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

Date	April 15, 2026
RFE Requestor Information	Name: Sylvia Nashed, PharmD, RPh Title: Medical Education Lead E-mail: Sylvia.Nashed@bms.com
RFE Code	RFE-26-IM-101
Therapeutic Area	Immunology
Area of Interest	Pulmonary Fibrosis (PF) It is our intent to support a comprehensive, innovative, and engaging initiative that educates on the disease burden and pathophysiology of PF, addresses the unmet medical needs in the diagnosis and management of PF, and explores the correlating mechanisms, such as LPA/LPAR1 signaling, and recent clinical trial data for new and emerging therapies.
Educational Design	Bristol Myers Squibb is interested in supporting a comprehensive educational initiative. Various formats and designs will be considered, with priority given to those that are most innovative, engaging, and provide resources/tools that will further aid pulmonologists and the multidisciplinary team in their practice, as well as patient educational resources. 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. A successful proposal should include: • Clear and concise statement of the goal, learning objectives, and expected outcomes of the educational initiative • Instructional design that incorporates innovative techniques designed to engage learners, promotes application of education into practice, and incorporates a patient-centered approach to care

	• Tools that provide HCP learners the opportunity to facilitate change to improve patient outcomes and address healthcare inequities • 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)	Pulmonologists, Rheumatologists, Radiologists, Pathologists, Pulmonology NPs and PAs
Budget/Budget Range	The maximum amount of funding available for this RFE is $125,000 - $200,000. Single or multi-supported initiatives will be considered.
Accreditation	ACCME, AAPA, AANP
Geographic Coverage	Unites States, Spain, France, Germany, Italy Note: UK healthcare professional learners will need to be geoblocked from participating in the educational activity that is granted IME support.
Deadline for Submission	May 18, 2026, by 5 PM EST

Background

Pulmonary fibrosis is a complex condition characterized by repeated injury to the alveoli, which triggers faulty epithelial cell repair, inflammation and vascular leakage, and fibroblast expansion and activation. This cascade leads to scarring and extracellular matrix buildup, resulting in reduced lung elasticity and impaired gas exchange.[1] Interstitial lung disease (ILD) encompasses a diverse group of parenchymal chronic restrictive disorders characterized by varying degrees of inflammation and/or fibrosis of the lung interstitium.[2,3] Among these, idiopathic pulmonary fibrosis (IPF) and progressive pulmonary fibrosis (PPF) significantly contribute to the overall ILD burden, with a global prevalence of 17.7 and 37.0 per 100,000 persons, respectively.[4-7]

Both IPF and PPF are characterized by progressive worsening of respiratory symptoms and lung function.[8-10] IPF is characterized by a hallmark usual interstitial pneumonia (UIP) pattern and probable UIP on high-resolution computed tomography (HRCT) or surgical lung biopsy.[9,11] Clinically, IPF often presents with non-specific respiratory symptoms, such as cough, dyspnea, or velcro crackles. Accurate diagnosis requires correlation of these clinical findings with radiohistopathologic evidence, including patchy dense fibrosis with architectural distortion, a predilection for subpleural and paraseptal lung parenchyma, fibroblast foci, and exclusion of other causes.[8,12-14] As IPF is a diagnosis of exclusion, multidisciplinary assessment is essential, though this can contribute to delays in diagnosis.[8,15] PPF is characterized by radiological evidence of pulmonary fibrosis and at least two of the following within the past year: worsening respiratory symptoms, physiologic or functional decline, and/or radiologic disease progression.[8] Like IPF, delays in diagnosing PPF can negatively impact clinical outcomes, resulting in higher hospitalization rates and worse progression-free survival.[8,16]

While IPF and PPF share overlapping clinical features and are both progressive, irreversible and fatal fibrotic lung diseases,[2,3,8,9,11] they are distinct clinical entities with differing diagnostic criteria, treatment strategies, and progression patterns. IPF is a specific form of chronic, progressive fibrosing ILD with no identifiable cause, whereas PPF refers to non-IPF fibrosing ILDs (of known or unknown etiology) with a progressive phenotype.[3,8,9]

Pharmacologic options for treating IPF and PPF have historically been limited to two antifibrotic agents — nintedanib and pirfenidone — approved over a decade ago. The joint ATS/ERS/JRS/ALAT Clinical Practice Guidelines recommend nintedanib and pirfenidone for IPF, while for PPF, nintedanib is conditionally recommended after the failure of standard management of non-IPF fibrotic ILD.[8] Although these antifibrotics may slow the rate of disease progression, there remain unmet needs in stabilizing disease progression, preserving patient's daily function and quality of life, improving survival, and ensuring tolerability.[17-20] Moreover, some patients may not receive these treatments due to concerns about efficacy, safety, or disease severity.[20,21] For example, a retrospective analysis of US claims data (2014 to 2019, N=10,996) showed that only 26.4% of IPF patients initiated treatment with nintedanib or pirfenidone since their FDA approval, with a mean duration of only ~10 months on antifibrotic treatment.[21]

The progression of pulmonary fibrosis is driven by multiple signaling pathways and mediators, which can be grouped into three main processes: epithelial cell repair, inflammation/vascular leakage, and fibrosis.[1,22-26] Current treatments do not address the three core processes simultaneously. Nerandomilast, a recently approved oral phosphodiesterase 4B (PDE4B) inhibitor, has demonstrated anti-inflammatory and antifibrotic efficacy in clinical studies, thereby targeting two of these processes.[27] Several other mediators are under investigation in phase 2 and 3 clinical trials,[22-24,28] including lysophosphatidic acid (LPA) and its receptor LPAR1, as well as prostacyclin.[23,24,28] LPA-LPAR1 signaling mediates all three processes driving fibrogenesis — impaired epithelial cell repair, inflammation/vascular leakage, and fibroblast expansion/activation.[24] Elevated LPA levels in IPF are associated with greater fibrosis and lung function decline.[29-31] Similar to PDE4B, prostacyclin mediates inflammation/vascular leakage and fibrosis; preclinical models of prostacyclin receptor agonists show antifibrotic effects through sustained nuclear cAMP signaling.[27,32]

The substantial clinical burden of IPF and PPF not only reduces patients' quality of life but also imposes a significant economic burden due to hospitalizations, acute exacerbations, and the need for supportive care. Early recognition and multidisciplinary evaluation are critical for timely diagnosis and optimal management. Education targeting pulmonologists and the multidisciplinary care team on the underlying pathophysiology of PF, unmet needs, diagnostic challenges and tools (e.g., high-resolution computed tomography [HRCT], pulmonary function tests, serologic studies), clinical implications of under- and misdiagnosis (i.e., delays in optimal care, disease progression), limitations of standard of care, and the potential for new and emerging therapies targeting multiple pathways in fibrogenesis (epithelial cell repair, inflammation/vascular leakage, and/or fibroblast expansion and activation) will help enhance competence in managing PF. Keeping clinicians informed about best practices for timely diagnosis, as well as the latest evidence and expert perspectives, will enable them to make informed treatment decisions for their patients.

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 the disease burden and pathophysiology of pulmonary fibrosis (PF), with a focus on the signaling pathways—such as LPA/LPAR1—that drive fibrogenesis and disease progression.
- Identify the unmet medical needs in the management of PF, including the prevalence of under- and mis-diagnosis, associated clinical implications, and the need for more effective and well-tolerated treatments to stabilize disease progression.
- Apply appropriate diagnostic approaches for PF by recognizing early signs and symptoms, utilizing advanced diagnostic tools, conducting multidisciplinary assessments, and differentiating IPF and PPF from other ILDs.
- Discuss the novel mechanisms of action of new and emerging treatments for PF that target multiple pathways in fibrogenesis, and summarize relevant clinical trial data supporting their use.

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. Ma H, Liu S, Li S, Xia Y. Targeting Growth Factor and Cytokine Pathways to Treat Idiopathic Pulmonary Fibrosis. Front Pharmacol. 2022;13. doi:10.3389/fphar.2022.918771.
2. Rajan SK, Cottin V, Dhar R et al. Progressive pulmonary fibrosis: an expert group consensus statement. Eur Respir J. 2023;61(3):2103187. doi:10.1183/13993003.03187-2021.
3. Wijsenbeek, M., & Cottin, V. (2020). Spectrum of fibrotic lung diseases. *New England Journal of Medicine*. 2020;*383*(10): 958-968. https://doi.org/10.1056/NEJMra2005230
4. Golchin N, Lesperance T, Scheuring J et al. EPH207 Incidence and Prevalence of Idiopathic Pulmonary Fibrosis (IPF): A Systematic Literature Review and Meta-Analysis.

Value in Health. 2024;27(12):S259-S260. doi:10.1016/j.jval.2024.10.1333.
5. Zheng Q, Cox IA, Campbell JA et al. Mortality and survival in idiopathic pulmonary fibrosis: a systematic review and meta-analysis. ERJ Open Res. 2022;8(1). doi:10.1183/23120541.00591-2021.
6. Golchin N, Lesperance T, Scheuring J et al. EPH261 Incidence and Prevalence of Non-Idiopathic Pulmonary Fibrosis (IPF) Progressive Fibrosing Interstitial Lung Disease (PF-ILD): A Systematic Literature Review and Meta-Analysis. Value in Health. 2024;27(12):S269. doi:10.1016/j.jval.2024.10.1385.
7. Nasser M, Larrieu S, Si-Mohamed S et al. Progressive fibrosing interstitial lung disease: a clinical cohort (the PROGRESS study). Eur Respir J. 2021;57(2):2002718. doi:10.1183/13993003.02718-2020.
8. Raghu G, Remy-Jardin M, Richeldi L et al. Idiopathic Pulmonary Fibrosis (an Update) and Progressive Pulmonary Fibrosis in Adults: An Official ATS/ERS/JRS/ALAT Clinical Practice Guideline. Am J Respir Crit Care Med. 2022;205(9):E18-E47. doi:10.1164/rccm.202202-0399ST.
9. Raghu G, Collard HR, Egan JJ et al; ATS/ERS/JRS/ALAT Committee on Idiopathic Pulmonary Fibrosis. An official ATS/ERS/JRS/ALAT statement: idiopathic pulmonary fibrosis: evidence-based guidelines for diagnosis and management. Am J Respir Crit Care Med. 2011 Mar 15;183(6):788-824. doi: 10.1164/rccm.2009-040GL.
10. Swigris J, Cutts K, Male N, et al. The Living with Pulmonary Fibrosis questionnaire in progressive fibrosing interstitial lung disease. ERJ Open Res. 2021 May 24;7(2):00145-2020. doi: 10.1183/23120541.00145-2020.
11. Lynch DA, Sverzellati N, Travis WD, et al. Diagnostic criteria for idiopathic pulmonary fibrosis: a Fleischner Society White Paper. Lancet Respir Med. 2018 Feb;6(2):138-153. doi: 10.1016/S2213-2600(17)30433-2. Epub 2017 Nov 15.
12. Glass DS, Grossfeld D, Renna HA et al. Idiopathic pulmonary fibrosis: Current and future treatment. Clin Respir J. 2022;16(2):84-96. doi:10.1111/crj.13466.
13. Tomassetti S, Poletti V, Ravaglia C et al. Incidental discovery of interstitial lung disease: diagnostic approach, surveillance and perspectives. Eur Respir Rev. 2022;31(164):210206. doi:10.1183/16000617.0206-2021.
14. Alsomali H, Palmer E, Aujayeb A, Funston W. Early Diagnosis and Treatment of Idiopathic Pulmonary Fibrosis: A Narrative Review. Pulmonary Therapy. 2023;9(2):177-193. doi:10.1007/s41030-023-00216-0.
15. Raghu G, Remy-Jardin M, Myers JL et al. Diagnosis of Idiopathic Pulmonary Fibrosis. An Official ATS/ERS/JRS/ALAT Clinical Practice Guideline. Am J Respir Crit Care Med. 2018;198(5):E44-E68. doi:10.1164/rccm.201807-1255ST.
16. Hoyer N, Prior TS, Bendstrup E, Shaker SB. Diagnostic delay in IPF impacts progression-free survival, quality of life and hospitalisation rates. BMJ Open Respir Res. 2022;9(1):E1276. doi:10.1136/bmjresp-2022-001276.
17. Flaherty KR, Wells AU, Cottin V et al. Nintedanib in Progressive Fibrosing Interstitial Lung Diseases. N Engl J Med. 2019;381(18):1718-1727. doi:10.1056/NEJMoa1908681.
18. Richeldi L, du Bois RM, Raghu G et al. Efficacy and Safety of Nintedanib in Idiopathic Pulmonary Fibrosis. N Engl J Med. 2014;370(22):2071-2082. doi:10.1056/NEJMoa1402584.
19. King TE, Bradford WZ, Castro-Bernardini S et al. A Phase 3 Trial of Pirfenidone in Patients with Idiopathic Pulmonary Fibrosis. N Engl J Med. Published online May 18, 2014. doi:10.1056/NEJMoa1402582.
20. Wijsenbeek M, Kreuter M, Olson A et al. Progressive fibrosing interstitial lung diseases: current practice in diagnosis and management. Curr Med Res Opin. 2019;35(11):2015-2024. doi:10.1080/03007995.2019.1647040.

21. Dempsey TM, Payne S, Sangaralingham L, Yao X, Shah ND, Limper AH. Adoption of the Antifibrotic Medications Pirfenidone and Nintedanib for Patients with Idiopathic Pulmonary Fibrosis. Ann Am Thorac Soc. 2021;18(7):1121-1128. doi:10.1513/AnnalsATS.202007-901OC.
22. Liu GY, Budinger GRS, Dematte JE. Advances in the management of idiopathic pulmonary fibrosis and progressive pulmonary fibrosis. BMJ. 2022;377(jun29 5):E66354. doi:10.1136/bmj-2021-066354.
23. Kolb M, Crestani B, Maher TM. Phosphodiesterase 4B inhibition: a potential novel strategy for treating pulmonary fibrosis. Eur Respir Rev. 2023;32(167):220206. doi:10.1183/16000617.0206-2022.
24. Volkmann ER, Denton CP, Kolb M et al. Lysophosphatidic acid receptor 1 inhibition: a potential treatment target for pulmonary fibrosis. Eur Respir Rev. 2024;33(172):240015. doi:10.1183/16000617.0015-2024.
25. Mei Q, Liu Z, Zuo H, Yang Z, Qu J. Idiopathic Pulmonary Fibrosis: An Update on Pathogenesis. Front Pharmacol. 2022 Jan 19;12:797292. doi: 10.3389/fphar.2021.797292.
26. Selman M, Pardo A. When things go wrong: exploring possible mechanisms driving the progressive fibrosis phenotype in interstitial lung diseases. Eur Respir J. 2021 Sep 9;58(3):2004507. doi: 10.1183/13993003.04507-2020.
27. Keith R, Nambiar AM. Potential of phosphodiesterase 4B inhibition in the treatment of progressive pulmonary fibrosis. Ther Adv Respir Dis. 2025;19. doi:10.1177/17534666241309795.
28. Stitham J, Midgett C, Martin KA, Hwa J. Prostacyclin: an inflammatory paradox. Front Pharmacol. 2011;2:24. doi:10.3389/fphar.2011.00024.
29. Tager Andrew M, LaCamera Peter, Shea Barry S et al. The lysophosphatidic acid receptor LPA1 links pulmonary fibrosis to lung injury by mediating fibroblast recruitment and vascular leak. Nature Medicine. 2008;14(1):45-54. doi:10.1038/nm1685.
30. Neighbors M, Li Q, Zhu S (Joe) et al. Bioactive lipid lysophosphatidic acid species are associated with disease progression in idiopathic pulmonary fibrosis. J Lipid Res. 2023;64(6). doi:10.1016/j.jlr.2023.100375.
31. Ahluwalia N, Shea BS, Tager AM. New therapeutic targets in idiopathic pulmonary fibrosis. Aiming to rein in runaway wound-healing responses. Am J Respir Crit Care Med. 2014 Oct 15;190(8):867-78. doi: 10.1164/rccm.201403-0509PP.
32. Roberts MJ, May LT, Keen AC et al. Inhibition of the Proliferation of Human Lung Fibroblasts by Prostacyclin Receptor Agonists is Linked to a Sustained cAMP Signal in the Nucleus. Front Pharmacol. 2021;12:669227. doi:10.3389/fphar.2021.669227.