# aTyr Pharma to Present Preclinical Research Demonstrating Anti-Fibrotic Effects of ATYR0101 in Lung and Kidney Fibrosis at Keystone Symposia on Fibrosis

December 9, 2024

**ATYR0101 interacts with LTBP-1 to induce myofibroblast apoptosis through a novel anti-fibrotic mechanism to reduce fibrosis and fibrotic markers in models of lung and kidney fibrosis.**

SAN DIEGO, Dec. 09, 2024 (GLOBE NEWSWIRE) -- aTyr Pharma, Inc. (Nasdaq: ATYR) (“aTyr” or the “Company”), a clinical stage biotechnology company engaged in the discovery and development of first-in-class medicines from its proprietary tRNA synthetase platform, today announced that the Company will present two posters related to its tRNA synthetase candidate ATYR0101 at the Keystone Symposia on Fibrosis: Inflammation, Drivers, and Therapeutic Resolution, which is scheduled to take place December 8 – 11, 2024, in Whistler, British Columbia, Canada.

ATYR0101 is a fusion protein derived from a proprietary extracellular domain of aspartyl-tRNA synthetase (DARS) that binds to latent transforming growth factor beta binding protein 1 (LTBP-1) to induce myofibroblast apoptosis.

“We are excited by these findings that further demonstrate the way in which ATYR0101 binds a known fibrosis target with pronounced effects in preclinical models of lung and renal fibrosis,” said Sanjay S. Shukla, M.D., M.S., President and Chief Executive Officer of aTyr. “These findings suggest that ATYR0101 has the potential to be a next generation anti-fibrotic for lung and kidney fibrosis with a differentiated mechanism of action compared to current standard of care that could potentially treat advanced fibrotic conditions.”

Details of the presentations appear below. The posters will be available on the aTyr website once presented.

**Title: A Newly Evolved Domain of Asp-tRNA Synthetase Interacts with Latent Transforming Growth Factor Beta Binding Protein 1 (LTBP-1) to Induce Myofibroblast Apoptosis**
**Authors:** Ying-Ting Wang, Kristina Hamel, Andrew Imfeld, Yeeting E. Chong, Kaitlyn Rauch, Wayne Liu, Zhiwen Xu, Ryan A. Adams, Leslie Nangle. aTyr Pharma, San Diego, CA.
**Poster Number:** 1046
**Session:** Poster Session #1
**Date and Time:** Monday, December 9, 2024, at 7:30 p.m. PST

The poster presents findings demonstrating that ATYR0101 binds directly to LTBP-1 resulting in caspase-3/7 mediated apoptosis in transforming growth factor beta (TGF*β*)-1-differentiated myofibroblasts while having no effect on undifferentiated fibroblasts, which was observed in multiple cell types demonstrating potential in several organ systems. The ATYR0101-induced myofibroblast apoptosis activity was confirmed to be dependent upon LTBP-1, TGF*β* activation and downstream gene expression changes. These findings suggest that ATYR0101 has promise as a novel and transformative anti-fibrotic therapeutic with a unique mechanism of action.

**Title: Anti-Fibrotic Activity Observed Across Preclinical Models of Pulmonary and Renal Fibrosis for a Potential Therapeutic Based on Asp-tRNA Synthetase**
**Authors:** Alison Barber, Clara Polizzi, Jasmine Stamps, Max Pastenes, Yeeting E. Chong, Andrew Imfeld, Chun Po Fung, Honglei Tian, Zhenguo Wu, Ryan A. Adams, Christoph Burkart, Leslie Nangle. aTyr Pharma, San Diego, CA, Hong Kong University of Science and Technology, Clear Water Bay, Kowloon, Hong Kong, China.
**Poster Number:** 1045
**Session:** Poster Session #1
**Date and Time:** Monday, December 9, 2024, at 7:30 p.m. PST

The poster presents findings investigating ATYR0101 in the bleomycin (BLM) model of lung fibrosis and ureteral obstruction (UUO) model of kidney fibrosis to examine the pharmacological activity of ATYR0101 in experimental models of fibrotic disease. In the lung BLM model, ATYR0101 treatment resulted in a significant reduction in Ashcroft score and collagen content, key measures of fibrosis, in addition to a pronounced reduction of myofibroblasts. In the UUO model, treatment with ATYR0101 resulted in reduced collagen content with a significant reduction of fibrosis. Importantly, ATYR0101 achieves these effects in a differentiated way compared to current standard of care. These findings suggest that ATYR0101 has the potential to be a novel anti-fibrotic therapeutic agent for lung and renal fibrosis with a differentiated profile compared to current standard of care.

**About aTyr**

aTyr is a clinical stage biotechnology company leveraging evolutionary intelligence to translate tRNA synthetase biology into new therapies for fibrosis and inflammation. tRNA synthetases are ancient, essential proteins that have evolved novel domains that regulate diverse pathways extracellularly in humans. aTyr's discovery platform is focused on unlocking hidden therapeutic intervention points by uncovering signaling pathways driven by its proprietary library of domains derived from all 20 tRNA synthetases. aTyr's lead therapeutic candidate is efzofitimod, a first-in-class biologic immunomodulator in clinical development for the treatment of interstitial lung disease, a group of immune-mediated disorders that can cause inflammation and progressive fibrosis, or scarring, of the lungs. For more information, please visit www.atyrpharma.com.

**Forward-Looking Statements**

This press release contains forward-looking statements within the meaning of the Private Securities Litigation Reform Act of 1995. Forward-looking statements are usually identified by the use of words such as "anticipate," “believes,” “designed,” “can,” “expects,” “intends,” “may,” “plans,” “potential,” suggest,” “will,” and variations of such words or similar expressions. We intend these forward-looking statements to be covered by such safe harbor provisions for forward-looking statements and are making this statement for purposes of complying with those safe harbor provisions. These forward-

looking statements include, among others, statements regarding the research and development activities related to and the potential therapeutic benefits and applications of our current and future product candidates, including ATYR0101 as a transformative, next generation anti-fibrotic differentiated from the current standard of care. These forward-looking statements also reflect our current views about our plans, intentions, expectations, strategies and prospects, which are based on the information currently available to us and on assumptions we have made. Although we believe that our plans, intentions, expectations, strategies and prospects, as reflected in or suggested by these forward-looking statements, are reasonable, we can give no assurance that the plans, intentions, expectations, strategies or prospects will be attained or achieved. All forward-looking statements are based on estimates and assumptions by our management that, although we believe to be reasonable, are inherently uncertain. Furthermore, actual results may differ materially from those described in these forward-looking statements and will be affected by a variety of risks and factors that are beyond our control including, without limitation, uncertainty regarding geopolitical and macroeconomic events, risks associated with the discovery, development and regulation of our product candidates (including the risk that future findings do not support the findings described in the posters), the risk that we or our partners may cease or delay preclinical or clinical development activities for any of our existing or future product candidates for a variety of reasons (including difficulties or delays in patient enrollment in planned clinical trials), the possibility that existing collaborations could be terminated early, and the risk that we may not be able to raise the additional funding required for our business and product development plans, as well as those risks set forth in our most recent Annual Report on Form 10-K, Quarterly Reports on Form 10-Q and in our other SEC filings. Except as required by law, we assume no obligation to update publicly any forward-looking statements, whether as a result of new information, future events or otherwise.

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