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Data from Prothena's Preclinical Program on Conformation-Specific Antibodies Against Misfolded Transthyretin Published in Amyloid

Antibodies bind specifically to misfolded, non-native forms of transthyretin, disrupting fibril formation

DUBLIN, Ireland, March 16, 2016 (GLOBE NEWSWIRE) -- Prothena Corporation plc (NASDAQ:PRTA), a late-stage clinical biotechnology company focused on the discovery, development and commercialization of novel protein immunotherapies, today announced the publication of preclinical data demonstrating that the company's conformation-specific antibodies developed against misfolded transthyretin (TTR) bind to and facilitate in vitro cellular uptake of amyloidogenic forms of TTR. The research was published online on March 16, 2016 in the peer-reviewed journal *Amyloid* in a manuscript titled "Novel conformation-specific monoclonal antibodies against amyloidogenic forms of transthyretin."

The published research describes monoclonal antibodies that specifically bind to misfolded forms of the TTR protein, leaving the native form of the protein unaffected. These antibodies disrupt the in vitro formation of TTR fibrils and recognize TTR amyloid deposits in cardiac tissue from confirmed ATTR amyloidosis patients. Additionally, once bound to misfolded forms of TTR, the ability of these antibodies to promote the phagocytic clearance of misfolded TTR suggests the in vivo potential to elicit the immune systems removal of misfolded TTR amyloid deposits from tissue. This preclinical work suggests that one of Prothena's conformation-specific monoclonal antibodies may hold potential as a therapy for patients suffering from transthyretin-mediated amyloidosis (ATTR amyloidosis).

"One of the fundamental challenges of developing an effective treatment for many amyloid diseases, including ATTR amyloidosis, is creating a therapeutic that not only reduces circulating levels of the misfolded protein, but one that can also prevent the formation of new fibrils and facilitate the elimination of fibrils that have deposited in tissue and cause progressive organ failure," said Gene Kinney, Ph.D., Prothena's Chief Scientific Officer and Head of Research and Development. "Our preclinical data demonstrate that these antibodies are highly selective for the misfolded form of TTR, can prevent fibril formation and can potentially recruit immune cells to clear amyloid fibrils from tissue. This could offer an important compliment to other therapies for TTR amyloidosis in development."

About ATTR Amyloidosis
Transthyretin-mediated amyloidosis (ATTR amyloidosis) is a rare and progressive disease characterized by deposition of aggregates of misfolded protein, or amyloid. There are three types of ATTR amyloidosis: familial amyloid polyneuropathy (FAP); familial amyloid cardiomyopathy (FAC); and wild-type (or senile systemic) ATTR. FAP and FAC are hereditary and can occur concurrently, whereas wild-type ATTR is not hereditary.

TTR protein is produced primarily in the liver and in its normal tetrameric form serves as a carrier for thyroxin and vitamin A, the latter via the binding of retinol binding protein. In hereditary FAP and FAC the body makes a mutant form of the TTR protein. There are more than 100 reported types of TTR mutations that promote amyloid fibril formation, which most commonly affect the heart and nervous system. Wild-type ATTR is similar to hereditary ATTR except that the protein that is deposited is the misfolded, non-mutated transthyretin protein.

For more information on ATTR, please visit the websites of the Amyloidosis Support Group and the Amyloidosis Foundation.

About Prothena

Prothena Corporation plc is a global biotechnology company seeking to fundamentally change the course of progressive diseases with its late-stage clinical pipeline of novel therapeutic antibodies. Fueled by its deep scientific understanding built over decades of research in protein misfolding and cell adhesion — the root causes of many serious or currently untreatable amyloid and inflammatory diseases — Prothena has advanced several drug candidates into clinical trials while pursuing discovery of additional novel therapies. Our clinical pipeline of antibody-based product candidates targets a number of potential indications including AL amyloidosis (NEOD001), Parkinson's disease and other related synucleinopathies (PRX002) and inflammatory diseases, including psoriasis (PRX003). For more information, please visit the company's web site at www.prothena.com.

Forward-looking Statements

This press release contains forward-looking statements. These statements relate to, among other things, whether

Prothena's preclinical antibodies developed against misfolded transthyretin (TTR) will elicit removal of misfolded TTR amyloid deposits or hold potential as a therapy for ATTR amyloidosis. These statements are based on estimates, projections and assumptions that may prove not to be accurate, and actual results could differ materially from those anticipated due to known and unknown risks, uncertainties and other factors, including but not limited to the risks, uncertainties and other factors described in the "Risk Factors" sections of our Annual Report on Form 10-K filed with the Securities and Exchange Commission (SEC) on February 25, 2016 and our subsequent Quarterly Reports on Form 10-Q filed with the SEC. Prothena undertakes no obligation to update publicly any forward-looking statements contained in this press release as a result of new information, future events or changes in Prothena's expectations.

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