Response to: 'Neutrophil extracellular traps and low-density granulocytes are associated with the interferon signature in systemic lupus erythematosus, but not in antiphospholipid syndrome' by van den Hoogen *et al*

We very much appreciate the interest of van den Hoogen *et al*[1] in our study.[2] Their independent confirmation of the linkage between the type I interferon (IFN) axis, disease activity, hypocomplementaemia and elevated low-density granulocyte (LDG) numbers in patients with systemic lupus erythematosus (SLE) is highly encouraging for the field considering the demographic differences between the Utrecht and National Institutes of Health cohorts.

In light of these aligned observations, it is intriguing that the authors did not find any significant association between the type 1 IFN gene signature and neutrophil extracellular trap release or LDG numbers in their patients with either primary or SLE-associated antiphospholipid syndrome (APS), despite observing increased prevalence of each component.[3] We look forward with great interest to future studies that may elucidate whether this observed uncoupling in APS is due to the presence of a discrete activatory stimulus driving the formation and activation of LDG or whether the type I IFN pathway may still be at play but not as readily observable by systemic gene signature as a result of local microenvironment constriction or the ability of standard-of-care medications to mask IFN gene signature detection.

Notably, we fully support the sentiments of van den Hoogen *et al*[1] and agree that the role of LDG in rheumatological disease is an emerging field deserving of continued exploration across populations and indications.

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