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Response to: “A combination of complement activation products with autoantibodies predicts transition of probable lupus to systemic lupus erythematosus”

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Dear editor,

We thank Dr Alexander and Dr Weinstein for their response to our review.¹ We welcome the attempt to develop a diagnostic test that can predict progression to systemic lupus erythematosus (SLE). Early identification of SLE is one of the main challenges faced by clinicians, and is hindered by both the heterogeneous presenting symptoms of the disease, as well as overlap with other diseases.^{2,3} Timely treatment, however, is important in order to limit disease progression, and prevent organ damage and mortality.⁴

In the study cited, patients with incomplete (or probable) SLE - defined as suspected of SLE by lupus experts and fulfilling 3 ACR classification criteria for SLE - were included in a follow up study.⁵ There were 68 subjects that underwent Multiple Analyte Panel (MAP) testing, which combined cell bound complement activation products and several auto-antibodies. The optimal cutoff was > 0.8, based on the Youden index. A MAP > 0.8 showed stronger predictive potential than individual biomarkers, including serum complement levels, and various antinuclear antibodies. In total, 16 patients (24%) had a MAP > 0.8 at baseline. After 9-18 months of follow up, 20/68 (29%) developed SLE. Of the 20 patients with incomplete SLE who developed SLE, 8 patients had at baseline a MAP > 0.8. However, the majority of future SLE patients (namely 12 out of 20, or 60%), were not detected by this test. So, although this test, containing cell bound complement activation, indeed seems to have more predictive potential than the current diagnostic markers, there is still need for improvement of the predictive potential of the diagnostic arsenal in incomplete SLE. Previous research showed that interferon (IFN)-inducible gene expression was higher in patients at risk of SLE who later developed SLE than in those who had stable disease.⁶ Therefore, it would be interesting to combine this MAP with IFN-inducible gene expression or IFN-related mediators in future prospective studies on incomplete SLE.

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