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. 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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Handling editor Josef S Smolen

Contributors Both authors were involved in the development, review and approval of the letter on behalf of all coauthors. Editorial support was paid for by AstraZeneca.

Funding This study was supported by Medlmmune, a member of the AstraZeneca Group (AR041199) and by the intramural research program at NIAMS/NIH.

Competing interests KAC was an employee at MedImmune during the time work was performed on this study.

Patient consent for publication Not required.

Provenance and peer review Commissioned; internally peer reviewed.

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To cite Kaplan MJ, Casey KA. *Ann Rheum Dis* Epub ahead of print: [please include Day Month Year]. doi:10.1136/annrheumdis-2019-215811

Received 13 June 2019 Accepted 15 June 2019



► http://dx.doi.org/10.1136/annrheumdis-2019-215781

Ann Rheum Dis 2019;0:1. doi:10.1136/annrheumdis-2019-215811

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