Results from Symic Bio Liver Fibrosis Program Presented at the International Liver Congress 2018

Matrix-targeting therapeutic candidate SBR-294 demonstrates efficacy of a new mechanism of action to reduce liver fibrosis in two preclinical models

SAN FRANCISCO, April 16, 2018 – Symic Bio, a biopharmaceutical company developing novel biotherapeutics targeting the extracellular matrix, presented results from a lead candidate therapy for liver fibrosis at the International Liver Congress 2018, the annual meeting of the European Association for the Study of the Liver, held April 11-15, 2018, in Paris. Results demonstrated that treatment with matrix-targeting therapeutic candidate SBR-294 resulted in decreased liver fibrosis in two distinct preclinical models of liver fibrosis.

"These results demonstrate the potential of our platform in an additional therapeutic area and build upon the capabilities of matrix-targeting therapeutics that have already been observed in the clinic with our other programs," said Ken Horne, Chief Executive Officer of Symic Bio. "SBR-294 is designed to have distinctive properties, including targeting to diseased liver tissue. Our results support the idea that SBR-294 acts to remove pro-fibrotic cues in the tissue environment. The ability of SBR-294 to demonstrate effects in these models builds upon the mechanism of our SB-030 therapeutic candidate in vascular disease, which also demonstrates inhibition of platelet-mediated fibrosis. Because of its mechanism of action, we would expect the effects of SBR-294 to be complementary to other types of therapies directed at liver fibrosis. We look forward to clinical development for the treatment of advanced liver fibrosis with the potential for the filing of an IND in 2019."

The results were presented by Kate Stuart, Symic Bio co-founder and Senior Director of Translational Biology, in an oral presentation: "Interfering with local fibrotic platelet activation significantly inhibits fibrosis in multiple animal models: suggestions of the importance of the platelet-wound healing axis for fibrosis" (abstract PS-165). Results addressed the hypothesis that SBR-294, a molecule designed to both inhibit platelet activation on collagen and block PDGF signaling, could reduce fibrosis progression. Using the carbon tetrachloride *in vivo* model of liver fibrosis, SBR-294 treatment significantly decreased the content of collagen protein in the liver and reduced mRNA levels of both collagen type I and type III. Using a separate *in vivo* model, the STAM model, SBR-294 treatment also reduced collagen protein in the liver. These results were also supported by *in vitro* studies demonstrating that SBR-294 treatment inhibits collagen-mediated platelet activation and inhibits hepatic stellate cell proliferation and activation. Platelet activation and stellate cell activation are processes linked to progression of liver fibrosis.

About Symic Bio

Symic Bio is a biopharmaceutical company developing novel matrix-targeting therapeutics, a new category of therapeutics focused on matrix biology. These therapeutics, with potential applications in a wide variety of disease states, are inspired by naturally occurring macromolecules that play key

regulatory roles within the extracellular matrix. Symic Bio currently has two clinical candidates: SB-061, directed at disease modification and pain management in the treatment of osteoarthritis, and SB-030, targeting the prevention of peripheral vein graft failure. In addition, Symic Bio is investigating applications in the areas of fibrosis, oncology and diseases of the central nervous system. For additional information please visit the company's website at www.symic.bio, LinkedIn page at www.linkedin.com/company/symic-bio or follow on Twitter at www.twitter.com/symicbio.

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