Disclosure of Interests: Burkhard Moeller Consultant for: Swissmedic Human Medicines Expert Committee Member (regulatory agency), Patrick Durez Speakers bureau: Bristol-Myers Squibb, Eli Lilly, Sanofi, Celltrion, Axel Finckh Grant/research support from: Bristol-Myers Squibb, Pfizer Inc, Consultant for: AbbVie, A2Bio, Bristol-Myers Squibb, MSD, Roche, Pfizer Inc, and UCB, Pedro López-Romero Shareholder of: Eli Lilly and Company, Employee of: Eli Lilly and Company, Clementine Perrier Shareholder of: Eli Lilly and Company, Employee of: Eli Lilly and Company, Francesco de Leonards Shareholder of: Eli Lilly and Company, Employee of: Eli Lilly and Company, Inmaculada De La Torre Shareholder of: Eli Lilly and Company, Employee of: Eli Lilly and Company, and one immediately prior to or around disease diagnosis points and one currently unclear how achieving biological remission and individual patient reported symptom remission. It is currently unclear how achieving biological remission and individual patient treatment goals overlap and whether patients who reach the T2T target also reach their individual treatment goals.

Objectives: To explore if achieving T2T biological remission and individual patient treatment goals overlap in RA patients with initially low, moderate, or high disease activity (LDA, MDA, HDA). The treat-to-target (T2T) strategy has been established as a key concept in the management of patients with rheumatoid arthritis (RA) aiming to achieve remission or at least low disease activity [1]. However, it might be more important for patients to set individual treatment goals which are related to their specific life context. For this reason, Ferreira at al [2] recently proposed a dual T2T strategy including both biological remission and individual patient goals, which are related to their specific life context. For this reason, Ferreira at al [2] recently proposed a dual T2T strategy including both biological remission and individual patient reported symptom remission. It is currently unclear how achieving biological remission and individual patient treatment goals overlap and whether patients who reach the T2T target also reach their individual treatment goals.

Objectives: Identification of biomarkers that could classify patients 6-18 months before the clinical diagnosis of RA. However, there is a more limited understanding of the association with disease progression, specifically those that are upregulated in patients 6-18 months before the clinical diagnosis of RA.

Methods: We identified 500 subjects with RA, 500 with Reactive Arthritis (ReA) (based on ICD-9-CM code) as well as 250 age, gender and time-matched healthy control subjects from the Defense Medical Surveillance System (DMSS). For each subject, up to four serum samples were obtained from the Department of Defense (DoD) serum repository, 3 pre-disease diagnosis points and one immediately prior to or around disease diagnosis. A discovery subset of these serum samples was assessed for soluble PD-1 (sPD-1), as well as 497 protein analytes measured by SomaLogic SOMAscan proteomic platform.

Results: Serum levels of sPD-1 increased over time from pre-diagnosis to RA but trended to decrease over time in ReA subjects and healthy controls. A composite score of 24 SOMAscan analytes associated with recently diagnosed RA increased in serum 6-8 months before RA diagnosis and, to a lesser extent, before ReA diagnosis. IFN-inducible chemokinases (CXCL10 (IP-10), CXCL9 (MIG), CXCL12 (I-TAC), and CXCL13 (BLC)) increased over time preceding RA but not ReA diagnosis. Acute phase proteins (CRP, SAA, haptoglobin) and MMP-3 increased before diagnosis in serum samples from both RA and ReA patients. These protein analytes represent potential new biomarkers of early RA.

Conclusion: Samples from the US DoD serum repository have identified novel biomarkers (sPD-1 and IFN-inducible chemokines) of early RA that are elevated within 8 months of disease diagnosis. These protein analyses may afford the opportunity to develop novel biomarker(s) for diagnosis and disease progression to early RA. An understanding of the role of these proteins could provide increased insight into RA pathogenesis prior to disease diagnosis.