News Release

September 4, 2014

Kamada Reports Final Results from Phase 2/3 Clinical Trial of Inhaled Alpha-1 Antitrypsin to Treat Alpha-1 Antitrypsin Deficiency

- *Primary and secondary endpoints did not achieve statistical significance in the Intent-to-Treat population*
- *First AAT deficiency treatment to show impact on lung function*
- *Kamada continues with plans for EMA submission based on concordance of exacerbation data and positive lung function differences*
- *Conference call with Kamada management and clinical trial investigators to be held today at 8:00 a.m. Eastern time*

NESS ZIONA, Israel (September 4, 2014) – Kamada Ltd. (Nasdaq and TASE: KMDA), a plasma-derived protein therapeutics company focused on orphan indications, announces results from the complete analysis of the European Phase 2/3 clinical study of its inhaled Alpha-1 antitrypsin (AAT) therapy for the treatment of Alpha-1 antitrypsin deficiency (AATD). Following a complete analysis of the study data, the Company confirms that the study's primary endpoint of "Time to the first moderate or severe exacerbation event" did not show a statistically significant difference between inhaled AAT and placebo in the Intent-to-Treat (ITT) population, as reported in May 2014. The Company also reports that the study did not show statistically significant differences between inhaled AAT and placebo in the secondary exacerbation endpoints measured in the ITT population.

Despite not meeting the primary or secondary endpoints for the ITT population, lung function parameters, including Forced Expiratory Volume in One Second (FEV_1) % of Slow Vital Capacity (SVC), FEV_1 % predicted, FEV_1 (liters) and Diffusing capacity (DLCO), which were collected to support safety endpoints, showed concordance of a potential treatment effect in the reduction of the inflammatory injury to the lung that is known to be associated with a reduced loss of respiratory function. These effects were most apparent in the frequently exacerbating patients (the "Most Frequent Exacerbators"), who represented more than 70% of those enrolled in the study.

Notably, the Company's inhaled AAT therapy showed clinically relevant changes in various lung function measurements for the entire ITT population, as well as for the Most Frequent Exacerbators population, a few of which were statistically significant. This suggests evidence of therapeutic activity resulting in a clinically relevant and meaningful effect for the first time in studies of any AAT treatment. The importance of these findings should be viewed in the context of all existing intravenous (IV) AAT therapies, which obtained regulatory approvals on the basis of pharmacokinetic and safety data only.

Safety data of inhaled AAT in this Phase 2/3 trial remains supportive and consistent with previous inhaled AAT studies conducted by Kamada, and continues to demonstrate a high safety and tolerability profile.

Management Commentary

"We strongly believe the clinically meaningful differences seen in lung function parameters are therapeutically relevant, particularly with regard to the Most Frequent Exacerbators, who have the greatest need for effective new therapies and comprise a major portion of this orphan patient population," noted David Tsur, co-Founder and Chief Executive Officer of Kamada. *"Based on orphan designation of the drug, prior discussions held with the regulator, the strength of these data and the persistent unmet need in this orphan indication, we will advance our discussions with the European Medicines Agency with the intent to submit for conditional approval in order to bring our inhaled AAT to patients with AATD in Europe, and will initiate discussion with the FDA to determine a U.S. path for registration."*

"We are reviewing the implications of the results of the complete analysis of the Phase 2/3 study of our inhaled AAT treatment and are encouraged by the lung function changes and, more specifically, the differences seen in the Most Frequent Exacerbators. This is the first clinical trial in AATD that has provided clear indications of favorable clinical differences, including the potential for slowing disease progression, as suggested by the favorable trends seen in all lung function results, which are the gold standard measurements for pulmonary diseases," stated Pnina Strauss, Vice President of Clinical Development and Intellectual Property.

"Changes in lung function decline are challenging to demonstrate, particularly in trials among this orphan patient population that necessarily consist of relatively small patient numbers and are of short duration. In addition, both the primary and the secondary exacerbation endpoints appear to show favorable changes in the Most Frequent Exacerbators population. We believe the concordance of lung function results in this trial suggests that inhaled AAT has a therapeutic benefit and a favorable impact on disease progression for AATD patients by reducing excess inflammation. This is also relevant for other patients with lung disease who don't carry the AATD mutations and suffer from diseases associated with lung inflammation, such as COPD," noted Naveh Tov, MD, PHD, Medical Director of Kamada.

Key investigators from the study also expressed their views on the potential of inhaled AAT:

"AAT deficiency is a debilitating and often life-threatening disease that has a profound impact on healthcare costs due to multiple hospitalizations, lung transplantations, maintenance therapy, loss of work days and more. Traditionally, patients are treated with chronic obstructive pulmonary disease (COPD) therapies such as steroids, antibiotics and bronchodilators. Unfortunately, these are not effective for AATD patients as the severe lung inflammation is caused by the genetic absence of AAT protein. AAT augmentation by weekly intravenous infusion is burdensome for patients with this chronic condition that requires life-long therapy. An effective inhaled AAT therapy would provide significant clinical and convenience benefits to AATD patients. 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 trends shown in the reduction in intensity of exacerbation events. I am encouraged by these results and hope that the regulatory authorities will acknowledge the progress in clinical research demonstrated in this trial," stated Jan Stolk, MD, Department of Pulmonology, Leiden University Medical Center and Principal Investigator of the Phase 2/3 clinical trial.

"Individuals with alpha-1 antitrypsin deficiency are genetically vulnerable to the early onset of emphysema, an illness punctuated by episodic worsening and the need for hospitalization. Until now, the only specific treatment for this deficiency has been intrusive; once-per-week intravenous infusions of the deficient protein. With this announcement, Kamada gives hope to such patients – hope that with simple, twice-daily inhalation they may reduce the chance of exacerbations, diminish the likelihood of hospitalization and will protect lung function. It's a remarkable development that patients and their caregivers will want to explore further," noted Kenneth R. Chapman, MD MSc FRCPC FACP FCCP, Director, Asthma & Airway Centre, University Health Network, Professor of Medicine, University of Toronto, and an Investigator in the Phase 2/3 clinical trial.

"The Kamada inhaled AAT trial is a breakthrough study that has approached the underlying cause of AAT deficiency - lung inflammation and exacerbations caused by the genetic absence of the AAT protein. The trial results show that inhaled AAT may affect the critical element of disease progression, as suggested by the average changes in lung functions and the initial signals in exacerbation events. This was a well-conducted study that has suggested potential benefits in the most frequently exacerbating patients with a high safety and tolerability profile. It is to be hoped that this novel treatment will soon be available for the benefit of the AATD patient community," commented Robert Stockley, MB ChB. MD DSc FERS, Professor of Medicine, University Hospital Birmingham, and an Investigator in the Phase 2/3 clinical.

Phase 2/3 Clinical Trial Results

For the Primary Endpoint of "Time to the first moderate or severe exacerbation event," the Survival Curve (Kaplan Meier Curve) and hazard ratio in the ITT population did not show a difference between the inhaled AAT group and the placebo group. Yet, the concordant clinical and functional differences observed with regards to changes in exacerbation events for primary and secondary endpoints in the Most Frequent Exacerbators provide an indication of therapeutic activity not detected in previous AAT clinical studies.

The primary endpoint of "Time to the first moderate or severe exacerbation event" in the Most Frequent Exacerbators suggested a favorable clinical difference between inhaled AAT treatment (n=67) and placebo (n=54), as reflected by the Survival Curve and hazard ratio 0.877 (95% CI 0.563, 1.364; p-value=NS[1]).

No differences were found for the ITT population between the groups for the secondary endpoint of "Time to first event-based exacerbation with a severity of mild, moderate or severe", yet for the Most Frequent Exacerbators, the median time until the first event was longer in the inhaled

[1] Not statistically significant as defined by p value > 0.05

AAT group compared with the placebo group. Hazard Ratio = 1.064 (95% CI 0.717, 1.578; p-value=NS).

Regarding the secondary endpoint of "Severity of the first exacerbation event" in the ITT population, the inhaled AAT group experienced a lower percentage of first severe ("Type 1") exacerbation events compared with the placebo group (18.8% and 31.1%, respectively; p-value=NS). For the same exacerbation definition, a lower percentage of first severe exacerbation events was also observed in the Most Frequent Exacerbators compared with placebo (19.4% and 35.2%, respectively; p-value=NS) as well as for both severe and moderate exacerbation events compared with placebo (44.8% and 51.9%, respectively; p-value=NS). These differences were also evident in the event-based definition. In the ITT population, the percentage of the first moderate event was lower in the inhaled AAT group compared with the placebo group (56.5% and 63.9%, respectively; p-value=NS); in the Most Frequent Exacerbators the percentage of the first moderate exacerbation events was 15.1% lower in the inhaled AAT group compared with the placebo group, and the percentage of both moderate or severe first exacerbation events was 4.7% lower in the inhaled AAT group than in the placebo group. These findings were not statistically significant.

For the secondary endpoint of "Rate of exacerbation events as reflected by the number of event-based exacerbation events in the study," there was no difference between the inhaled AAT group and the placebo group in the ITT population. There has been no difference for the number of severe exacerbation events in the study. However**,** for the Most Frequent Exacerbators there was a reduction of 10% in the inhaled AAT group compared with the placebo group in all types of exacerbation events (number of exacerbation events including mild/ moderate/ severe), a reduction of 13% in the number of moderate event-based exacerbations and a reduction of 12% in the number of moderate/ severe event-based exacerbations. These findings were not statistically significant.

With regard to lung function data in the ITT population that was measured under the safety endpoints, the FEV_1 % of SVC, mean change from baseline in the inhaled AAT group at study week 50 showed a statistically significant increase compared with a decline in the placebo group (+0.34% and -1.17%, respectively; p-value=0.033).

Positive concordant clinical pattern trends were seen in all other lung function tests in favor of inhaled AAT in the ITT population.

For FEV_1 % predicted, the placebo decline was more than two-fold greater than that in the inhaled AAT treated population (-1.37 vs. -0.509, respectively; p-value=NS). Similarly, FEV_1 (liters) showed a steeper decline in the placebo group compared with the inhaled AAT group (-52ml and -25ml, respectively; p-value= NS).

The diffusing capacity (DLCO [mMol/min/Kpa]) in the inhaled AAT group declined less than in the placebo group (-0. 168 and -0.28, respectively; p-value=NS).

The inhaled AAT group of Most Frequent Exacerbators also showed favorable results across all lung function measurements. A statistically significant difference was seen for FEV_1 % of SVC

with an increase in the inhaled AAT group compared with a decline in the placebo group, as expressed by the mean change from baseline at week 50 placebo (+0.2251% and -1.68%, respectively; p-value=0. 0208). FEV_1 % predicted declined less compared with the placebo group (-0.58 vs. -1.21, respectively; p-value=NS). FEV_1 (liters) decreased less in the AAT group compared with placebo group (-18ml and. -51ml, respectively). These changes were not statistically significant at week 50, though concordant with other spirometry data. Diffusing capacity (DLCO [mMol/min/Kpa]) showed a greater decline in placebo patients compared with inhaled AAT patients (-0.336% and -0.206%, respectively). Although this change was not statistically significant at week 50, it remains concordant with the spirometry data.

"We remain committed to AATD patients worldwide and to maintaining our leadership role in the development of innovative new therapies for this orphan lung disease. As such, we remain steadfast in conducting continued clinical work in support of our inhaled AAT, including our ongoing open-label extension study in Europe and Phase 2 U.S. study, as well as other future studies that would support its global licensure and expand its use into other lung diseases. Moreover, these very promising data put us in an attractive position as we advance our ongoing discussions with partners for potential licensing opportunities in markets outside of Europe," added Mr. Tsur.

"As always, we appreciate the support of the Alpha-1 Antitrypsin Foundation and thank our principal investigators and patients for their commitment to this study. We look forward to the opportunity to present the complete data set at an upcoming medical conference and to having it published in a peer-reviewed journal. More importantly, if approved by the EMA, this is an exciting opportunity for Kamada to bring the first inhaled therapy to patients suffering from this debilitating, life-threatening, orphan lung disease," concluded Mr. Tsur.

Kamada management will be participating at the European Respiratory Society's (ERS) International Congress 2014, taking place September 6-10, 2014 at the Internationales Congress Center München in Munich, Germany and are available to further discuss these results with those in attendance. Interested investors attending ERS may contact Anne Marie Fields, Senior Vice President at LHA, at 212-838-3777 or at afields@lhai.com to arrange a meeting with Kamada management during the Congress.

Phase 2/3 Clinical Trial Design

The multicenter randomized, double-blind, placebo-controlled study evaluated the safety and efficacy of Kamada's inhaled formulation of human AAT to treat AATD in 168 patients. The study involved the daily inhalation of 160 mg of human AAT or placebo via the eFlow® device for 50 weeks. The primary endpoint of the study was "Time to the first moderate or severe exacerbation event," calculated by continuous and real-time clinical evaluation from the time of randomization until the end of treatment. Secondary endpoints included additional parameters of exacerbation events (i.e., severity, rate, cardinal symptoms reported). The trial also measured lung function data, safety and additional exploratory endpoints that included CT densitometry in a subset of subjects, Quality of Life measurements, piM protein levels in serum and more.

Eligible patients from this Phase 2/3 trial were given the option to participate in a follow-up 50-week, open-label safety study. A majority of eligible patients consented to participate in this study. The Company expects to use the additional data from this follow-up study as part of its regulatory submissions.

Commercialization of Inhaled AAT for the Treatment of AATD

In August 2012 Kamada signed an exclusive agreement for the distribution of its inhaled AAT for the treatment of AATD in Europe with Chiesi Farmaceutici S.p.A., a fully integrated European pharmaceutical company focused on respiratory diseases and special care products. Under the agreement, Kamada is eligible to receive milestone payments of up to $60 million, subject to achievement of certain regulatory and sales targets.

Recently, Kamada initiated a U.S. Phase 2 clinical trial with its inhaled AAT for AATD, and expects that the data from the European trial together with the data from the U.S trial will provide the foundation from which to develop its regulatory strategy for licensure in the U.S.

Conference Call

Kamada management will host a conference call today at 8:00 a.m. Eastern time, featuring several clinical trial investigators who are experts in AATD, to discuss these results and to answer investor questions. Shareholders and other interested parties may participate in the call by dialing (888) 803-5993 (domestic), 1-809-45-7877 (from Israel) or (706) 634-5454 (international) and referencing conference ID number 95044902. The call will also be webcast live and archived on the Company's website at www.kamada.com.

A replay of the conference call will be accessible beginning two hours after its completion through September 10, 2014, by dialing (855) 859-2056 (domestic) or (404) 537-3406 (international) and referencing conference ID number 95044902. The call will also be archived for 90 days on the Company's website at www.kamada.com.

About Alpha-1 Antitrypsin Deficiency

Alpha-1 antitrypsin, also called AAT, is a protein made in the liver. Normally the protein travels through the bloodstream and helps protect the body's organs from the harmful effects of other proteins. The lungs are one of the main organs that the AAT protein protects. AAT deficiency (AATD or inherited emphysema) occurs if the AAT proteins made in the liver are not the right shape, and they get stuck inside liver cells and cannot get into the bloodstream. As a result, not enough AAT proteins travel to the lungs to protect them, which increase the risk of lung disease. Also, liver disease can develop because too many AAT proteins are stuck in the liver. Severe AATD occurs when blood levels of the AAT protein fall below the lowest amount needed to protect the lungs.

AATD is an inherited condition that occurs in all ethnic populations, yet most often in Caucasians of European descent. It is not known how many people have AAT deficiency and many people who have the condition may not know they have it. According to the National

Institutes of Health, estimates of disease incidence range from about 1 in every 1,600 people to about 1 in every 5,000 people.

About eFlow® Technology and PARI Pharma
The Company's inhaled AAT is delivered by an investigational eFlow® Nebulizer System developed by PARI Pharma and optimized specifically for Kamada. The optimized device uses eFlow Technology to enable highly efficient aerosolization of medication including liposomal formulations via a vibrating, perforated membrane that includes thousands of laser-drilled holes. Compared with other nebulization technologies, eFlow Technology produces aerosols with a very high density of active drug, a precisely defined droplet size and a high proportion of respirable droplets delivered in the shortest possible period of time. eFlow Technology is not an ultrasonic nebulizer technology and is not a general purpose electronic aerosol generator nebulizer technology. Combined with its quiet mode of operation, small size, light weight and battery use, eFlow Technology reduces the burden of taking daily, inhaled treatments.

About Chiesi Farmaceutici
Chiesi Farmaceutici is a research-focused international group, with nearly 80 years of experience headquartered in Parma (Italy). Chiesi researches, develops and commercializes innovative pharmaceutical solutions in the respiratory therapeutics, specialist medicine and rare diseases areas. In 2013, Chiesi achieved sales of over 1.2 billion Euros, constituting double digit growth over 2012. Its R&D centers in Parma (Italy), Paris (France), Rockville (USA), Chippenham (UK) and the R&D team of the newly-acquired Danish company Zymenex, integrate their efforts to advance Chiesi's pre-clinical, clinical and registration programs. The Chiesi Group employs approximately 3900 people, 480 of which are dedicated to R&D activities.
For more information, please visit www.chiesi.com.

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 trial in Europe and has initiated Phase II 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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