



August 15, 2017

Prothena to Present New Research on Cardiac Biomarker NT-proBNP in AL Amyloidosis at HFSA Annual Meeting

- | **Preclinical Research Demonstrating a Relationship Between Amyloid Light Chain Toxicity and NT-proBNP Production**
- | **Clinical Outcomes Research Showing a Correlation between NT-proBNP and Health-related Quality of Life**

DUBLIN, Ireland, Aug. 15, 2017 (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 new clinical and preclinical research on the cardiac biomarker NT-proBNP will be presented in both oral and poster sessions at the Heart Failure Society of America (HFSA) Annual Scientific Meeting to be held September 16 — 19 in Dallas, Texas. NT-proBNP is a cardiac biomarker that has been consistently shown, in multiple independent studies, to be predictive of survival in patients with AL amyloidosis (Merlini, et. al, *Leukemia*, 2016).

New preclinical data demonstrating that misfolded light chains promote oxidative stress and cellular toxicity and increase NT-proBNP production in cardiomyocytes will be highlighted in oral and poster sessions. The findings provide mechanistic insight into how misfolded light chain protein induces cardiotoxicity and support the relationship between lowering of NT-proBNP and improved survival in patients with AL amyloidosis.

(Abstract #017) Aggregated Light Chain Increases Brain Natriuretic Peptide Expression and Induces Oxidative Stress Response in Cardiomyocytes

- | Presenter: Stephen J. Tam, Senior Scientist, Prothena
- | Session: Rapid Fire Abstract Session I
- | Date and Time: Sunday, September 17, 1:00 PM, CT
- | Location: Gaylord Texan Hotel and Convention Center, Grapevine 1-3
- | The abstract will also be presented as a poster in the Exhibit Hall, Saturday, September 16 — Monday, September 18

In addition, new outcomes research that demonstrates NT-proBNP response is associated with clinically meaningful improvements in health-related quality of life in patients with AL amyloidosis will also be presented in a poster session. The research supports that NT-proBNP may be useful as a surrogate for clinical measures such as health-related quality of life.

(Abstract #319) Improvements in Cardiac Biomarkers are Associated with Better Health Related Quality of Life in Patients with Light Chain Amyloidosis

- | Presenter: Tiffany Quock, Director, Health Economics and Outcomes Research, Prothena
- | Session: Quality of Care / Outcomes
- | Date and Time: Saturday, September 16, 6:15 - 7:15 PM, CT
- | Location: Gaylord Texan Hotel and Convention Center, Exhibit Hall

About NEOD001

NEOD001 is an investigational first-in-class antibody that specifically targets disease-causing misfolded light chain aggregates in AL amyloidosis. There are two ongoing global clinical studies for NEOD001. The PRONTO study, a global, Phase 2b, double-blind, placebo-controlled, registration-directed study, will evaluate NEOD001 vs. placebo in previously-treated patients with AL amyloidosis and persistent cardiac dysfunction, and will assess best response over 12 months of the cardiac 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 vs. placebo in newly-diagnosed, treatment-naïve patients with AL amyloidosis and cardiac dysfunction, with both arms of the study receiving standard of care. The VITAL study will assess a composite endpoint 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 AL Amyloidosis

Systemic amyloidoses are a complex group of diseases caused by tissue deposition of misfolded proteins that result in progressive organ damage. AL amyloidosis, the most common type, is a rare, progressive, and typically fatal disease caused by extracellular deposition of misfolded immunoglobulin light chains. An excess of light chains prone to misfolding are produced by clonal plasma cells. Soluble toxic aggregates and deposited fibrils (amyloid) lead to progressive failure of vital organs including the heart, kidneys and nervous system, causing significant morbidity and mortality. It is estimated that approximately 30,000 — 45,000 patients in the U.S. and Europe suffer from this disease. There are no approved treatments for AL amyloidosis, although patients may be treated with off-label therapies directed at the plasma cell dyscrasia. There is a large unmet need for therapies that specifically target soluble toxic aggregates and deposited fibrils, thereby improving vital organ function. For more information on AL amyloidosis, please visit the websites of the [Amyloidosis Support Groups](#), [The Amyloidosis Research Consortium](#), and the [Amyloidosis Foundation](#).

About Prothena

Prothena Corporation plc is a global, late-stage clinical biotechnology company establishing fully-integrated research, development and commercial capabilities. 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 seeks to fundamentally change the course of progressive diseases associated with this biology. The Company's pipeline of antibody therapeutic candidates targets a number of indications including AL amyloidosis (NEOD001), Parkinson's disease and other related synucleinopathies (PRX002/RG7935), inflammatory diseases, including psoriasis and psoriatic arthritis (PRX003), and ATTR amyloidosis (PRX004). The Company continues discovery of additional novel therapeutic candidates where its deep scientific understanding of disease pathology can be leveraged. For more information, please visit the Company's website at www.prothena.com.

Forward-looking Statements

This press release contains forward-looking statements. These statements relate to, among other things, the relationship between amyloid light chain toxicity and NT-proBNP production; the correlation between NT-proBNP and health-related quality of life; the relationship between lowering of NT-proBNP and improved survival in patients with AL amyloidosis; and whether NT-proBNP may be useful as a surrogate for clinical measures such as health-related quality of life. 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 27, 2017 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.

Media & Investor Contact:

Ellen Rose, Head of Communications

650-922-2405, ellen.rose@prothena.com