

CORRECTION



Correction: An immunogenomic exome landscape of triple positive primary antiphospholipid patients

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Correction to: *Genes & Immunity* <https://doi.org/10.1038/s41435-024-00255-w>, published online 24 January 2024

In this article the affiliation details for A. Cobat and B. Boisson were incorrectly given as:

A. Cobat⁴, B. Boisson^{4,5},

but should have been:

A. Cobat^{4,5,13}, B. Boisson^{4,5,8,13},

Two duplicate sentences have been deleted from the original abstract, which now reads as follows:

Primary antiphospholipid syndrome is characterized by thrombosis and autoantibodies directed against phospholipids or associated proteins. The genetic etiology of PAPS remains unknown. We enrolled 21 patients with thromboembolic events associated

to lupus anticoagulant, anticardiolipin and anti β 2 glycoprotein1 autoantibodies. We performed whole exome sequencing and a systematic variant-based analysis in genes associated with thrombosis, in candidate genes previously associated with APS or inborn errors of immunity. Data were compared to public databases and to a control cohort of 873 non-autoimmune patients. Variants were identified following a state-of-the-art pipeline. Enrichment analysis was performed by comparing with the control cohort. We found an absence of significant HLA bias and genetic heterogeneity in these patients, including when testing combinations of rare variants in genes encoding for proteins involved in thrombosis and of variants in genes linked with inborn errors of immunity. These results provide evidence of genetic heterogeneity in PAPS, even in a homogenous series of triple-positive patients. At the individual scale, a combination of variants may participate to the breakdown of B cell tolerance and to the vessel damage.

The original article has been corrected.