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Prothena to Present Wide Range of Studies on Amyloidosis, Including New Clinical Data on NEOD001, at International Symposium on Amyloidosis

- **New clinical data to be presented from expansion cohort of Phase 1/2 trial of NEOD001 for the potential treatment of AL amyloidosis**
- **Additional presentations to highlight data from preclinical and quality of life studies showcases commitment to advancing new therapies for multiple forms of amyloidosis**

DUBLIN, Ireland, May 23, 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 that it will present on a broad range of topics, including clinical and preclinical research from Prothena's programs in AL and ATTR amyloidosis, as well as data from several quality of life studies, at the 15th International Symposium on Amyloidosis (ISA) to be held July 3-7 in Uppsala, Sweden.

Further information regarding the date and time of the presentations will be communicated when they are finalized by the conference organizers.

About AL Amyloidosis

Systemic amyloidoses are a complex group of progressive diseases caused by tissue deposition of misfolded proteins that result in progressive organ damage. The most common type, AL amyloidosis or primary amyloidosis, involves a hematological disorder caused by plasma cells that produce misfolded immunoglobulin light chain resulting in deposits of abnormal AL protein (amyloid) in the tissues and organs of individuals with this disease. There are no approved treatments for AL amyloidosis, and none that directly target potentially toxic forms of the AL protein. AL amyloidosis is a rare disorder and it is estimated that about 30,000 to 45,000 patients in the U.S. and Europe suffer from this disease. Both the causes and origins of AL amyloidosis remain poorly understood. For more information on AL amyloidosis, please visit the websites of the Amyloidosis Support Groups and the Amyloidosis Foundation.

About NEOD001

NEOD001 is a monoclonal antibody that specifically targets the circulating soluble amyloid and deposited insoluble amyloid that accumulates in both the AL and AA forms of amyloidosis. Patients with AL amyloidosis may be eligible to enroll in one of two clinical trials for NEOD001. The VITAL Amyloidosis Study, a double-blind, placebo-controlled, global Phase 3 registrational trial, is evaluating NEOD001 in newly-diagnosed, treatment-naïve patients with AL amyloidosis, and will assess a composite of all-cause mortality or cardiac hospitalizations in addition to biomarker, functional and quality of life endpoints. The PRONTO trial, a double-blind, placebo-controlled, global Phase 2b registration-directed trial, is evaluating NEOD001 in previously-treated patients with AL amyloidosis and persistent cardiac dysfunction, and will assess best response over 12 months of the cardiac functional biomarker NT-proBNP, defined by the consensus criteria of NT-proBNP change, in addition to other functional biomarker, and quality of life endpoints. More information on The VITAL Amyloidosis Study and the PRONTO trial is available at www.clinicaltrials.gov, by searching on NCT #02312206 for VITAL and NCT #02632786 for PRONTO.

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 Groups and the Amyloidosis Foundation.

About Prothena

Prothena Corporation plc is a global, late-stage clinical biotechnology company seeking to fundamentally change the course of progressive diseases with its 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).

Forward-looking Statements

This press release contains forward-looking statements. These statements relate to, among other things, our goal to develop new therapies for multiple forms of 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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