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Lupus Science and Medicine: the Editors present highlights for the bedside and for the bench in the inaugural issue

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In clinical medicine, we seek to heal, and it is a long held tenet that a 'response' to treatment should be congruent between a strictly defined objective outcome and the physician's perception of improvement. As we advance in our understanding of the pathogenesis of lupus and molecularly targeted therapies are being studied, pivotal FDA trials are relying on two major indices to gauge response in extra-renal activity; the BILAG-based Composite Lupus Assessment (BICLA), and the Systemic Lupus Responder Index (SRI). Accordingly, it is timely that Thanou *et al*[1] address the critical issue of whether these instruments are faithfully reflective of what the clinician 'really thinks'. The authors point out that both instruments have shortcomings. BICLA requires partial improvement but in all organs, while the SRI requires full improvement in some manifestation(s) but not in all. Evaluating 91 patients on at least two visits in which the BILAG and SLEDAI were scored, and the patients had active disease at the first visit, efficacy, as denoted by the instrument was in accord with the impression of the physicians. Perhaps not unexpectedly, patients rated as improving by the physician may not have met the SRI response criteria because they did not achieve a less than 4-point improvement in SLEDAI. Importantly, this study affirms that our instruments do not identify response when the physician has not. But…there may be room to improve since overly stringent medication restrictions may result in an instrument falling short of response when the physician observes improvement.

Soriano *et al*[2] studied the utility of repeat renal biopsies in patients with SLE. Whereas a diagnostic renal biopsy is widely used to establish the diagnosis and ascertain the nature and severity of lupus nephritis, it has not been equally clear whether there is sufficient clinical utility to warrant the risks and costs of a repeated renal biopsy after suitable treatment has been in place for some time. The contribution by Soriano *et al* provides very useful information on this issue.

Draborg *et al*[3] analysed specific T-cell-mediated immune responses to Epstein–Barr virus (EBV). Different lines of research have previously implicated this elusive virus as a potentially important cofactor in the complex aetiology of SLE, and the study reported here adds important insights into the nature of immunological reactivities involved in host-defence against EBV in patients with lupus.

Compagno *et al*[4] revisited one of the most time-tested laboratory markers of SLE: the anti-DNA antibody test. Several laboratory methods are available for measuring such antibodies, and all come with some strengths and weaknesses. Here, the authors present a detailed examination of the relationships between the different types of tests and the clinical manifestations of SLE in each patient.

Manson *et al*[5] studied the effects of serum from patients with lupus nephritis on cultured human podocytes, cells of major importance in maintaining the integrity of the glomerulus, and found that such, sera as well as their IgG, significantly decreased tyrosine phosphorylation of podocyte proteins including tubulin. These results point towards a potential mechanism by which IgG (auto-)antibodies may cause diminished podocyte function and, thereby, allow proteinuria and the nephrotic syndrome.

Competing interests None.

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