# Protego Presents New Data at ISA 2026 that Advances Evidence for Light-Chain Stabilization with PROT-001 in AL Amyloidosis

- *PROT-001, the company's investigational oral small molecule drug for AL amyloidosis, demonstrated an acceptable safety and tolerability profile in a healthy volunteer Phase 1 study*
- *In human cardiomyocyte and plasma cell studies, PROT-001 reduced markers of stress and toxicity, and demonstrated compatibility with standard-of-care bortezomib*
- *Additional presentations highlight advanced detection of amyloidogenic light chains with Protego's AmyLiteDx™ assay, and use of NT-proBNP as an early clinical biomarker of cardiac response*
- *Results support evaluating PROT-001 in planned Phase 2/3 SHIELD-AL study in AL amyloidosis patients*

**SAN DIEGO, Nov. 16, 2026 --** Protego Biopharma, Inc., a clinical-stage biotechnology company developing small-molecule therapies for protein misfolding diseases, today announced results from five presentations that highlight the Phase I clinical and preclinical profile of PROT-001, the company's potential first-in-class, investigational lambda light chain kinetic stabilizer to treat light chain (AL) amyloidosis. The presentations at the XX International Symposium on Amyloidosis (ISA 2026), taking place November 15-18, 2026, come on the heels of Protego receiving clearance from the United States Food and Drug Administration (FDA) to proceed with SHIELD-AL, its Phase 2/3 trial of PROT-001 in AL amyloidosis patients.

PROT-001 is designed as a kinetic stabilizer of amyloidogenic light chains (LCs) and to neutralize their toxicity. Across the five ISA 2026 presentations, Protego presented data that, collectively, support the advancement of PROT-001 into pivotal studies.

"Cardiac involvement is a major driver of morbidity and mortality in AL amyloidosis. Plasma cell-directed therapies reduce the production of light chains, but circulating amyloidogenic light chains can continue to damage the heart," said Brent J. Warner, MBA, Chief Executive Officer of Protego. "PROT-001 is designed to complement these treatments by stabilizing amyloidogenic light chains and reducing their toxicity. The data presented at ISA 2026 strengthen the rationale for SHIELD-AL, where we will evaluate whether this approach can deliver earlier and deeper cardiac responses."

Detailed results from Protego's five poster presentations are as follows:

**PROT-001, a first-in-class lambda light chain kinetic stabilizer for AL amyloidosis: Results from the Phase I PRO-1A-101 Study (Abstract 32458)**
PRO-1A-101 evaluated the safety, tolerability, pharmacokinetics (PK), and pharmacodynamics (PD) of PROT-001 in 102 healthy volunteers, in four study parts: a randomized, placebo-controlled single ascending dose (SAD), a multiple

ascending dose (MAD) study, an open-label food effect (FE) study, and an open-label drug-drug interaction (DDI) study.

In the study, twice-daily oral dosing of PROT-001 was generally safe and well-tolerated and achieved plasma levels associated with sustained ex vivo stabilization of amyloidogenic lambda light chains, supporting the proposed mechanism of action and further development of PROT-001 in patients with AL amyloidosis. The PK and PD results supported further development of orally administered PROT-001 dosed 300-600 mg twice a day.

**PROT-001 Suppresses Amyloidogenic Light Chain Induced BNP Signaling and Cardiotoxicity in Human Cardiomyocytes (Abstract 32266)**
PROT-001 helped protect human heart cells from the harmful effects of amyloid-forming light chains, reducing markers of stress and damage in cell-based studies. The results demonstrate the potential of PROT-001 as a disease-modifying therapy that may provide an organ-protective benefit.

Amyloidogenic LCs reduced cardiomyocyte viability by >50%, while non-amyloidogenic LCs did not affect viability. PROT-001 restored viability in a dose-dependent manner and selectively suppressed expression of BNP and troponin, which are markers of cardiac stress and cardiotoxicity, respectively.

**PROT-001 Demonstrates Compatibility with Bortezomib-Based Regimens While Preserving Plasma Cell Cytotoxicity (Abstract Number 32147)**
Data demonstrate that PROT-001 did not reduce the plasma cell-killing activity of bortezomib, a component of standard-of-care treatment regimens, or worsen its toxic effects on human heart cells. These findings support further evaluation of PROT-001 if used with bortezomib-based regimens.

**AmyLiteDx™: A Mass Spectrometry-Based Platform for Sensitive and In-Depth Analysis of Amyloidogenic Immunoglobulin Light Chains in AL Amyloidosis (Abstract 32645)**
Current immunoassay-based methods measure circulating LC concentration but do not directly assess LC instability. Protego's AmyLiteDx assay is a next-generation, mass spectrometry-based platform that enables deeper characterization and monitoring of unstable, toxic LCs alongside conventional biomarkers.

**Retrospective Study Evaluating Cardiac Biomarkers in Patients with AL Cardiac Amyloidosis (Abstract 32646)**
This retrospective study evaluated NT-proBNP, a biomarker of cardiac stress and toxicity, in newly diagnosed Mayo stage II-IIIb AL amyloidosis patients receiving standard-of-care therapy. The study data showed that NT-proBNP reduction in six-month cardiac organ responders was most apparent within the first 28 days, and that early cardiac response was associated with improved overall survival over the long term. These findings support investigational therapeutic approaches that can more

rapidly and potently address amyloidogenic light-chain toxicity to improve rates of early cardiac organ response.

**About Protego Biopharma**
Protego Biopharma is a clinical-stage biotechnology company developing first-in-class small molecule therapies for AL amyloidosis and other protein misfolding diseases. Protego aims to translate advances in protein stabilization into medicines that address the misfolding and toxicity responsible for progressive organ damage. Its therapies are designed to intervene in this disease process, with the goal of preserving organ function and improving patients' lives. For more information, visit www.protegobio.com.

**About PROT-001**
PROT-001 is Protego Biopharma's lead investigational small molecule pharmacological chaperone, being developed for the treatment of immunoglobulin light chain (AL) amyloidosis. PROT-001 is designed to stabilize amyloidogenic lambda light chain proteins and prevent misfolding and the formation of proteotoxic species. The goal of this therapy is to limit the toxic effects that contribute to organ injury, including direct damage to cardiomyocytes in AL patients. PROT-001 represents a complementary approach to plasma cell-directed therapies, which reduce light chain production. By targeting the amyloidogenic light chain protein before it can adopt a pathogenic conformation, PROT-001 has the potential to complement existing treatments and address an additional mechanism of disease.

**About PRO-1A-101**
PRO-1A-101 (NCT06981299) was a first-in-human, four-part Phase 1 study that evaluated the safety, tolerability, pharmacokinetics (PK), pharmacodynamics (PD), food effect (FE) and drug-drug interaction (DDI) potential of PROT-001 in healthy adult participants. Part 1 randomized participants to receive single ascending doses (SAD) of up to 1350 mg of PROT-001 or placebo; Part 2 randomized participants to receive multiple ascending doses (MAD) of up to 600 mg twice daily of PROT-001 or placebo for 14 days. Part 3 consisted of a non-randomized FE study in participants receiving one dose of PROT-001 after fasting and one dose immediately after consuming meal. Part 4 was a non-randomized, open-label study that evaluated the potential DDIs of PROT-001 on the PK of a two-cocktail probe containing digoxin and rosuvastatin.

**About AL amyloidosis**
Immunoglobulin light chain (AL) amyloidosis is a rare, life-threatening disease affecting over 5,100 newly diagnosed patients annually and 28,000 US adults in which abnormal plasma cells produce light chain proteins that can misfold and aggregate. The direct toxicity of disease-related light chains and their accumulation as amyloid deposits in tissues both result in injury to organs, including the heart and kidneys. Cardiac injury is particularly serious, as toxic light chains can directly impair cardiomyocyte function, while amyloid deposits disrupt the structure and function of

the heart. Current treatments focus on reducing light chain production with plasma cell-directed therapies and managing organ complications, leaving patients with a substantial need for complementary approaches that directly address light chain toxicity and improve organ outcomes.