Dr. Matteson, *et al* reply

To the Editor:

Dr. Amezcua-Guerra makes the point that it is overly simplistic to use the erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) as measures of the inflammatory response. We concur with this assessment. Since their introduction in the 1920s, the ESR and CRP have served as measures, surrogates, and markers of disease activity for autoinflammatory diseases. Their limitations in terms of sensitivity and specificity have long been recognized. As the biology of inflammation has become better understood, it has also become clear why these markers are insufficient as global markers of disease activity in conditions such as rheumatoid arthritis, systemic lupus erythematosus, and even (and especially after initial treatment response) in polymyalgia rheumatica and giant cell arteritis, to mention but a few[1,2]. While it is too early to proclaim their death, their utility for many applications in the clinic and, for example, clinical trials in rheumatoid arthritis, is marginal at best[2]. At this time, however, these are the only biomarkers of inflammation widely used in clinical practice and accepted by major regulatory authorities (US Food and Drug Administration, European Medicines Evaluation Agency) and are part of the validated, well established measures of disease activity in the American College of Rheumatology and Disease Activity Score (European League Against Rheumatism) response criteria. Doubtless, the development of more disease-specific markers of activity and damage should result in their clinical adoption and a decline in the use of these older markers.

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