Career situation of first and presenting author Post-doctoral fellow.

**Introduction** RA is a heterogenous disease and there is a substantial evidence to indicate the contribution of type I interferon (IFN-I) in 20%-40% of RA patients with possibly more local IFNβ role; compared to systemic IFN-α action in SLE. Patients with RA have an increased risk of cardiovascular disease (CVD) equivalent to type 2 diabetes, predominantly driven by excess atherosclerosis (ATS). Pre-clinical and human data suggest IFN-I plays a key role in the development of CVD in RA.

**Patients with RA** have an increased risk of cardiovascular disease (CVD) in EstRA as well as in HC n=71. Next RA patients were stratified for CVD using multi-parametric cardiac MRI (CMR) evaluation that included aortic distensibility (vascular stiffness), LV mass/BSA (LV geometry) and Myocardial T1 (indicating myocardial fibrosis) as follows: (i) ‘ERA-no CVD’ n=37 (no abnormalities on CMR), (ii) ‘ERA-sub CVD’ n=37 (at least one of the three parameters abnormal), (iii) ‘EstRA-no CVD’, n=14 (iv) ‘EstRA-sub CVD’, n=54 (v) ‘RA-CVD’, n=32 (defined as per ‘Major Adverse Cardiovascular Event’ (MACE)).

**Results** We confirmed a higher IFN score B than A in RA patients, similar to the observation made in the original paper that developed the scoring system. Significantly higher IFN score A and B in was observed in ERA than in EstRA and HCs. There was no association between IFN scores and markers of inflammation. Increase in expression of IFN score B was observed across a CVD continuum (i.e. from RA-no CVD to RA-sub CVD to RA-CVD) in EstRA but not in ERA.

**Conclusions** IFN-I may play a particularly important pathological role at time of development of disease (ERA). CVD stratification suggests that genes included in IFN Scores A and B may be implicated in the progression along a CVD continuum; and appearing to associate with a pro-atherogenic role. This observation only in EstRA may reflect CVD burden over time. If confirmed, these data imply multiple organ specificities for the IFN scores. Further work is planned to interrogate IFN status in RA-CVD, towards improved risk stratification and tailored management of CVD co-morbidity.

**REFERENCE**


**Disclosure of Interest** None declared.