Celgene, Compugen, Novartis, Roche, UCB Pharma, Consultant for: AbbVie, Celgene, Galvani, Lilly, Novartis, Pfizer, UCB Pharma

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THURSDAY, 13 JUNE 2019

13:30:00 – 15:00:00

Exercise – more than a wonderdrug

SP0059

HELPING PEOPLE MAINTAIN PHYSICAL ACTIVITY

Keegan Knittle. *University of Helsinki, Faculty of Social Sciences, Helsinki, Finland*

Background: Regular physical activity (PA) has clear benefits for individuals with rheumatic diseases, but PA levels among these individuals consistently fall below recommended levels. While interventions to increase PA are often successful in the short term, these effects are not generally maintained over time. Therefore, to be able to optimally support PA maintenance, rheumatology health professionals should be familiar with the relevant theory and evidence base for behavioral maintenance.

Objectives: This talk will outline several psychological theories of behavioral maintenance, and present evidence on the interventions and behavior change techniques most strongly associated with physical activity maintenance.

Methods: A review of the most recent and relevant health psychology and behavioral science literature surrounding issues of physical activity maintenance, particularly among individuals with rheumatic conditions.

Results: Theories of behavioral maintenance focus on social and environmental influences, motives, resources, self-regulation, and habits. Several studies will be presented to highlight the influence of these theoretical domains in PA maintenance among individuals with rheumatic disease. Physical activity interventions which specifically target these theoretical domains have the greatest likelihood to produce PA maintenance, and several case studies from successful interventions will be presented.

Conclusions: While helping patients to maintain PA is always a challenge, especially in the face of degenerative or progressive arthropathies, behavioral science