Response to: ‘Belimumab and the measurement of fatigue’ by Mazzoni

We agree that fatigue is a common and important symptom of patients with active systemic lupus erythematosus (SLE), and that it is multidimensional, as has been elegantly described in patients with rheumatoid arthritis.1 In fact, randomised controlled trials in SLE have assessed fatigue, as early as the prasterone dehydroepiandrosterone (DHEA) studies (published in 2002–2004) that employed the (Krupp) Fatigue Severity Scale (FSS), developed for SLE and multiple sclerosis.2 3

We agree that belimumab treatment does reduce fatigue, particularly in Systemic Lupus Erythematosus Responder Index (SRI) responders as reported in a more recent publication. In a combined analysis of BLISS-52 and BLISS-76, SRI responders, across all three treatment groups, reported statistically significant and clinically meaningful improvements compared with non-responders in Functional Assessment of Chronic Illness Severity (FACIT), SF-36 summary and all domain scores.4 That neither FACIT nor SF-36 data were statistically significant at the predefined 24-week time point is likely due to the fact that the protocol allowed increases in glucocorticoid doses until 24 weeks and immunosuppressive/antimalarial agents up to 16 weeks following baseline—thereby ‘rescuing’ placebo patients.

FACIT is a unidimensional scale and has been validated in SLE across many studies.5–8 The belimumab data that you are querying were analysed in 2010–2011, presented to the Food and Drug Administration Arthritis Advisory Committee in November 2011 and subsequently published.6 9 To our knowledge, the second Cella paper had not yet been published when these detailed analyses were completed. It is an interesting idea to segregate items of the FACIT into experience and impact scores; however, this would necessarily require validation in SLE as well as ‘qualification’ that this is relevant and clinically meaningful to patients with SLE. Unfortunately, the sponsorship of this database has changed (from HGS to GSK) and much time has passed, and we are unable to undertake further analyses to address your question.

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Competing interests None declared.
Provenance and peer review Commissioned; internally peer reviewed.

Received 29 April 2015
Accepted 1 May 2015
Published Online First 19 May 2015

http://dx.doi.org/10.1136/annrheumdis-2015-207693


REFERENCES