Response to: 'Risk of systemic lupus erythematosus in patients with idiopathic thrombocytopenic purpura: population-based cohort study' by Goulielmos and Zervou

We thank Goulielmos *et al*¹ for their interests on our article entitled 'Risk of systemic lupus erythematosus (SLE) in patients with idiopathic thrombocytopenic purpura (ITP): a population-based cohort study'.²

Goulielmos *et al* raised possible mechanism and explanation about the link of ITP and SLE. We appreciated their review and comments on sensitised platelets, shared genetic background and similar molecular signatures of these two diseases. We also agree that these genetic and molecular background, especially interferon signatures in ITP might lead to development autoimmune diseases, such as SLE.

In this study, we use a big data approach to explore the linkage of ITP and SLE and demonstrated strong association of these two diseases. It is not our purpose to look at the mechanism of this association. We appreciate those creative comments and hypothesis that Goulielmos *et al* proposed. A recent study also found active role of platelet activation in pathogenesis of SLE.³ It definitely need further bench works to prove above hypothesised mechanism.

One possible linkage which Goulielmos *et al* did not mention is the environmental factors, especially infection. Besides well-known virus, such as Epstein-Barr virus, cytomegalovirus and parvovirus B19, in our previous studies, we also found that SLE is associated various micro-organisms, including human Papillomavirus, scrub typhus, *Helicobacter pylori*, *Mycoplasma pneumonia* and non-typhoid Salmonella (data in submission). We think that SLE and other autoimmune diseases all had strong interaction with genetic and environmental factors.

In conclusion, we agree that SLE is a complex disease spectrum with various phenotypes. ITP and certain subtypes of SLE might share molecular and genetic backgrounds. Clinician should recognise ITP might be a subtype of subclinical SLE in managing patients.

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Handling editor Josef S Smolen

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Funding Funding The present study was supported by the Programme of Scientific and Technology Project (Guilin Science Research and Technology Development; grant no. 2016012706–2).

Competing interests None declared.

Patient and public involvement Patients and/or the public were not involved in the design, or conduct, or reporting, or dissemination plans of this research.

Patient consent for publication Not required.

Provenance and peer review Commissioned; internally peer reviewed.

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To cite Zhu FX, Huang J-Y, Wei JC-C. Ann Rheum Dis 2022;81:e113.

Received 24 June 2020 Accepted 25 June 2020 Published Online First 22 July 2020



► http://dx.doi.org/10.1136/annrheumdis-2020-218128

Ann Rheum Dis 2022;81:e113. doi:10.1136/annrheumdis-2020-218177

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