June 21, 2016

Prothena to Present at 15th International Symposium on Amyloidosis Showcasing Broad Commitment to Advancing New Therapies for Systemic Amyloidoses

- **New clinical data to be presented from Phase 1/2 dose-escalation and expansion trial of NEOD001 for the potential treatment of AL amyloidosis**
- **Presentations to highlight data from clinical, preclinical and quality-of-life studies**
- **Investor webcast planned for July 5, 2016 at 10:30 AM EDT**

DUBLIN, Ireland, June 21, 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 multiple Prothena studies, including an oral presentation of new clinical data from the Phase 1/2 dose-escalation and expansion trial of NEOD001 in previously-treated patients with AL amyloidosis and persistent organ dysfunction, will be presented at the 15th International Symposium on Amyloidosis (ISA), to be held July 3-7, 2016 in Uppsala, Sweden.

"For more than two decades, Prothena scientists have been researching the biology of amyloid caused by misfolded proteins and our commitment to advancing new therapies for amyloid diseases has never been stronger," stated Dale Schenk, PhD, President and Chief Executive Officer of Prothena, "At ISA we look forward to the opportunity to present a broad range of studies, including new clinical data from our lead investigational compound, NEOD001, for the potential treatment of AL amyloidosis, preclinical research from a new program we plan to advance into the clinic, PRX004, in ATTR amyloidosis, as well as new data on quality of life, and additional amyloid research targets."

New clinical data on safety, tolerability, and organ response, from the Phase 1/2 trial of NEOD001 in previously-treated patients with AL amyloidosis and persistent organ dysfunction, will be presented in the following oral session:

(Abstract #25263) NEOD001 Demonstrates Organ Biomarker Responses in Patients with Light Chain Amyloidosis and Persistent Organ Dysfunction: Results from the Expansion Phase of a Phase 1/2 Study

- Presenter: Morie A. Gertz, MD, Professor of Medicine, Mayo Clinic
- Session: Gene Therapy and Innovative Drugs Trial
- Date and Time: Oral presentation - July 5, 3:15-3:30 PM CEST (9:15-9:30 AM EDT)
- Location: Main Auditorium, Uppsala Konsert and Kongress (UKK)

An investor conference call and webcast is planned to discuss the data on July 5, 2016 at 10:30 AM EDT, and dial-in details will be made available in advance of the call.

Additional preclinical studies related to Prothena's R&D pipeline programs will be presented in the following sessions:

(Abstract #25338) Novel Conformation-Specific Monoclonal Antibodies Against Amyloidogenic Forms of Transthyretin Bind Specifically to TTR Deposits Present in Disease Tissue Derived from ATTR Amyloidosis Patients

- Presenter: Jeffrey Higaki, PhD, Senior Scientist, Prothena Biosciences Inc
- Session: Poster Session A
- Date and Time: July 4, 12:00-1:30 PM CEST
- Location: Level 6

(Abstract #25256) NEOD001 Specifically Binds Aggregated Light Chain Infiltrates in Multiple Organs of Patients with AL Amyloidosis and Promotes Phagocytic Clearance of Light Chain Aggregates In Vitro

- Presenter: Wagner Zago, PhD, Head of Research, Prothena Biosciences Inc
- Session: Poster Session C
- Date and Time: July 6, 12:30-2:00 PM CEST
- Location: Level 6

Quality of life and disease education studies will be presented in the following sessions:

(Abstract #26305) Recommendations from the Amyloidosis Research Consortium Education Roundtable at the American College of Cardiology Annual Meeting, April 1, 2016

- Presenter: Martha Grogan, Assistant Professor of Medicine, Mayo Clinic College of Medicine
- Session: Poster Session A
- Date and Time: July 4, 12:00-1:30 PM CEST
- Location: Level 6

(Abstract #25767) The Relative Burden of AL Amyloidosis on Health-Related Quality of Life

- Presenter: Spencer Guthrie, Head of Development Affairs, Prothena Biosciences Inc
- Session: Poster Session C
- Date and Time: July 6, 12:30-2:00 PM CEST
- Location: Level 6

(Abstract #25766) Treatment History, Tolerability and Impact on Health-Related Quality of Life in AL Amyloidosis

- Presenter: Spencer Guthrie, Head of Development Affairs, Prothena Biosciences Inc
- Session: Poster Session C
- Date and Time: July 6, 12:30-2:00 PM CEST
- Location: Level 6

(Abstract #25772) The Impact of AL Amyloidosis on Absenteeism, Reduced Productivity and Job Loss

- Presenter: Michelle K. White, PhD, Sr. Scientist and Sr. Director, Optum
- Session: Poster Session C
- Date and Time: July 6, 12:30-2:00 PM CEST
- Location: Level 6

(Abstract #25763) Psychometric Validation of the SF-36v2® Health Survey in an AL Amyloidosis Population

- Presenter: Michelle K. White, PhD, Sr. Scientist and Sr. Director, Optum
- Date and Time: July 6, 12:30-2:00 PM CEST
- Location: Level 6

Nonclinical research on amyloid targets that are not part of Prothena's pipeline will be presented in the following sessions:

(Abstract #25252) Novel Anti-Medin Antibodies Detect Medin Deposits in Aortic Aneurysm, Marfan Syndrome and Other Cardiovascular Diseases

- Presenter: Wagner Zago, PhD, Head of Research, Prothena Biosciences Inc
- Session: Poster Session A
- Date and Time: July 4, 12:00-1:30 PM CEST
- Location: Level 6

(Abstract #25243) Passive Treatment with Monoclonal Antibodies to Islet Amyloid Polypeptide Significantly Improved Both Hemoglobin A1C and Glucose Tolerance and Decreased Extracellular Amyloid Deposits in the Transgenic HIP Rat Model of Type 2 Diabetes

- Presenter: Wagner Zago, PhD, Head of Research, Prothena Biosciences Inc
- Session: More AA and Other Amyloid Forms
- Date and Time: Oral presentation - July 6, 2:15-2:30 PM CEST
- Location: Main Auditorium, Uppsala Konsert and Kongress (UKK)

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 Group and the Amyloidosis Foundation.

About NEOD001

NEOD001 is a humanized 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 PRONTO trial, a global, Phase 2b, double-blind, placebo-controlled, registration-directed trial, will evaluate NEOD001 in previously-treated patients with AL amyloidosis and persistent cardiac dysfunction, and will 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 biomarker, quality of life and functional endpoints. The VITAL Amyloidosis Study, a global, Phase 3, double-blind, placebo-controlled, registrational study, 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, quality of life and functional endpoints. More information on the PRONTO study and The VITAL Amyloidosis Study is available at www.clinicaltrials.gov, by searching NCT#02632786 for PRONTO and NCT #02312206 for VITAL or www.clinicaltrialsregister.eu, by searching EudraCT #2015-004318-14 for PRONTO and EudraCT #2014-003865-11 for VITAL.

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 PRX004

PRX004 is a monoclonal antibody designed to specifically target and clear the misfolded forms of the amyloid TTR protein found in ATTR amyloidosis, and leave the native form of the protein unaffected. Currently in preclinical development, Prothena plans to advance PRX004 into the clinic as a potential therapy for ATTR amyloidosis.

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 pipeline of antibody-based product candidates targets a number of potential indications including AL amyloidosis (NEOD001), Parkinson's disease and other related synucleinopathies (PRX002), inflammatory diseases including psoriasis (PRX003), and ATTR amyloidosis (PRX004).

Forward-looking Statements

This press release contains forward-looking statements. These statements relate to, among other things, our commitment to advancing new therapies for amyloid diseases; and our plan to advance PRX004 into the clinic. 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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