News Release

Kamada Reports Additional Data from European Phase 2/3 Clinical Study of Inhaled Alpha-1 Antitrypsin to Treat AAT Deficiency

Demonstrates efficacy in a variety of lung function measurements based on post hoc statistical analyses as recommended by EMA

NESS ZIONA, Israel (April 20, 2015) – Kamada Ltd. (Nasdaq and TASE: KMDA), a plasma-derived protein therapeutics company focused on orphan indications, announces additional results from its European Phase 2/3 clinical trial of the Company's inhaled alpha-1 antitrypsin (AAT) to treat AAT deficiency (AATD). Kamada performed these post hoc analyses in accordance with guidance received following the Company's meeting with European Medicines Agency (EMA) Rapporteurs in December 2014. The goal of these analyses is to further evaluate lung function results using the most rigorous statistical methods to confirm that the efficacy signal previously identified is valid and robust.

Lung Function Results

Results from the post hoc analyses indicate that after one year of daily inhalation of Kamada's AAT, clinically and statistically significant improvements were seen in spirometric measures of lung function, particularly in bronchial airflow measurements FEV_1 (L), FEV_1% predicted and FEV_1/SVC. These favorable results were even more evident when analyzing the overall treatment effect throughout the full year.

For lung function, overall one year effect:

- FEV_1 (L) rose significantly in AAT treated patients and decreased in placebo treated patients (+15ml for AAT vs. -27ml for placebo, a 42 ml difference, p=0.0268)
- There was a trend towards better FEV_1% predicted (0.54% for AAT vs. -0.62% for placebo, a 1.16% difference, p=0.065)
- FEV_1/SVC% rose significantly in AAT treated patients and decreased in placebo treated patients (0.62% for AAT vs. -0.87% for placebo, a 1.49% difference, p=0.0074)

For lung function change at week 50 vs. baseline:

- There was a trend towards reduced FEV_1 (L) decline (-12ml for AAT vs. -62ml for placebo, a 50 ml difference, p=0.0956)

- There was a trend towards a reduced decline in FEV1% predicted (-0.1323% for AAT vs. -1.6205% for placebo, a 1.4882% difference, p=0.1032)
- FEV_1/SVC% rose significantly in AAT treated patients and decreased in placebo treated patients (0.61% for AAT vs. -1.07% for placebo, a 1.68% difference, p=0.013)

“Based on the encouraging and important lung function outcomes of the trial, we are proceeding with our plan to submit the Marketing Authorization Application (MAA) to the EMA within the coming year for Conditional Approval of our inhaled AAT to treat AAT Deficient patients. The EMA has agreed to evaluate these post hoc analyses from this innovative study,” stated David Tsur, Co-founder and CEO of Kamada. “The clinical strategy of the file will be based on primary analyses of lung functions and symptom improvements. Importantly, we believe the combination of lung functions, which are the gold standard measurements for pulmonary diseases, and symptom improvements, along with the safety profile of the product, gives us confidence that these data meet the risk/benefit balance required by EMA. We are also planning to initiate discussions this year with the U.S. Food and Drug Administration (FDA) on the regulatory pathway for registration of the product in the U.S.”

Prof. Jan Stolk, M.D., Department of Pulmonology, Leiden University Medical Center, Principal Investigator of the Phase 2/3 clinical trial and acting Chairman of the Alpha 1 International Registry (AIR), stated, "The study results demonstrated, primarily in the overall treatment effect on lung functions, are of significant clinical value. This study is the first study ever that is indicative of inhaled AAT’s ability to potentially reduce lung inflammation as expressed by its preservation of lung function and the changes shown in symptoms. As the Chairman of the Alpha 1 International Registry, I am encouraged by these results and I hope that the regulatory authorities will acknowledge the clinical significance of this study for the benefit of the AAT deficient patients in Europe, who currently in many cases are suffering from severe lung disease with an unmet medical need."

Prof. Kenneth Chapman, M.D., Director of the Canadian Registry for the Alpha-1 Antitrypsin Deficiency (Asthma and Airway Centre in Toronto Western Hospital, University of Toronto) and an investigator in the Phase 2/3 clinical trial, commented, “These new analyses confirm the clinically-meaningful lung function improvement seen with inhaled AAT patients in this study. This is the first time that a controlled, randomized trial in AAT deficiency has demonstrated efficacy using widely accepted clinical endpoints. As we know, all currently marketed therapies were approved based on pharmacokinetic data. These results are impressive and underscore the initial findings from this study. In my opinion, inhaled AAT has shown to be an efficacious treatment for this orphan disease.”

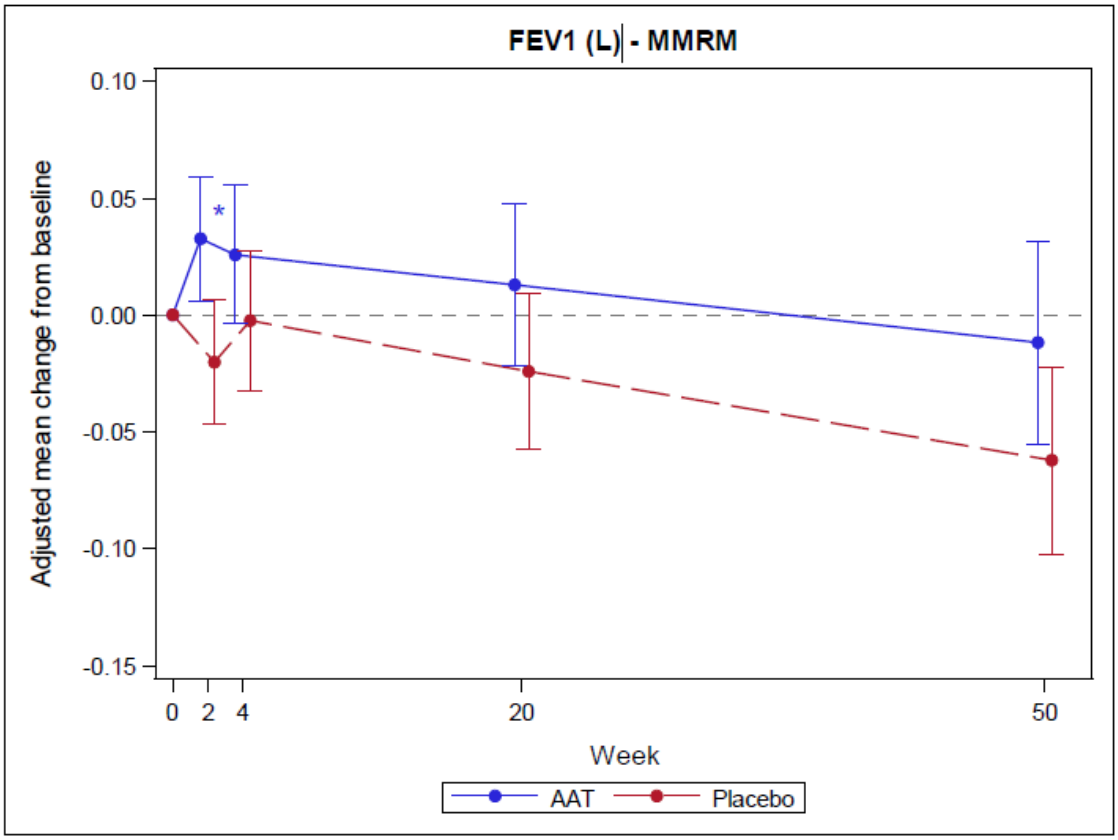
Robert A. Sandhaus, Ph.D., M.D., FCCP, Founder and Director of the Alpha1-Antitrypsin Deficiency Program at National Jewish Health in Denver, Colorado, and the Clinical Director of the Alpha-1 Foundation, expressed enthusiasm regarding the data, stating, “The study analysis suggests exciting results that may lead to wider acceptance of the inhaled route of administration of alpha-1 antitrypsin augmentation therapy, which could be a real breakthrough for AATD patients.”

The additional results after performing analyses of the data using Mixed Model Repeated Measures method (MMRM) are presented in the table below.

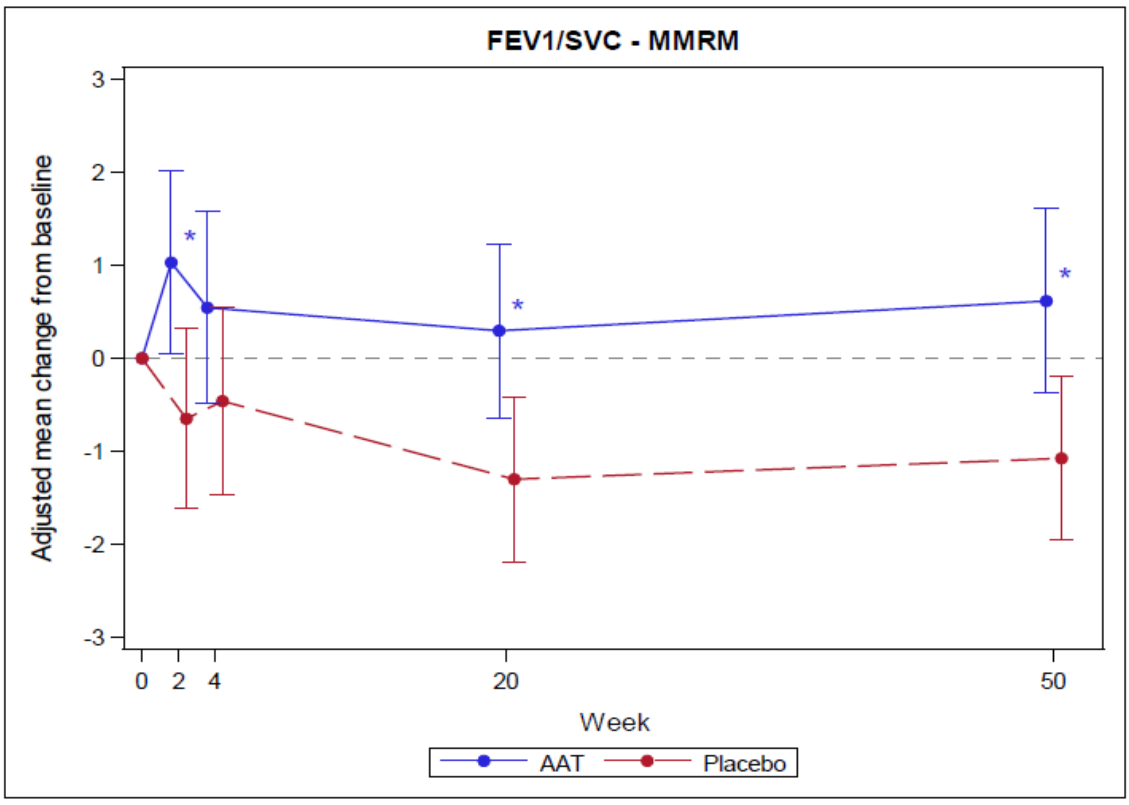
Table 1: Spirometry analyses for changes at week 50 vs. baseline and for overall treatment effect in the study [MMRM method]

Lung Function	**Least Squares Means (SEM) (Changes at Week 50 from Baseline)**		**P-Value* (Changes at Week 50)**	**Least Squares Means (SEM) (overall treatment effect)**		**P-Value* (Overall Effect)**
	AAT (n= 84)	**Placebo (n= 81)**		**AAT (n= 84)**	**Placebo (n= 81)**	
FEV_1 (L)	-0.01183 **= -12mL** (0.02196)	-0.06216 **= -62mL** (0.02036)	**0.0956**	0.01503 **= +15mL** (0.01338)	-0.02718 **= -27mL** (0.01322)	**0.0268**
FEV_1 (% of predicted)	-0.1323 (0.6649)	-1.6205 (0.6140)	0.1032	0.5404 (0.4451)	-0.6273 (0.4425)	**0.0658**
FEV_1 / SVC (%)	0.6183 (0.5015)	-1.0723 (0.4455)	**0.0132**	0.6230 (0.3931)	-0.8715 (0.3804)	**0.0074**

Graph 1: FEV_1 (L) change from baseline to week 50



Graph 2: FEV_1/ SVC change from baseline to week 50



The MMRM method takes into consideration both fixed effects and random effects, and is useful in cases where repeated measures are taken. This method is favorably accepted among regulatory bodies, especially as it is advantageous in dealing with missing values, a frequent occurrence in orphan disease trials. In addition, the regulatory authorities recommended that Kamada apply different imputation models that in aggregate may help provide a stronger indication on the robustness of the results. This included methods known as multiple imputations, jump to reference, copy reference, missing at random and others. All analysis of the spirometric measurement of lung function using those multiple imputation methods reinforced the same directional trends as seen in the table above.

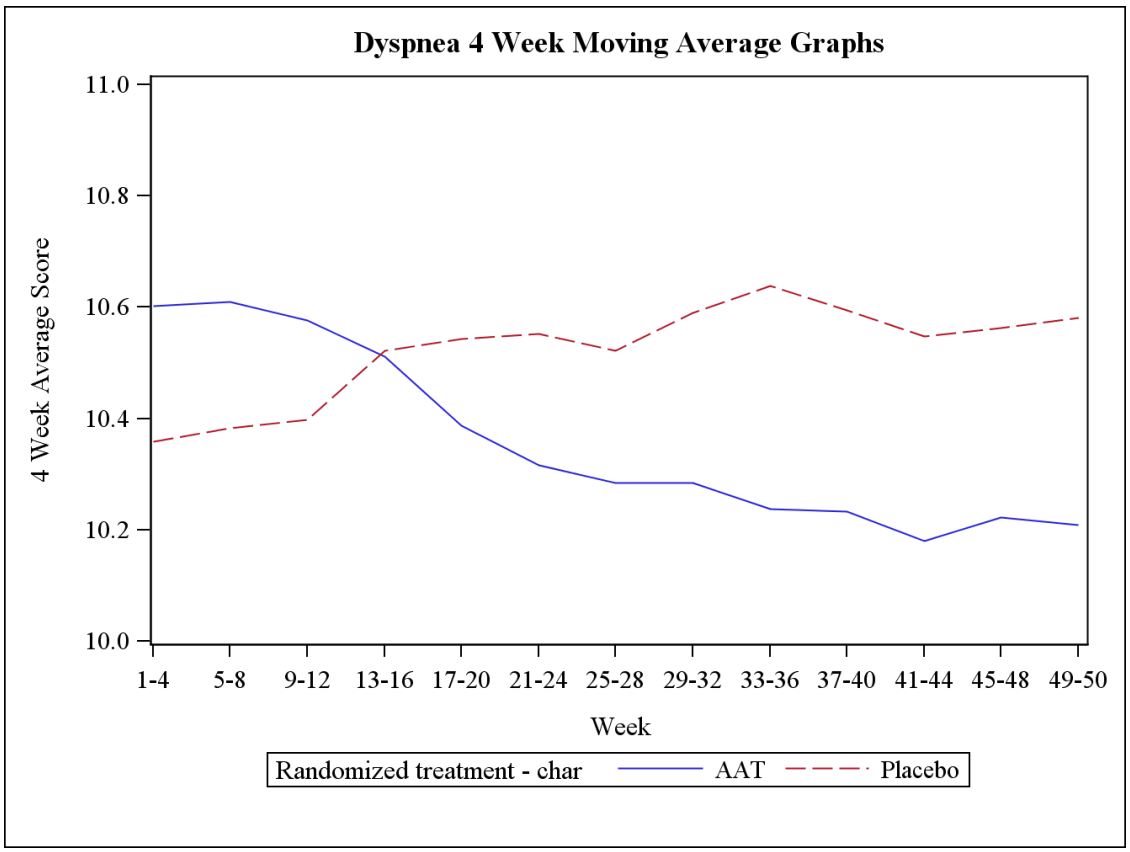
The diffusion measures in lung function didn't show differences between groups such as DLCO % of predicted and DLCO/VA % of predicted.

The diffusion capacity (DLCO) represents the gas exchange capacity of AATD patients, which is specifically affected by the extent of damage to the alveolar parenchyma in the emphysematous tissue. The scarred emphysematous tissue is not operational and is not functioning properly in the exchange of gases. Considering its anti-protease, anti-inflammatory and anti-apoptotic properties, it is likely that a longer treatment period would be required to potentially reverse tissue damage in the middle stages of the disease processes. Kamada expects that DLCO will be one of several lung function measurements evaluated during post-marketing studies.

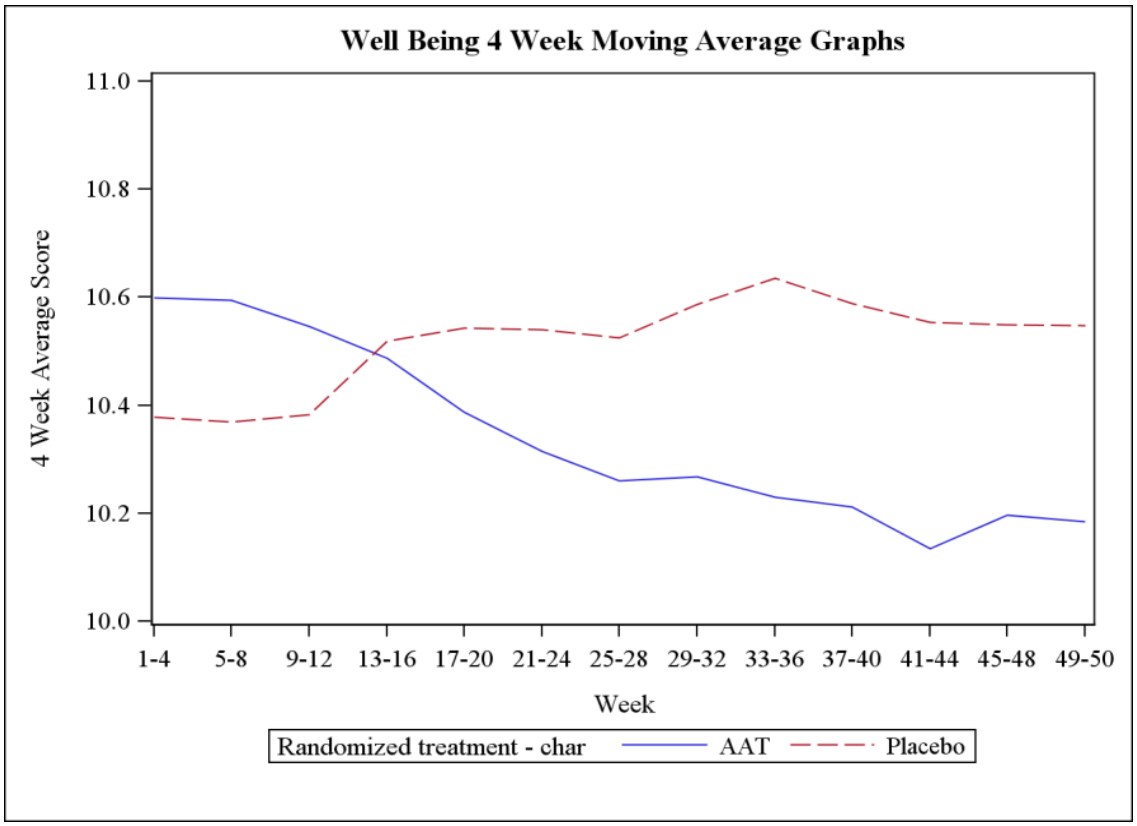
Dyspnea and Well-being Scores

Additional data collected throughout the trial for exacerbation symptom score and well-being score are shown in the following graphs. The changes in symptoms of dyspnea and well-being are suggested as those that most influence the change in patients' health, and quality of life (QoL) status and determine the need for additional therapy: the lower the score, the better the patient condition (score values are between 5 to 20). The results showed trends in favor of the AAT-treated group for both dyspnea and well-being but were not statistically significant, largely due to the small sample size.

Graph 3: Dyspnea Score, Continuous Analysis from week 1 to week 50



Graph 4: Well-being Score, Continuous Analysis from week 1 to week 50



A scoring system developed by Prof. R.A. Stockley, M.D., Professor of Medicine at Birmingham University and Medical Director of the Lung Resource Centre, Queen Elizabeth Hospital, Birmingham, U.K. and a principal investigator of the European Phase 2/3 study, was used as a monitoring tool to assess exacerbations within an electronic diary that was recorded daily by the patients. This tool (Bronkotest) includes a simple grading system to determine the magnitude of exacerbation symptoms change during the trial.

Of the three symptoms that comprise the Anthonisen Exacerbation Criteria (i.e., dyspnea, sputum volume and sputum color), dyspnea is the predominant, most common and distressing symptom in Chronic Obstructive Pulmonary Disease (COPD) and AATD. It is considered to be one of the most debilitating symptoms that has a major effect on patients' suffering,adversely affecting their quality of life. Dyspnea symptom scores were electronically captured in this study on a daily basis through the patients' e-diaries. In addition, the diary included a score for patient well-being, which is also considered to be one of the critical indicators of patients' disease state and their drive for treatment during development of an exacerbation.

According to Prof. Stockley, "This study has enlightened our understanding about the course of exacerbation events, specifically with respect to its composite symptoms, exacerbation severity and frequency with linkage to patients' baseline disease. Importantly, the improvements seen in well-being and dyspnea in the inhaled AAT treated patients suggest that in addition to lung function improvements, these patients are seeing important improvement in their symptoms, which are correlated to quality of life."

Previously Reported Data

Previous data reported by Kamada in September 2014 included:

- Exacerbations (primary and secondary endpoints of the study: Time to, Rate, Duration, Severity of the first) – No signal or significance/trend towards placebo
- Safety – No difference between groups

The improvement in dyspnea and well-being further correlates with the fact that patients inhaling AAT had better preserved airflow than patients inhaling placebo. Despite this correlation to lung function, change in exacerbation events parameters (primary and secondary endpoints) was not seen in this study, most probably as a result of a limited sample size and between-groups imbalance of other clinical parameters at baseline that affected the ability to discern changes in these exacerbation endpoints. When the study protocol was being designed, these parameters were selected as primary and secondary endpoints based on existing regulatory draft guidance and on further regulatory guidance given on several occasions. Other endpoints were considered to be less suitable given the small sample sizes available due to the orphan nature of the disease.

"Originally, this study was conducted to examine the hypothesis that AAT by inhalation may affect the duration and severity of exacerbation events in AATD patients prone to having frequent moderate-to-severe exacerbations. Having evaluated real-time data collected on disease and/or exacerbation symptoms, an important and clinically meaningful improvement in symptoms associated with the exacerbations was discovered. In particular, favorable changes observed in the two most critical parameters of dyspnea and well-being follow a similar pattern throughout the study," noted Naveh Tov, M.D., Ph.D., Medical Director of Kamada. "The graphs shown above clearly indicate that after several weeks of treatment with inhaled AAT there is an improvement in both dyspnea and well-being in the AAT group while such an improvement is not seen in the placebo group. These changes were not statistically significant between the two groups as expected in post hoc analysis, but showed a clear and consistent trend."

"We will present the study results in their entirety at a panel meeting led by dedicated Key Opinion Leaders during the American Thoracic Society (ATS) Annual Meeting next month in Denver, Colorado. This forum will provide an opportunity for the Company to receive further clinical feedback from different lung disease medical specialists and will be the first time during which we publicly discuss a conclusive set of results from our inhaled AAT trial. We are very excited by this opportunity and look forward to sharing the totality of these data in that forum," commented Pnina Strauss, Vice President for Clinical Development and Intellectual Property at Kamada. "We also look forward to sharing the results from our U.S. Phase 2 study of inhaled AAT in our follow on discussions with the EMA and to reporting additional data from the open-label extension portion of this Phase 2/3 study by year end."

About Kamada

Kamada Ltd. is focused on plasma-derived protein therapeutics for orphan indications, and has a commercial product portfolio and a robust late-stage product pipeline. The Company uses its proprietary platform technology and know-how for the extraction and purification of proteins from human plasma to produce Alpha-1 Antitrypsin (AAT) in a highly-purified, liquid form, as well as other plasma-derived proteins. AAT is a protein derived from human plasma with known and newly-discovered therapeutic roles given its immunomodulatory, anti-inflammatory, tissue-protective and antimicrobial properties. The Company's flagship product is Glassia®, the first and only liquid, ready-to-use, intravenous plasma-derived AAT product approved by the U.S. Food and Drug Administration. Kamada markets Glassia in the U.S. through a strategic partnership with Baxter International. In addition to Glassia, Kamada has a product line of nine other pharmaceutical products that are marketed through distributors in more than 15 countries, including Israel, Russia, Brazil, India and other countries in Latin America, Eastern Europe and Asia. Kamada has five late-stage plasma-derived protein products in development, including an inhaled formulation of AAT for the treatment of AAT deficiency that completed a pivotal Phase 2/3 clinical trials in Europe and has initiated Phase 2 clinical trials in the U.S. Kamada also leverages its expertise and presence in the plasma-derived protein therapeutics market by distributing 10 complementary products in Israel that are manufactured by third parties.

Cautionary Note Regarding Forward-Looking Statements

This release includes forward-looking statements within the meaning of Section 27A of the U.S. Securities Act of 1933, as amended, Section 21E of the US Securities Exchange Act of 1934, as amended, and the safe harbor provisions of the U.S. Private Securities Litigation Reform Act of 1995. Forward-looking statements are statements that are not historical facts, such as statements regarding assumptions and results related to financial results forecast, commercial results, clinical trials, Intellectual Property, the EMA and U.S. FDA filings and authorizations and timing of clinical trials. Forward-looking statements are based on Kamada's current knowledge and its present beliefs and expectations regarding possible future events and are subject to risks, uncertainties and assumptions. Actual results and the timing of events could differ materially from those anticipated in these forward-looking statements as a result of several factors including, but not limited to, unexpected results of clinical trials, delays or denial in the U.S. FDA or the EMA approval process, additional competition in the AATD market or further regulatory delays. The forward-looking statements made herein speak only as of the date of this announcement and Kamada undertakes no obligation to update publicly such forward-looking statements to reflect subsequent events or circumstances, except as otherwise required by law.

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