# Bristol Myers Squibb
# Independent Medical Education
## Request for Educational Support (RFE)

| | |
|---|---|
| **Date** | May 5, 2026 |
| **RFE Requestor Information** | Name: Sylvia Nashed, PharmD, RPh<br>Title: Medical Education Lead<br>E-mail: Sylvia.Nashed@bms.com |
| **RFE Code** | RFE-26-IM-102 |
| **Therapeutic Area** | Immunology |
| **Area of Interest** | Systemic Lupus Erythematosus<br><br>It is our intent to support a comprehensive, innovative, and engaging initiative that enhances understanding of SLE pathophysiology, addresses unmet medical needs—including challenge of achieving sustained disease control with minimized corticosteroid use—and aligns emerging mechanistic insights with current and evolving clinical trial evidence within a treat-to-target framework. |
| **Educational Design** | Bristol Myers Squibb is interested in supporting an innovative, engaging and comprehensive educational initiative that includes the following:<br>• Live hybrid (in-person/virtual) satellite symposium at ACR Convergence 2026 (November 6 – 11, 2026)<br>• Simultaneous live streaming on social media platforms during the meeting<br>• On-demand web-based enduring activity leveraging the medical content from the live meeting<br><br>Proposals that offer online resources and tools that will further aid rheumatologists in their practice will be given higher priority.<br><br>The activity(ies) should measure improvement of learners' knowledge, confidence, competence, and performance and should achieve at least a Moore's Level 4 impact. Activities that achieve Moore's Levels 5 and 6 outcomes are highly favored and recommended when possible.<br>A successful proposal should include: |

| | |
|---|---|
| | • Clear and concise statement of the goal, learning objectives, and expected outcomes of the educational initiative<br>• Instructional design that incorporates innovative techniques designed to engage learners, promotes application of education into practice, and incorporates a patient-centered approach to care<br>• Tools that provide HCP learners the opportunity to facilitate change to improve patient outcomes and address healthcare inequities<br>• Measurement of outcomes, inclusive of learner progression throughout the activity, extent to which the activity closed the identified practice gaps, and patient impact |
| **Intended Audience (may include, but not limited to)** | Community and Academic Rheumatologists |
| **Budget/Budget Range** | The maximum amount of funding available for this RFE is $375,000.<br><br>Single or multi-supported initiatives will be considered. |
| **Accreditation** | ACCME |
| **Deadline for Submission** | June 1, 2026, by 5 PM EST |

## Background

Systemic lupus erythematosus (SLE) is a chronic, heterogeneous autoimmune disease characterized by loss of immune tolerance, production of autoantibodies, immune complex deposition, and multisystem organ involvement. The disease disproportionately affects women of childbearing age and individuals from racial and ethnic minority populations, contributing to disparities in disease burden, severity, and long-term outcomes.[1-3] Despite advances in the understanding of disease mechanisms, SLE remains associated with substantial morbidity, impaired quality of life, and progressive, irreversible organ damage over time.[4,5]

SLE pathogenesis is driven by complex dysregulation of innate and adaptive immune responses, with a central role for interferon (IFN)–mediated signaling. Sustained activation of type I IFN pathways promotes dendritic cell activation, B-cell differentiation, autoantibody production, and amplification of inflammatory cascades that contribute to multisystem disease manifestations.[6-8] Several cytokines implicated in SLE pathophysiology, including type I IFNs as well as interleukin (IL)-12 and IL-23, signal through intracellular Janus kinase pathways in which tyrosine kinase 2 (TYK2) plays a key regulatory role.[9-11] Dysregulation of TYK2-mediated signaling has been linked to persistent immune activation and inflammatory disease states, providing rationale for targeted therapeutic approaches that more selectively modulate cytokine signaling pathways compared with conventional broad immunosuppressive strategies.[12,13]

Despite advances in understanding SLE pathogenesis, many patients with SLE continue to experience persistent undertreatment, including delayed escalation of therapy, incomplete suppression of disease activity, and recurrent flares.[14,15] Ongoing disease activity and repeated inflammatory insults are strongly associated with cumulative and irreversible organ damage, leading to worsened long-term outcomes.[16,17] A major contributor to treatment-related harm remains prolonged exposure to systemic corticosteroids, which are widely used for rapid disease control but are associated with dose-dependent risks of infection, cardiovascular disease, osteoporosis, metabolic complications, and damage accrual.[18-20] Consequently, minimizing long-term corticosteroid use while maintaining adequate disease control has emerged as a critical unmet need in SLE management.

Treat-to-target (T2T) strategies have been proposed to address these challenges by emphasizing early and sustained disease control defined by achievement of low disease activity or remission and subsequent tapering and complete discontinuation of oral corticosteroids.[21,22] Validated clinical targets—including lupus low disease activity state (LLDAS) and consensus remission definitions such as DORIS—have demonstrated strong associations with reduced flare frequency and lower rates of organ damage accrual and incorporate explicit thresholds for low-dose or withdrawn corticosteroid use.[23-26] However, implementation of T2T principles in routine clinical practice has been inconsistent, in part due to disease heterogeneity, limitations of existing therapies, and challenges in achieving meaningful reductions in corticosteroid use while sustaining disease control.

Ongoing clinical trial and long-term extension data have increasingly focused on outcomes relevant to these unmet needs, including the achievement and durability of low disease activity and remission, particularly early in disease course, and the feasibility of steroid-sparing treatment strategies in SLE.[25,27-38] Interpretation of these data within the context of T2T frameworks is essential to inform evidence-based decision-making and optimize long-term management approaches. Independent medical education that integrates evolving understanding of SLE pathogenesis, recognition of undertreatment and steroid-related harm, application of T2T principles, and interpretation of clinical and long-term evidence is needed to support improved patient outcomes.

## Educational Needs and Professional Practice Gaps:

BMS has identified, through insights from educational needs assessments, literature search, learning outcomes, and other methods, the need to address the following existing professional practice gaps:

- Describe SLE pathogenesis and interferon signaling and explain how targeted therapies differ from conventional immunosuppressants in supporting disease control.
- Recognize the impact of persistent undertreatment in SLE, including delayed disease control and the risk of cumulative and irreversible organ damage, as well as limitations of current treatment approaches.
- Explain key principles of treat-to-target (T2T) management in SLE, including the importance of early and sustained disease control and minimization of long-term corticosteroid exposure to improve patient outcomes.
- Interpret ongoing clinical trial and long-term data in SLE, including durability of low disease activity or remission (eg, LLDAS, DORIS) and data informing steroid-sparing strategies that may better support T2T-aligned care.

The content and/or the format of the CME/CE activity and its related materials must be current and designed in such a way that it addresses the educational needs of the intended audiences as described in this RFE.

*Grant Proposals should include, but not be limited to, the following information:*

- **Executive Summary: The Executive Summary should consist of 1-2 pages and highlight the key areas as described below.**

- **Needs Assessment/Gaps/Barriers:** Needs assessment should be referenced and demonstrate an understanding of the specific gaps and barriers of the target audiences. The needs assessment must be independently developed and validated by the educational provider through triangulation.

- **Target Audience and Audience Generation:** Target audience for educational program must be identified within the proposal. In addition, please describe methods for reaching target audience(s) and any unique recruitment methods that will be utilized. The anticipated or estimated participant reach should also be included, with a breakdown for each modality included in the proposal, as applicable (e.g., number of participants for the live activity, the live webcast, and enduring activity).

- **Learning Objectives:** The learning objectives must be written in terms of what the learner will achieve as a result of attending. The objectives must be clearly defined, measurable, attainable, and address the identified gaps and barriers.

- **Program Evaluation and Outcomes Reporting:** Description of the approach to evaluate the quality of the educational program. Describe methods used for determining the impact of the educational program on closing identified healthcare gaps.
    - Please refer to "Guidance for Outcomes Report" (on the BMS grants website) for a detailed explanation of preferred outcomes reporting methods and timelines.
    - Remember that knowledge, performance and competency-based outcome measures according to Moore's Level 4 are required. Levels 5 and 6 outcomes are highly favored and recommended when possible.

- **Educational Design and Methods:** Describe the approach used to address knowledge, competence, and performance gaps that underlie identified healthcare gaps. The proposal should include strategies that ensure reinforcement of learning through use of multiple educational interventions and include practice resources and tools, as applicable.

- **Communication and Publication Plan:** Provide a description of how the provider will communicate the progress and outcomes of the educational program to the supporter. It is highly recommended to describe how the results of the activity will be presented, published, or disseminated.

- **Innovation:** Describe how this project is innovative and engages the learners to

improve knowledge, competence and/or performance. Further describe how this project might build on existing work, pilot projects or ongoing projects developed either by your institution or other institutions related to this topic.

- **Budget:** Detailed budget with rationale of expenses, including breakdown of costs, content cost per activity, out-of-pocket cost per activity, and management cost per activity.

**Note:** The accredited provider and, if applicable, the medical education partner (MEP) or other third party executing the activities, are expected to comply with current ethical codes and regulations. They must have a conflict-of-interest policy in place to identify and resolve all conflicts of interest from all contributors and staff involved in developing the content of the activity prior to delivery of the program and must have a separate company providing/accrediting independent medical education if they are also performing promotional activities.

*If your organization wishes to submit an educational grant request, please use the online application available on the Bristol Myers Squibb Independent Medical Education website. http://www.bms.com/responsibility/grantsandgiving*

References


1. Carter EE, Barr SG, Clarke AE. The global burden of systemic lupus erythematosus: prevalence, health disparities and socioeconomic impact. *Nat Rev Rheumatol*. 2016;12(10):605–620. doi:10.1038/nrrheum.2016.137.
2. Izmirly PM, Wan I, Sahl S, et al. Prevalence of systemic lupus erythematosus in the United States: updated estimates from national claims data. *Lupus Sci Med*. 2021;8:e000614. doi:10.1136/lupus-2021-000614.
3. Limaye MA, Wilson H, Drenkard C, et al. Socioeconomic disparities in systemic lupus erythematosus outcomes: current evidence and future directions. *Prenat Diagn*. 2020;40(9):1066–1076. doi:10.1002/pd.5710.
4. Urowitz MB, Gladman DD, Ibanez D, et al. Accrual of organ damage in systemic lupus erythematosus over time. *Arthritis Rheumatol*. 2014;66(1):169–178. doi:10.1002/art.38295.
5. Fortune C, Bruce IN, Hanly JG, et al. Health-related quality of life in patients with systemic lupus erythematosus: a systematic review. *Rheumatology (Oxford)*. 2021;60(3):1425–1434. doi:10.1093/rheumatology/keaa051.
6. Rönnblom L, Leonard D. Interferon pathway in SLE: one key to unlocking the mystery of the disease. *Lupus Sci Med*. 2019;6(1):e000270. doi:10.1136/lupus-2018-000270.
7. Pisetsky DS. The role of innate immunity in the pathogenesis of systemic lupus erythematosus. *J Autoimmun*. 2020;110:102356. doi:10.1016/j.jaut.2020.102356.
8. Goel RR, Kaplan MJ. Deadly partnerships: autoreactive B cells and T cells in systemic lupus erythematosus. *Nat Rev Rheumatol*. 2021;17(6):349–360. doi:10.1038/s41584-021-00606-1.
9. Ghoreschi K, Laurence A, O'Shea JJ. Janus kinases in immune cell signaling. *Immunol Rev*. 2009;228(1):273–287. doi:10.1111/j.1600-065X.2008.00754.x.
10. Baker KF, Isaacs JD. Novel therapies for immune-mediated inflammatory diseases: what can we learn from Janus kinase inhibition? *Ann Rheum Dis*. 2018;77(2):175–187. doi:10.1136/annrheumdis-2017-212988.

11. Rusiñol L, Puig L. Understanding TYK2 signaling in immune-mediated inflammatory diseases. *Int J Mol Sci*. 2023;24(4):3391. doi:10.3390/ijms24043391.
12. Dörner T, Furie R. Novel paradigms in systemic lupus erythematosus. *Lancet*. 2019;393(10188):2344–2358. doi:10.1016/S0140-6736(19)30546-X.
13. Winthrop KL, Harigai M, Genovese MC, et al. The emerging safety profile of JAK inhibitors in rheumatic disease. *Nat Rev Rheumatol*. 2017;13(4):234–243. doi:10.1038/nrrheum.2017.23.
14. Wallace DJ, Navarra SV, Petri MA, et al. Barriers to optimal management of systemic lupus erythematosus in clinical practice. *Lupus Sci Med*. 2019;6(1):e000310. doi:10.1136/lupus-2018-000310.
15. Fanouriakis A, Bertsias G, Boumpas DT, et al. Update of the EULAR recommendations for the management of systemic lupus erythematosus. *Ann Rheum Dis*. 2019;78(6):736–745. doi:10.1136/annrheumdis-2019-215089.
16. Franklyn K, Lau CS, Navarra SV, et al. Definition and initial validation of a lupus low disease activity state (LLDAS). *Ann Rheum Dis*. 2016;75(9):1615–1621. doi:10.1136/annrheumdis-2015-207726.
17. Golder V, Kandane-Rathnayake R, Hoi AY, et al. Lupus low disease activity state and damage accrual: a multicentre cohort study. *Arthritis Rheumatol*. 2019;71(7):114–124. doi:10.1002/art.40754.
18. Ruiz-Irastorza G, Danza A, Khamashta M, et al. Glucocorticoids in systemic lupus erythematosus: conclusions from a systematic review. *Ann Rheum Dis*. 2010;69(1):20–28. doi:10.1136/ard.2008.102681.
19. Al Sawah S, Zhang X, Zhu B, et al. Risk factors for organ damage in systemic lupus erythematosus. *Lupus Sci Med*. 2015;2(1):e000066. doi:10.1136/lupus-2014-000066.
20. Strehl C, Bijlsma JW, de Wit M, et al. Glucocorticoid-induced immunosuppression: mechanisms and management. *Nat Rev Rheumatol*. 2016;12(3):133–143. doi:10.1038/nrrheum.2015.167.
21. van Vollenhoven RF, Mosca M, Bertsias G, et al. Treat-to-target in systemic lupus erythematosus: recommendations from an international task force. *Ann Rheum Dis*. 2014;73(6):958–967. doi:10.1136/annrheumdis-2013-205139.
22. Aringer M, Johnson SR. Treat-to-target in systemic lupus erythematosus: development and implementation. *Rheumatology (Oxford)*. 2020;59(Suppl 5):v4–v11. doi:10.1093/rheumatology/keaa438.
23. van Vollenhoven RF, Doria A, Zen M, et al. DORIS definition of remission in systemic lupus erythematosus. *Lupus Sci Med*. 2021;8(1):e000538. doi:10.1136/lupus-2021-000538.
24. Tani C, Vagelli R, Stagnaro C, et al. Remission and low disease activity in systemic lupus erythematosus: clinical relevance. *Ann Rheum Dis*. 2018;77(10):1402–1408. doi:10.1136/annrheumdis-2018-213509.
25. Morand EF, Golder V, Abreu G, et al. Attainment and duration of low disease activity and remission in SLE clinical trials. *Arthritis Rheumatol*. 2023;75(2):242–252. doi:10.1002/art.42303.
26. Golder V, Kandane-Rathnayake R, Lai K, et al. Achieving low disease activity is associated with reduced damage accrual in SLE. *Lancet Rheumatol*. 2019;1(2):e95–e102. doi:10.1016/S2665-9913(19)30004-0.
27. Arriens C, Morand EF, van Vollenhoven RF, et al. Integrated efficacy and long-term outcomes from the PAISLEY systemic lupus erythematosus program: pooled phase 2 and long-term extension analyses. *Annals of the Rheumatic Diseases*. 2026;85(Suppl 1). Abstract presented at: EULAR European Congress of Rheumatology 2026.

28. Morand EF, van Vollenhoven RF, Furie RA, et al. LLDAS and remission attainment with anifrolumab treatment in patients with systemic lupus erythematosus: results from the TULIP and long-term extension randomised controlled trials. *Annals of the Rheumatic Diseases*. 2025;84(5):777–788. doi:10.1136/ard-2024-225071.
29. Sciascia S, Foddai SG, Arbrile M, et al. Assessing the steroid-sparing effect of biological agents in randomized controlled trials for lupus: a scoping review. *Immunologic Research*. 2024;72(4):538–553. doi:10.1007/s12026-024-09463-y
30. Gatto M, et al. New therapeutic strategies in systemic lupus erythematosus management. *Autoimmunity Reviews*. 2024;23:103612. doi:10.1016/j.autrev.2024.103612.
31. Kojima K, Ichinose K, Umeda M, et al. Enhancing systemic lupus erythematosus treatment outcomes with an early initiation of belimumab: insights from a multicenter retrospective study within the first five years. *Arthritis Research & Therapy*. 2025;27:116. doi:10.1186/s13075-025-03581-0.
32. Gatto M, Saccon F, Zen M, et al. Early disease and low baseline damage as predictors of response to belimumab in patients with systemic lupus erythematosus in a real-life setting. *Arthritis & Rheumatology*. 2020;72(8):1314–1324. doi:10.1002/art.41253.
33. Furie R, Petri M, Zamani O, et al.; BLISS-76 Study Group. A phase III, randomized, placebo-controlled study of belimumab, a monoclonal antibody that inhibits B-lymphocyte stimulator, in patients with systemic lupus erythematosus. *Arthritis & Rheumatism*. 2011;63(12):3918–3930. doi:10.1002/art.30613.
34. Navarra SV, Guzmán RM, Gallacher AE, et al.; BLISS-52 Study Group. Efficacy and safety of belimumab in patients with active systemic lupus erythematosus: a randomised, placebo-controlled, phase 3 trial. *The Lancet*. 2011;377(9767):721–731. doi:10.1016/S0140-6736(10)61354-2.
35. Morand EF, Furie R, Tanaka Y, et al.; TULIP-2 Trial Investigators. Trial of anifrolumab in active systemic lupus erythematosus. *New England Journal of Medicine*. 2020;382(3):211–221. doi:10.1056/NEJMoa1912196.
36. Sebastiani GD, Spinelli FR, Bartoloni E, et al. Baseline characteristics of systemic lupus erythematosus patients included in the Lupus Italian Registry of the Italian Society for Rheumatology. *Lupus*. 2021;30(8):1233–1243. doi:10.1177/09612033211012470.
37. Kikuchi J, Hanaoka H, Saito S, et al. Lupus low disease activity state within 12 months is associated with favourable outcomes in severely active systemic lupus erythematosus. *Rheumatology* (Oxford). 2022;61(9):3777–3791. doi:10.1093/rheumatology/keac002.
38. Fanouriakis A, Kostopoulou M, Andersen J, et al. EULAR recommendations for the management of systemic lupus erythematosus: 2023 update. *Annals of the Rheumatic Diseases*. 2024;83(1):15–29. First published online October 12, 2023. doi:10.1136/ard-2023-224762.