NCF1-339 polymorphism and systemic lupus erythematosus

We read the publication on 'NCF1-339 polymorphism is associated with altered formation of neutrophil extracellular traps, high serum interferon activity and antiphospholipid syndrome (APS) in systemic lupus erythematosus (SLE)' with great interest.¹ Linge *et al* concluded that "we revealed a striking connection between the ROS deficient NCF1-339 genotypes and the presence of phospholipid antibodies and APS".¹ The NCF1-339 rs201802880 polymorphism is a single mutation that might cause the molecular change. Due to the mutation, the molecular weight change and the alteration of the phenotypic expression is the result. In the present report, Linge *et al* studied on only one genetic polymorphism and did not assess the possible effects of other genetic polymorphisms that might have clinical association with SLE. The examples of those genetic polymorphisms are paraoxonase-1 gene and PAL-1 polymorphisms.^{2 3} Further studies to access the possible confounding factors of other genetic polymorphisms are required.

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Competing interests None declared.

Patient consent for publication Not required.

Provenance and peer review Not commissioned; internally peer reviewed.

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To cite Joob B, Wiwanitkit V. *Ann Rheum Dis* Epub ahead of print: [please include Day Month Year]. doi:10.1136/annrheumdis-2019-216629

Received 11 November 2019

Accepted 17 November 2019

Ann Rheum Dis 2019;**0**:1. doi:10.1136/annrheumdis-2019-216629

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