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Gilead Sciences, Scholar Rock unite to develop remedies for fibrotic diseases

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Gilead Sciences, Inc. and Scholar Rock Holding Corporation announced that the companies have entered into a strategic collaboration to discover and develop highly specific inhibitors of transforming growth factor beta (TGF?) activation for the treatment of fibrotic diseases.

Under the collaboration, Gilead has exclusive options to license worldwide rights to product candidates that emerge from three Scholar Rock TGF? programs: inhibitors that target activation of latent TGF?1 with high affinity and specificity, inhibitors that selectively target activation of latent TGF?1 localized to the extracellular matrix, and a third TGF? discovery program.

Scholar Rock is responsible for antibody discovery and preclinical research through product candidate nomination, after which, upon exercising the option for a program, Gilead will be responsible for the program's preclinical and clinical development and commercialization. Scholar Rock retains exclusive worldwide rights to discover, develop, and commercialize certain TGF? inhibitors for oncology and cancer immunotherapy.

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In addition, Scholar Rock will receive a one-time milestone payment of $25 million upon the successful completion of specific preclinical studies and be eligible to receive up to an additional $1,425 million in potential payments aggregated across all three programs based on the successful achievement of certain research, development, regulatory and commercialization milestones. Scholar Rock would also receive high single-digit to low double-digit tiered royalties on sales of potential future products originating from the collaboration.

Fibrosis is a debilitating pathological feature of many diseases that scars tissues and vital organs and is a major cause of morbidity and mortality. TGF?-driven signaling is thought to be a central regulator of fibrosis.

Inhibitors of TGF? signaling discovered through Scholar Rock's proprietary platform have been shown to selectively prevent the activation of the growth factor in the fibrotic matrix in vitro and in preclinical models. By targeting the disease microenvironment, these highly specific inhibitors of TGF? activation may offer a novel approach to suppressing pro-fibrotic signaling in multiple organs.