UNITED STATES
SECURITIES AND EXCHANGE COMMISSION
Washington, DC 20549

FORM 10-Q

(Mark One)

☑ **QUARTERLY REPORT PURSUANT TO SECTION 13 OR 15(d) OF THE SECURITIES EXCHANGE ACT OF 1934**

For the quarterly period ended September 30, 2016
or

☐ **TRANSITION REPORT PURSUANT TO SECTION 13 OR 15(d) OF THE SECURITIES EXCHANGE ACT OF 1934**

For the transition period from _____ to _____

Commission file number **001-15771**

ABEONA THERAPEUTICS INC.

(Exact name of registrant as specified in its charter)

Delaware	**83-0221517**
(State or other jurisdiction of incorporation or organization)	(I.R.S. Employer I.D. No.)

3333 Lee Parkway, Suite 600, Dallas, TX 75219
(Address of principal executive offices)

(214) 665-9495
(Registrant's telephone number, including area code)

N/A
(Former name, former address and former fiscal year, if changed since last report)

Indicate by check mark whether the registrant: (1) has filed all reports required to be filed by Section 13 or 15(d) of the Securities Exchange Act of 1934 during the preceding 12 months (or for such shorter period that the registrant was required to file such reports), and (2) has been subject to such filing requirements for the past 90 days.
Yes ☑ No ☐

Indicate by check mark whether the registrant has submitted electronically and posted on its corporate Web site, if any, every Interactive Data File required to be submitted and posted pursuant to Rule 405 of Regulation S-T (§232.405 of this chapter) during the preceding 12 months (or for such shorter period that the registrant was required to submit and post such files). Yes ☑ No ☐

Indicate by check mark whether the registrant is a large accelerated filer, an accelerated filer, a non-accelerated filer or a smaller reporting company. See the definitions of "large accelerated filer" "accelerated filer" and "smaller reporting company" in Rule 12b-2 of the Exchange Act. (Check one):

Large accelerated filer ☐	Accelerated filer ☐	Non-accelerated filer ☐ (Do not check if a smaller reporting company)	Smaller reporting company ☑

Indicate by check mark whether the registrant is a shell company (as defined in Rule 12b-2 of the Exchange Act). Yes ☐ No ☑

Indicate the number of shares outstanding of each of the issuer's classes of common stock, as of the latest practicable date.

The number of shares outstanding of the registrant's common stock as of November 14, 2016 was 39,959,568 shares.

ABEONA THERAPEUTICS INC.

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PART I –FINANCIAL INFORMATION

This Quarterly Report on Form 10-Q (including the information incorporated by reference) contains ''forward-looking statements'' within the meaning of Section 27A of the Securities Act of 1933, as amended, and Section 21E of the Securities Exchange Act of 1934, as amended, and that involve risks and uncertainties. These statements and other risks described below as well as those discussed elsewhere in this Quarterly Report Form 10-Q, documents incorporated by reference and other documents and reports that we file periodically with the Securities and Exchange Commission ("SEC") include, without limitation, statements relating to uncertainties associated with research and development activities, clinical trials, our ability to raise capital, the timing of and our ability to achieve regulatory approvals, dependence on others to market our licensed products, collaborations and our ability to attract licensing partners, future cash flow, the future success of our marketed products and products in development, our belief that advances in biotechnology will provide significant opportunities to develop new treatments for rare diseases, our sales projections, and the sales projections of our licensing partners, the size of the prospective markets in which we may offer products, anticipated product launches and our commercialization strategies, anticipated product approvals and timing thereof, product opportunities, clinical trials and U.S. Food and Drug Administration (''FDA'') applications, as well as our drug development strategy, our clinical development organization expectations regarding our rate of technological developments and competition, that we expect that a more complete analysis of ABO-102 (AAV-SGSH) data will be presented from the low-dose cohort and initial high dose cohort at a scientific conference in the first quarter of 2017 our expectations regarding minimizing development risk and developing and introducing technology, the terms of future licensing arrangements, our ability to secure additional financing for our operations, our ability to establish new relationships and maintain current relationships, our ability to attract and retain key personnel, our belief that we will not pay any cash dividends in the foreseeable future, our belief that a failure to obtain necessary additional capital in the future will result in our operations being jeopardized, our belief that we have a rich pipeline of products and product candidates, our belief that recently licensed technology will enable us to provide new therapeutic applications and expand market opportunities while enhancing margins, our expectations to incur losses for the next several years as we continue to invest in product research and development, preclinical studies, clinical trials and regulatory compliance, our ability to achieve profitability on a sustained basis or at all, our expected cash burn rate. These statements relate to future events or our future financial performance. In some cases, you can identify forward-looking statements by terminology such as "may," "will," "should," "expects," "plans," "could," "anticipates," "believes," "estimates," "predicts," "potential" or "continue" or the negative of such terms or other comparable terminology. We intend the forward-looking statements to be covered by the safe harbor for forward-looking statements in these sections. The forward-looking information is based on various factors and was derived using numerous assumptions.

Forward-looking statements necessarily involve risks and uncertainties, and our actual results could differ materially from those anticipated in the forward-looking statements due to a number of factors. The forward-looking statements contained in this Quarterly Report on Form 10-Q represent our judgment only as of the date of this report. We caution readers not to place undue reliance on such statements. Except as required by law, we undertake no obligation to update publicly any forward-looking statements for any reason, even if new information becomes available or other events occur in the future.

ITEM 1. FINANCIAL STATEMENTS

The response to this Item is submitted as a separate section of this report. See page 17.

ITEM 2. MANAGEMENT'S DISCUSSION AND ANALYSIS OF FINANCIAL CONDITION AND RESULTS OF OPERATIONS

OVERVIEW

Abeona Therapeutics Inc. (together with our subsidiaries, "we", "our", "Abeona" or the "Company") is a Delaware corporation. We are a clinical stage biopharmaceutical company developing gene and plasma-based therapies for life-threatening rare genetic diseases. Our lead programs are ABO-102 (AAV-SGSH) and ABO-101 (AAV-NAGLU), adeno-associated virus (AAV) based gene therapies for Sanfilippo syndrome (MPS IIIA and IIIB, respectively). We are also developing EB-101 (gene-corrected skin grafts) for recessive dystrophic epidermolysis bullosa (RDEB), EB-201 for epidermolysis bullosa (EB), ABO-201 (AAV-CLN3) gene therapy for juvenile Batten disease (JNCL), ABO-202 (AAV-CLN1) gene therapy for treatment of infantile Batten disease (INCL), and ABO-301 (AAV-FANCC) for Fanconi anemia (FA) disorder and ABO-302 using a novel CRISPR/Cas9-based gene editing approach to gene therapy for rare blood diseases. In addition, we have a plasma-based protein therapy pipeline, including SDF Alpha™ (alpha-1 protease inhibitor) for inherited COPD, using our proprietary SDFTM (Salt Diafiltration) ethanol-free process. Our principal executive office is located at 3333 Lee Parkway, Suite 600, Dallas, Texas 75219. Our website address is *www.abeonatherapeutics.com*.

Recent Developments

On November 1, 2016, we closed an underwritten public offering of 6,000,000 shares of common stock, at a public offering price of $7.00 per share. The gross proceeds to the Company were $42,000,000, before deducting the underwriting discounts and commissions and estimated offering expenses payable by the Company.

On October 25, 2016, we announced that the U.S. Food and Drug Administration (FDA) granted Fast Track designation for ABO-102, a single intravenous injection of AAV gene therapy for subjects with MPS IIIA (Sanfilippo syndrome type A).

On October 20, 2016, we announced an update on clinical results through 30 days post-injection for the completed low-dose cohort (n=3) in the ongoing Phase 1/2 trial for ABO-102 (AAV-SGSH). The ongoing Phase 1/2 study is designed to evaluate safety and preliminary indications of efficacy of ABO-102 in subjects suffering from Mucopolysaccharidosis Type A (MPS IIIA or Sanfilippo syndrome type A). Observations 30 days post-injection for the low dose cohort demonstrated:

- ABO-102 was well-tolerated in subjects injected with the low dose of 5E12 vg/kg ABO-102 with no treatment related adverse events or serious adverse events (SAEs). Following favorable review of the safety data by the independent Data Safety Monitoring Board (DSMB), enrollment in the high dose cohort has commenced.
- In the natural history study evaluating MPS III subjects, urine and cerebral spinal fluid GAG (heparan sulfate or "HS") were significantly elevated in the subject population as a symptom of disease pathology.
- All subjects in the low-dose cohort experienced reductions from baseline in both urinary HS and CSF. At 30 days post-injection, urinary HS reduction was 57.6% +/- 8.2%. Reduction in CSF HS was 25.6% +/- 0.8%, suggesting that ABO-102 crossed the blood brain barrier after intravenous administration.

- The natural history study in 25 subjects with MPS III (*Truxal et. al., 2016, Mol. Genet. Metab*.) demonstrated that study subjects had increased liver and spleen volumes averaging 116% and 88%, respectively, at baseline that did not change over a year of follow up.
- All three subjects demonstrated significant reductions in liver volume (17.7% +/- 1.9%), and spleen volume (17.6% +/- 7.1%) from baseline, as measured by MRI at 30 days post-injection.

We expect that a more complete analysis of these data will be presented from the low-dose cohort and initial high-dose cohort at a scientific conference in the first quarter of 2017. The Data Safety Monitoring Board approved dose escalation of the high-dose cohort in the fourth quarter of 2016.

On October 18, 2016, we announced that the European Medicines Agency (EMA) Committee for Orphan Medicinal Products has granted Orphan Drug Designation for our lead gene therapy program ABO-102 for the treatment of patients with Sanfilippo syndrome type A (MPS IIIA).

On October 7, 2016, we announced that preclinical data supporting clinical trials for ABO-201 (AAV-CLN3), the AAV-based single intravenous gene therapy program for juvenile Batten disease, (juvenile neuronal ceroid lipofuscinosis, JNCL), were published in the September issue of the Journal of Neuroscience. Researchers concluded that a single intravenous injection "led to widespread virus biodistribution in the brain, spinal cord, and eye" that was capable of "improving motor function, attenuating microglial and astrocyte activation, and reducing lysosomal pathology, all hallmarks of JNCL" at an age when significant lysosomal pathology had already manifested.

On September 26, 2016, we announced that the first patient was enrolled in the Phase 2 portion of the clinical trial for EB-101 (gene-corrected skin grafts).

On September 21, 2016, we announced the exclusive worldwide license of a next-generation gene therapy AAV capsid portfolio from University of North Carolina at Chapel Hill. The AIM™ vector system is a next generation platform of AAV capsids capable of widespread central nervous system gene transfer and can be used to confer high transduction efficiency for various therapeutic indications. Studies indicate that AIM vectors can efficiently and broadly target CNS tissue, and may provide a treatment for patients that have inhibitory antibodies to natural AAV serotypes. Importantly, the AIM vector system may provide second-generation treatment approaches for patients that have received a previous AAV injection.

On September 8, 2016, we announced that the fifth patient was enrolled in the Phase 1/2 clinical trial for EB-101 (gene-corrected skin grafts). The Phase 1/2 clinical trial with gene-corrected skin grafts has shown promising wound healing and safety in patients with RDEB. Investigators at Stanford are now expanding enrollment to adolescent patients for the Phase 1/2 trial to determine the safety and efficacy of COL7A1 gene-corrected grafts on wound healing efficacy. Clinical data on the initial four patients in the Phase 1/2 trial were recently presented at the opening Plenary Session of the Society for Investigative Dermatology.

On August 9, 2016, we announced, along with the EB Research Partnership and EB Medical Research Foundation, a collaboration for the development of treatments for recessive dystrophic epidermolysis bullosa (RDEB). Clinical results for the lead EB program (EB-101) were presented at the opening Plenary Session of the Society for Investigative Dermatology in May 2016, and Investigators at Stanford are recruiting patients for a Phase 2 clinical trial of EB-101 in adolescents age 13 and older to determine the effect of type VII collagen gene corrective grafts on wound healing efficacy.

Product Development Strategy

Abeona is focused on developing and delivering gene therapy and plasma-based products for severe and life-threatening rare diseases. A rare disease is one that affects fewer than 200,000 people in the United States. There are nearly 7,000 rare diseases, which may involve chronic illness, disability, and often, premature death. More than 25 million Americans and 30 million Europeans have one. While rare diseases can affect any age group, about 50% of people affected are children (15 million) and rare diseases account for 35% of deaths in the first year of life. These rare diseases are often poorly diagnosed, very complex, and have no treatment or not very effective treatment — over 95% of rare diseases do not have a single FDA or EMA approved drug treatment. However, most rare diseases are often caused by changes in genes — approximately 80% are genetic in origin and can present at any stage of life. We believe emerging insights in genetics and advances in biotechnology, as well as new approaches and collaboration between researchers, industry, regulators and patient groups, provide significant opportunities to develop breakthrough treatments for rare diseases.

Developing Next Generation Gene Therapy

Gene therapy is the use of DNA as a potential therapy to treat a disease. In many disorders, particularly genetic diseases caused by a single genetic defect, gene therapy aims to treat a disease by delivering the correct copy of DNA into a patient's cells. The healthy, functional copy of the therapeutic gene then helps the cell function correctly. In gene therapy, DNA that encodes a therapeutic protein is packaged within a "vector", often a "naked" virus, which is used to transfer the DNA to the inside of cells within the body. Gene therapy can be delivered by a direct injection, either intravenously (IV) or directly into a specific tissue in the body, where it is taken up by individual cells. Once inside cells, the correct DNA is expressed by the cell machinery, resulting in the production of missing or defective protein, which in turn is proposed to treat the patient's underlying disease and can provide long-term benefit.

Abeona is developing next-generation adeno-associated virus (AAV) gene therapies. Viruses such as AAV are utilized because they have evolved a way of encapsulating and delivering one or more genes of the size needed for clinical application, and can be purified in large quantities at high concentration. Unlike AAV vectors found in nature, the AAV vectors used by Abeona have been genetically-modified such that they do not replicate. Although the preclinical studies in animal models of disease demonstrate the promising impact of AAV-mediated gene expression to affected tissues such as the heart, liver and muscle, our programs use a specific virus that is capable of delivering therapeutic DNA across the blood brain barrier and into the central nervous system (CNS) and the somatic system (body), making them attractive for addressing lysosomal storage diseases which have severe CNS manifestations of the disease.

Lysosomal storage diseases (LSDs) are a group of rare inborn errors of metabolism resulting from deficiency in normal lysosomal function. These diseases are characterized by progressive accumulation of storage material within the lysosomes of affected cells, ultimately leading to cellular dysfunction. Multiple tissues ranging from musculoskeletal and visceral to tissues of the central nervous system are typically involved in disease pathology. Since the advent of enzyme replacement therapy (ERT) to manage some LSDs, general clinical outcomes have significantly improved; however, treatment with infused protein is lifelong and continued disease progression is still evident in patients. Thus, AAV-based gene therapy may provide a viable alternative or adjunctive therapy to current management strategies for LSDs.

Our initial programs are focused on LSDs such as Mucopolysaccharidosis (MPS) IIIA and IIIB. MPS III is also known as Sanfilippo syndromes type A and type B, is a progressive neuromuscular disease with profound CNS involvement. Our lead product candidates, ABO-101 and ABO-102, have been developed to replace the damaged, malfunctioning enzymes within target cells with the normal, functioning version. ABO-201 is a similar product, using an AAV to deliver the correct lysosomal gene that is defective in juvenile neuronal ceroid lipofuscinosis. Delivered via a single injection, these drugs are only given once.

ABO-101 for MPS III B and ABO-102 for MPS III A (Sanfilippo syndrome)

Mucopolysaccharidosis (MPS) type III (Sanfilippo syndrome) is a group of four inherited genetic diseases, described as type A, B, C or D, which cause enzyme deficiencies that result in the abnormal accumulation of glycosaminoglycans (sugars) in body tissues. MPS III is a lysosomal storage disease, a group of rare inborn errors of metabolism resulting from deficiency in normal lysosomal function. The incidence of MPS III (all four types combined) is estimated to be 1 in 70,000 births.

Mucopolysaccharides are long chains of sugar molecules used in the building of connective tissues in the body. There is a continuous process in the body of replacing used materials and breaking them down for disposal. Children with MPS III are missing an enzyme which is essential in breaking down the used mucopolysaccharides. The partially broken down mucopolysaccharides remain stored in cells in the body causing progressive damage. Babies may show little sign of the disease, but as more and more cells become damaged, symptoms start to appear.

In MPS III, the predominant symptoms occur due to accumulation within the central nervous system (CNS), including the brain and spinal cord, resulting in cognitive decline, motor dysfunction, and eventual death. To date, there is no cure for MPS III and treatments are largely supportive.

Abeona is developing next-generation AAV-based gene therapies for MPS III (Sanfilippo syndrome), which involves a one-time delivery of a normal copy of the defective gene to cells of the central nervous system with the aim of reversing the effects of the genetic errors that cause the disease.

After a single dose in Sanfilippo preclinical models, ABO-101 and ABO-102 induced cells in the CNS and peripheral organs to produce the missing enzymes which helped repair the damage caused to the cells. Preclinical *in vivo* efficacy studies in Sanfilippo syndrome have demonstrated functional benefits that remain for months after treatment. A single dose of ABO-101 or ABO-102 significantly restored normal cell and organ function, corrected cognitive defects that remained months after drug administration, increased neuromuscular control and increased the lifespan of animals with MPS III over 100% one year after treatment compared to untreated control animals. These results are consistent with studies from several laboratories suggesting AAV treatment could potentially benefit patients with Sanfilippo Syndrome Type A and B. In addition, safety studies conducted in animal models of Sanfilippo syndromes have demonstrated that delivery of AB0-101 or AB0-102 are well tolerated with minimal side effects.

ABO-201 for Juvenile Neuronal Ceroid Lipofuscinoses (JNCL) (or Juvenile Batten Disease (JBD)) and ABO-202 (AAV-CLN1) gene therapy for treatment of infantile Batten disease (INCL)

ABO-201 (AAV CLN3) is an AAV-based gene therapy which has shown promising preclinical efficacy in delivery of a normal copy of the defective CLN3 gene to cells of the central nervous system with the aim of reversing the effects of the genetic errors that cause JNCL. JNCL is a rare, fatal, autosomal recessive (inherited) disorder of the nervous system that typically begins in children between 4 and 8 years of age. Often the first noticeable sign of JNCL is vision impairment, which tends to progress rapidly and eventually result in blindness. As the disease progresses, children experience the loss of previously acquired skills (developmental regression). This progression usually begins with the loss of the ability to speak in complete sentences. Children then lose motor skills, such as the ability to walk or sit. They also develop movement abnormalities that include rigidity or stiffness, slow or diminished movements (hypokinesia), and stooped posture. Beginning in mid- to late childhood, affected children may have recurrent seizures (epilepsy), heart problems, behavioral problems, and difficulty sleeping. Life expectancy is greatly reduced. Most people with juvenile Batten disease live into their twenties or thirties. As yet, no specific treatment is known that can halt or reverse the symptoms of JNCL disease.

JNCL disease is the most common form of a group of disorders known as neuronal ceroid lipofuscinoses (NCLs). Collectively, all forms of NCL affect an estimated 2 to 4 in 100,000 live births in the United States. NCLs are more common in Finland, where approximately 1 in 12,500 individuals are affected, as well as Sweden, other parts of northern Europe, and Newfoundland, Canada.

Most cases of JNCL disease are caused by mutations in the CLN3 gene, which is the focus of our AAV-based gene therapy approach. These mutations disrupt the function of cellular structures called lysosomes. Lysosomes are compartments in the cell that normally digest and recycle different types of molecules. Lysosome malfunction leads to a buildup of fatty substances called lipopigments and proteins within these cell structures. These accumulations occur in cells throughout the body, but neurons in the brain seem to be particularly vulnerable to damage. The progressive death of cells, especially in the brain, leads to vision loss, seizures, and intellectual decline in children with JNCL disease.

ABO-202 (AAV9 CLN1) is an AAV-based gene therapy which has shown promising preclinical efficacy in delivery of a normal copy of the defective CLN1 gene to cells of the central nervous system with the aim of reversing the effects of the genetic errors that cause an infantile form of Batten disease (also known as infantile neuronal ceroid lipofuscinosis).

ABO-301 for Fanconi Anemia (FA) and ABO-302 for rare blood diseases using a novel CRISPR/Cas9-based gene editing approach to gene therapy for rare blood diseases

ABO-301 (AAV FANCC) is an AAV-based gene therapy which has shown promising preclinical efficacy in delivery of a normal copy of the defective gene to cells of the hematopoietic or blood system with the aim of reversing the effects of the genetic errors that cause Fanconi anemia (FA). FA is a rare (1 in 160,000) pediatric, autosomal recessive (inherited) disease characterized by multiple physical abnormalities, organ defects, bone marrow failure, and a higher than normal risk of cancer. The average lifespan for people with FA is 20 to 30 years.

The major function of bone marrow is to produce new blood cells. In FA, a DNA mutation renders the FANCC gene nonfunctional. Loss of FANCC causes patient skeletal abnormalities and leads to bone marrow failure. FA patients also have much higher rates of hematological diseases, such as acute myeloid leukemia (AML) or tumors of the head, neck, skin, gastrointestinal system, or genital tract. The likelihood of developing one of these cancers in people with FA is between 10 and 30 percent. Aside from bone marrow transplantation (BMT), there are no specific treatments known that can halt or reverse the symptoms of FA. Repairing fibroblast cells in FA patients with a functional FANCC gene is the focus of our AAV-based gene therapy approach.

Using a novel CRISPR (clustered, regularly interspaced short palindromic repeats)-Cas9 (CRISPR associated protein 9) system, researchers used a protein-RNA complex composed of an enzyme known as Cas9 bound to a guide RNA molecule that has been designed to recognize a particular DNA sequence. The RNA molecules guide the Cas9 complex to the location in the genome that requires repair. CRISPR-Cas9 uniquely enables surgically efficient knock-out, knock-down or selective editing of defective genes in the context of their natural promoters, unlocking the potential to treat both recessive and dominant forms of genetic diseases. Most importantly, this approach has the potential to allow more precise gene modification.

EB-101 For the Treatment of Recessive Dystrophic Epidermolysis Bullosa and EB-201 For the Correction of Gene Mutations in Skin Cells (Keratinocytes)

EB-101 (LZRSE-Col7A1 Engineered Autologous Epidermal Sheets (LEAES)), is an ex vivo gene therapy for the treatment of recessive dystrophic epidermolysis bullosa (RDEB). EB-201 (AAV DJ COL7A1) is a pre-clinical candidate targeting a novel, AAV-mediated gene editing and delivery approach to correct gene mutations in skin cells (keratinocytes). We entered into an agreement (the ‘‘EB Agreement’’) with EB Research Partnership (‘‘EBRP’’) and Epidermolysis Bullosa Medical Research Foundation (‘‘EBMRF’’) to collaborate on gene therapy treatments for EB. The EB Agreement became effective August 3, 2016, on the execution of two licensing agreements with The Board of Trustees of Leland Stanford Junior University (‘‘Stanford’’) described below.

EBRP and EBMRF have the contractual right to license from Stanford EB-101 (LZRSE-Col7A1 Engineered Autologous Epidermal Sheets (LEAES)), and authorized us to exercise such rights and enter into a license with Stanford for such technology, and to perform preclinical development and perform clinical trials of a gene therapy treatment for Epidermolysis Bullosa based upon such in-licensed technology.

We also entered into a license with Stanford for the AAV-based gene therapy EB-201 (AAV DJ COL7A1) technology, and we shall perform preclinical development and perform clinical trials of a gene therapy treatment for EB based upon such in-licensed technology. EB-201 (AAV DJ COL7A1) is a pre-clinical candidate targeting a novel, AAV-mediated gene editing and delivery approach (known as homologous recombination) to correct gene mutations in skin cells (keratinocytes) for patients with recessive dystrophic epidermolysis bullosa (RDEB).

Plasma-based Therapeutics using the SDF™ technology platform

Abeona's proprietary Salt Diafiltration Process™ (SDF) focuses on ethanol-free extraction of therapeutic biologics from human plasma. Plasma biologics are biopharmaceutical proteins extracted, purified, and formulated from human blood plasma by the use of biotechnological processing techniques including precipitation, diafiltration, affinity chromatography, and ion-exchange chromatography. These products are rendered virus-safe by means of chemical treatment, nanofiltration, and pasteurization. Plasma biologics primarily address indications arising from genetic deficiencies, which are increasingly being identified by means of newly available rapid and low-cost diagnostic genetic tests. Examples of plasma biologics include Alpha-1 Antitrypsin (also known as alpha-1 proteinase inhibitor, A1PI), Intravenous Immune Globulin (IVIG), Anti-Hemophilic Factor VIII (AHF) and Albumin.

Plasma biologics are currently obtained from human plasma by a fractionation process known as the Cohn Cold Ethanol Fractionation Process (Cohn Process), which was developed prior to World War II to provide a stable solution of human albumin for the rapid treatment of hemorrhagic shock on the battlefield. This process employs various concentrations of ethanol combined with adjustments of pH, ionic strength, and temperature to bring about the necessary separations by precipitation. Ethanol can inactivate many of the plasma proteins.

In contrast to the highly denaturing Cohn Process, Abeona's patented SDFTM method involves a short two-step, ethanol-free salt precipitation process optimized to extract a wide range of therapeutically useful biologic proteins from human blood plasma. SDFTM enables the production of higher yields of these proteins compared with the Cohn Process.

PTB-101 SDF Alpha™ (alpha-1 protease inhibitor) for emphysema or chronic obstructive pulmonary disease (COPD) due to severe congenital deficiency of A1PI (alpha-1-antitrypsin deficiency)

Alpha-1 antitrypsin deficiency is a rare (1 in 1,500 to 3,500) genetic (inherited) autosomal disorder that may cause lung disease from an inability to neutralize the enzyme neutrophil elastase and liver disease from retained misfolded protein. Alpha-1 antitrypsin deficiency occurs worldwide, but its prevalence varies by population. Alpha-1 antitrypsin is also known as alpha-1 proteinase inhibitor (A1PI).

About 10% of infants with alpha-1 antitrypsin deficiency develop liver disease, which often causes yellowing of the skin and whites of the eyes (jaundice). Approximately 15% of adults with alpha-1 antitrypsin deficiency develop liver damage (cirrhosis) due to the formation of scar tissue in the liver. Signs of cirrhosis include a swollen abdomen, swollen feet or legs, and jaundice. Individuals with alpha-1 antitrypsin deficiency are also at risk of developing a type of liver cancer called hepatocellular carcinoma.

Alpha-1 antitrypsin deficiency is inherited with an autosomal codominant pattern, which means that two different versions of the gene may be active (expressed), and both versions contribute to the genetic trait. The most common version (allele) of the SERPINA1 gene, called M, produces normal levels of alpha-1 antitrypsin. Most people in the general population have two copies of the M allele (MM) in each cell. Other versions of the SERPINA1 gene lead to reduced levels of alpha-1 antitrypsin. For example, the S allele produces moderately low levels of this protein, and the Z allele produces very little alpha-1 antitrypsin. Individuals with two copies of the Z allele (ZZ) in each cell are likely to have alpha-1 antitrypsin deficiency. Those with the SZ combination have an increased risk of developing liver and lung diseases such as chronic obstructive pulmonary disease (COPD).

It is estimated that about 200,000 individuals in the United States and Europe have severe alpha-1 antitrypsin deficiency. However, only about 5% of this number have been diagnosed as symptoms caused by this deficiency are very similar to asthma and chronic obstructive pulmonary disease (COPD) from non-genetic causes. Only about 1-2% of COPD patients have severe alpha-1 antitrypsin deficiency. The Global Initiative for Chronic Obstructive Lung Disease (GOLD) defines COPD as group of airflow-limited diseases including emphysema and chronic bronchitis. While severe alpha-1 antitrypsin deficiency can lead to or exacerbate all forms of COPD, it is considered to be the dominant cause of Panacinar Emphysema, a form of emphysema which causes gradual destruction of all lung aveoli.

Abeona is developing PTB-101 SDF Alpha™ (alpha-1 proteinase inhibitor) for chronic augmentation and maintenance therapy in adults with clinically evident panacinar emphysema and other forms of COPD due to severe deficiency of alpha-1 proteinase inhibitor.

Polymer Hydrogel Technology (PHT™)

MuGard® (mucoadhesive oral wound rinse) approved for mucositis, stomatitis, aphthous ulcers, and traumatic ulcers

MuGard® is our marketed product for the management of oral mucositis, a frequent side-effect of cancer therapy for which there is no other established treatment. MuGard, a proprietary nanopolymer formulation, has received marketing clearance from the FDA in the U.S. as well as Europe, China, Australia, New Zealand and Korea. We launched MuGard in the U.S. in 2010 and licensed MuGard for commercialization in the U.S. to AMAG Pharmaceuticals, Inc. (AMAG) in 2013. We licensed MuGard to RHEI Pharmaceuticals, N.V. (RHEI) for China and other Southeast Asian countries in 2010; Hanmi Pharmaceutical Co. Ltd. (Hanmi) for South Korea in 2014; and Norgine B.V. (Norgine) for the European Union, Switzerland, Norway, Iceland, Lichtenstein, Australia and New Zealand in 2014.

LIQUIDITY AND CAPITAL RESOURCES

We have funded our operations primarily through public and private sales of common stock, preferred stock, convertible notes and through licensing agreements. Our principal source of liquidity is cash and cash equivalents. Licensing payments and royalty revenues provided limited funding for operations during the period ended September 30, 2016. As of September 30, 2016, our cash and cash equivalents were $31,185,000.

As of September 30, 2016, our working capital was $25,432,000. Our working capital at September 30, 2016 represented a decrease of $13,659,000 as compared to our working capital of $39,091,000 as of December 31, 2015. The decrease in working capital at September 30, 2016 reflects nine months of net operating losses and changes in current assets and liabilities and the reclassification of the payable to Plasma Technologies, LLC ($4,000,000) from long-term liabilities to current liabilities. The payable to Plasma Technologies, LLC may be paid in cash or stock at our discretion.

Net cash used in operating activities for the nine months ended September 30, 2016 was $9,621,000 as compared to $7,546,000 for the same period in 2015, an increase of $2,075,000. The increase was primarily due to higher research and development spending in the first nine months of 2016 offset by a $1.0 million license payment made in the first quarter of 2015.

On November 1, 2016, we closed an underwritten public offering of 6,000,000 shares of common stock, at a public offering price of $7.00 per share. The gross proceeds to the Company were $42,000,000, before deducting the underwriting discounts and commissions and estimated offering expenses payable by the Company.

If we raise additional funds by selling additional equity securities, the relative equity ownership of our existing investors will be diluted and the new investors could obtain terms more favorable than previous investors.

We have incurred negative cash flows from operations since inception, and have expended, and expect to continue to expend in the future, substantial funds to complete our planned product development efforts. Since inception, our expenses have significantly exceeded revenues, resulting in an accumulated deficit as of September 30, 2016 of $325,471,000. We cannot provide assurance that we will ever be able to generate sufficient product sales or royalty revenue to achieve profitability on a sustained basis, or at all.

Since our inception, we have devoted our resources primarily to fund our research and development programs. We have been unprofitable since inception and to date have received limited revenues from the sale of products. We expect to incur losses for the next several years as we continue to invest in product research and development, preclinical studies, clinical trials and regulatory compliance.

THIRD QUARTER 2016 COMPARED TO THIRD QUARTER 2015

Our licensing revenue for the third quarter of each of 2016 and 2015 was $151,000. We recognize licensing revenue over the period of the performance obligation under our licensing agreements.

We recorded royalty revenue for MuGard of $33,000 for third quarter of 2016 and $134,000 for the same period of 2015, a decrease of $101,000. We licensed MuGard to AMAG on June 6, 2013 and currently receive quarterly royalties from AMAG under our agreement.

Total research and development spending for the third quarter of 2016 was $2,745,000, as compared to $1,581,000 for the same period of 2015, an increase of $1,164,000. The increase in expenses was primarily due to:

- increased development work for the manufactured product for ABO-102 and other gene therapy products ($1,003,000);
- increased clinical costs for our clinical trial for ABO-102 and preparation for other clinical trials ($188,000);
- increased salary and related costs ($128,000) from the hiring of scientific staff;
- other net increases in research spending ($134,000); and
- offset by decreased stock based compensation expense for granted stock ($213,000) and granted stock options ($76,000).

Total general and administrative expenses were $2,391,000 for the third quarter of 2016, as compared to $4,717,000 for the same period of 2015, a decrease of $2,326,000. The decrease in expenses was due primarily to the following:

- decreased stock based compensation expense for granted stock expense ($1,438,000) and granted stock options ($307,000);
- decreased investor relations fees ($799,000);
- decreased legal fees ($108,000);
- offset by increased salary and related costs ($152,000); and
- offset by increased net other general and administrative expenses ($174,000).

Depreciation and amortization was $222,000 for the third quarter of 2016 as compared to $151,000 for the same period in 2015, an increase of $71,000. We are amortizing the licenses for SDF Alpha, ABO-101 and ABO-201, and EB-102 and EB-102 over the life of the patents. The increase is due to amortization of licensed technology of $38,000 and depreciation of $33,000.

Total operating expenses for the third quarter of 2016 were $5,358,000 as compared to total operating expenses of $6,449,000 for the same period of 2015, a decrease of $1,091,000 for the reasons listed above.

Interest and miscellaneous income was $2,551,000 for the third quarter of 2016 as compared to $92,000 for the same period of 2015, an increase of $2,459,000. Miscellaneous income is higher in third quarter than for the same period in 2015 due to the change in the fair value of our contingent consideration liability ($2,000,000) related to the acquisition of Abeona Therapeutics LLC, the settlement of an agreement ($500,000) and less other income ($41,000).

Interest and other expense for the third quarter of 2016 and 2015 was $1,000 for each period.

Net loss for the third quarter of 2016 was $2,624,000, or a $0.08 basic and diluted loss per common share as compared to a net loss of $6,073,000, or a $0.19 basic and diluted loss per common share, for the same period in 2015, a decreased loss of $3,449,000.

NINE MONTHS ENDED SEPTEMBER 30, 2016 COMPARED TO NINE MONTHS ENDED SEPTEMBER 30, 2015

Our licensing revenue for each of the first nine months of 2016 and 2015 was $452,000. We recognize licensing revenue over the period of the performance obligation under our licensing agreements.

We recorded royalty revenue for MuGard of $181,000 for the first nine months of 2016 and $373,000 for the same period of 2015, a decrease of $192,000. We licensed MuGard to AMAG on June 6, 2013 and currently receive quarterly royalties from AMAG under our agreement.

Total research and development spending for the first nine months of 2016 was $7,618,000, as compared to $2,644,000 for the same period of 2015, an increase of $4,974,000. The increase in expenses was primarily due to:

- increased development work for the manufactured product for ABO-102 and other gene therapy products ($2,469,000);
- increased clinical costs for our clinical trial for ABO-102 and preparation for other clinical trials ($440,000);

- increased salary and related costs ($1,091,000) from the hiring of scientific staff and annual bonus payments;
- increased stock based compensation expense for granted stock options ($477,000);
- increased travel and entertainment ($216,000); and
- other net increases in research spending ($281,000).

Total general and administrative expenses were $10,487,000 for the first nine months of 2016, as compared to $10,073,000 for the same period of 2015, an increase of $414,000. The increase in expenses was due primarily to the following:

- increased stock based compensation expense for granted stock options ($134,000) and granted stock ($1,101,000);
- increased salary and related costs and annual bonus payments ($467,000);
- increased net other general and administrative expense ($296,000).
- offset by decreased investor relations fees ($1,246,000); and
- offset by decreased legal fees ($338,000).

Depreciation and amortization was $577,000 for the first nine months of 2016 as compared to $401,000 for the same period in 2015, an increase of $176,000. We are amortizing the licenses for SDF Alpha, ABO-101 and ABO-201, and EB-101 and EB-102 over the life of the patents. The increase is due to amortization of licensed technology of $82,000 and depreciation of $94,000.

Total operating expenses for the first nine months of 2016 were $18,682,000 as compared to total operating expenses of $13,118,000 for the same period of 2015, an increase of $5,564,000 for the reasons listed above.

Interest and miscellaneous income was $3,182,000 for the first nine months of 2016 as compared to $111,000 for the same period of 2015, an increase of $3,071,000. Miscellaneous income is higher in 2016 than for the same period in 2015 due to the change in the fair value of our contingent consideration liability ($2,591,000) related to the acquisition of Abeona Therapeutics LLC, settlement of an agreement ($500,000) less other income ($20,000).

Interest and other expense for the first nine months of 2016 and 2015 was $4,000 for each period.

Net loss for the nine months of 2016 was $14,871,000, or a $0.45 basic and diluted loss per common share as compared to a net loss of $12,186,000, or a $0.47 basic and diluted loss per common share, for the same period in 2015, an increased loss of $2,685,000.

ITEM 3. QUANTITATIVE AND QUALITATIVE DISCLOSURES ABOUT MARKET RISK

Not applicable.

ITEM 4. CONTROLS AND PROCEDURES

Under the supervision and with the participation of our management and consultants, including the Executive Chairman (our principal executive officer) and Vice President Finance (our principal accounting officer), we have evaluated the effectiveness of the design and operation of our disclosure controls and procedures, as such term is defined in Exchange Act Rules 13a-15(e) and 15d-15(e), as of the end of the period covered by this report.

Our management is responsible for establishing and maintaining adequate internal control over financial reporting, as such term is defined in Rules 13a-15(f) and 15d-15(f) of the Exchange Act. Our internal control system was designed to provide reasonable assurance regarding the reliability of financial reporting and the preparation of financial statements for external purposes, in accordance with generally accepted accounting principles. Because of inherent limitations, a system of internal control over financial reporting may not prevent or detect misstatements. Also, projections of any evaluation of effectiveness to future periods are subject to the risk that controls may become inadequate due to change in conditions, or that the degree of compliance with the policies or procedures may deteriorate.

Our management, including our principal executive officer and principal accounting officer, conducted an evaluation of the effectiveness of our internal control over financial reporting using the criteria set forth by the Committee of Sponsoring Organizations of the Treadway Commission (COSO) in Internal Control—Integrated Framework.

Based on such evaluation, our management concluded in our Annual Report on Form 10-K for the year ended December 31, 2015 that there is no material weakness in our internal control as defined under the standards established by the Public Company Accounting Oversight Board (United States). A material weakness is a deficiency, or a combination of control deficiencies, in internal control over financial reporting such that there is a reasonable possibility that a material misstatement of the Company's annual or interim financial statements will not be prevented or detected on a timely basis.

Changes In Internal Control Over Financial Reporting

There were no changes in our internal control over financial reporting that occurred during the quarter ended September 30, 2016 that have materially affected, or are reasonable likely to materially affect, our internal control over financial reporting.

PART II -- OTHER INFORMATION

ITEM 1. LEGAL PROCEEDINGS.

None.

ITEM 2. UNREGISTERED SALES OF EQUITY SECURITIES AND USE OF PROCEEDS.

On August 3, 2016, we issued 750,000 shares of restricted common stock, at a price of $3.27 to two foundations per an agreement.

ITEM 3. DEFAULTS UPON SENIOR SECURITIES.

None.

ITEM 6. EXHIBITS.

Exhibits:

31.1	Principal Executive Officer Certification pursuant to 18 U.S.C. Section 1350, as adopted pursuant to Section 302 of the Sarbanes-Oxley Act of 2002
31.2	Principal Financial Officer Certification pursuant to 18 U.S.C. Section 1350, as adopted pursuant to Section 302 of the Sarbanes-Oxley Act of 2002
32.1*	Principal Executive Officer Certification pursuant to 18 U.S.C. Section 1350, as adopted pursuant to Section 906 of the Sarbanes-Oxley Act of 2002
32.2*	Principal Financial Officer Certification pursuant to 18 U.S.C. Section 1350, as adopted pursuant to Section 906 of the Sarbanes-Oxley Act of 2002
101	The following materials from Abeona's Quarterly Report on Form 10-Q for the quarter ended September 30, 2016, formatted in XBRL (Extensible Business Reporting Language): (i) Condensed Consolidated Balance Sheets at September 30, 2016 and December 31, 2015, (ii) Condensed Consolidated Statements of Operations for the three and nine months ended September 30, 2016 and September 30, 2015, (iii) Condensed Consolidated Statements of Stockholders' Equity for the three and nine months ended September 30, 2016, (iv) Condensed Consolidated Statements of Cash Flows for the nine months ended September 30, 2016 and September 30, 2015, and (v) Notes to Condensed Consolidated Financial Statements, tagged as blocks of text.

* This exhibit shall not be deemed "filed" for purposes of Section 18 of the Securities Exchange Act of 1934 or otherwise subject to the liabilities of that Section, nor shall it be deemed incorporated by reference in any filings under the Securities Act of 1933 or the Securities and Exchange Act of 1934, whether made before or after the date hereof and irrespective of any general incorporation language in any filing.

SIGNATURES

Pursuant to the requirements of the Securities Exchange Act of 1934, the registrant has duly caused this report to be signed on its behalf by the undersigned, thereunto duly authorized.

ABEONA THERAPEUTICS INC.

Date: November 14, 2016

By: /s/ Steven H. Rouhandeh
Steven H. Rouhandeh
Executive Chairman
(Principal Executive Officer)

Date: November 14, 2016

By: /s/ Stephen B. Thompson
Stephen B Thompson
Vice President Finance
(Principal Accounting Officer)

Abeona Therapeutics Inc. and Subsidiaries

Condensed Consolidated Balance Sheets

	September 30, 2016 (unaudited)	December 31, 2015
ASSETS		
Current assets		
Cash and cash equivalents	$ 31,185,000	$ 40,138,000
Receivables	606,000	115,000
Prepaid expenses and other current assets	156,000	315,000
Total current assets	31,947,000	40,568,000
Property and equipment, net	748,000	350,000
Licensed technology, net	8,587,000	6,609,000
Goodwill	32,466,000	32,466,000
Other assets	66,000	62,000
Total assets	$ 73,814,000	$ 80,055,000
LIABILITIES AND STOCKHOLDERS' EQUITY		
Current liabilities		
Accounts payable	$ 1,913,000	$ 875,000
Current portion of deferred revenue	602,000	602,000
Payable due Plasma Technologies, LLC	4,000,000	-
Total current liabilities	6,515,000	1,477,000
Contingent consideration liability	-	2,591,000
Payable due Plasma Technologies, LLC	-	4,000,000
Long-term deferred revenue	3,814,000	4,266,000
Total liabilities	10,329,000	12,334,000
Commitments and contingencies		
Stockholders' equity		
Common stock - $.01 par value; authorized 200,000,000 shares; issued, 33,703,732 at September 30, 2016 and 32,743,013 at December 31, 2015	338,000	328,000
Additional paid-in capital	388,618,000	377,993,000
Accumulated deficit	(325,471,000)	(310,600,000)
Total stockholders' equity	63,485,000	67,721,000
Total liabilities and stockholders' equity	$ 73,814,000	$ 80,055,000

The accompanying notes are an integral part of these condensed consolidated statements.

Abeona Therapeutics Inc. and Subsidiaries

Condensed Consolidated Statements of Operations
(unaudited)

	Three months ended September 30,		Nine months ended September 30,	
	2016	2015	2016	2015
Revenues				
License revenues	$ 151,000	$ 151,000	$ 452,000	$ 452,000
Royalties	33,000	134,000	181,000	373,000
Total revenues	184,000	285,000	633,000	825,000
Expenses				
Research and development	2,745,000	1,581,000	7,618,000	2,644,000
General and administrative	2,391,000	4,717,000	10,487,000	10,073,000
Depreciation and amortization	222,000	151,000	577,000	401,000
Total expenses	5,358,000	6,449,000	18,682,000	13,118,000
Loss from operations	(5,174,000)	(6,164,000)	(18,049,000)	(12,293,000)
Interest and miscellaneous income	2,551,000	92,000	3,182,000	111,000
Interest and other expense	(1,000)	(1,000)	(4,000)	(4,000)
	2,550,000	91,000	3,178,000	107,000
Net loss	$ (2,624,000)	$ (6,073,000)	$ (14,871,000)	$ (12,186,000)
Basic and diluted loss per common share	$ (0.08)	$ (0.19)	$ (0.45)	$ (0.47)
Weighted average number of common shares outstanding	33,274,829	31,787,777	32,935,232	25,865,739

The accompanying notes are an integral part of these condensed consolidated statements.

Abeona Therapeutics Inc. and Subsidiaries

Condensed Consolidated Statements of Stockholders' Equity
(unaudited)

	Common Stock		Additional paid-in	Accumulated	Total stockholders'
	Shares	Amount	capital	deficit	equity
Balance December 31, 2015	32,743,013	$ 328,000	$ 377,993,000	$ (310,600,000)	$ 67,721,000
Restricted common stock issued to employees and directors	-	-	1,892,000	-	1,892,000
Stock option compensation expense	-	-	1,592,000	-	1,592,000
Net loss	-	-	-	(5,544,000)	(5,544,000)
Balance March 31, 2016	32,743,013	$ 328,000	$ 381,477,000	$ (316,144,000)	$ 65,661,000
Restricted common stock issued to employees and directors	-	-	987,000	-	987,000
Restricted common stock issued for $2.85	52,690	-	150,000	-	150,000
Stock option compensation expense	-	-	1,385,000	-	1,385,000
Net loss	-	-	-	(6,703,000)	(6,703,000)
Balance June 30, 2016	32,795,703	$ 328,000	$ 383,999,000	$ (322,847,000)	$ 61,480,000
Restricted common stock issued to employees and directors	-	-	241,000	-	241,000
Common stock issued for $3.27 share for licenses	750,000	8,000	2,444,000	-	2,452,000
Common stock issued for an average of $6.44 share net of costs	158,029	2,000	1,017,000	-	1,019,000
Stock option compensation expense	-	-	917,000	-	917,000
Net loss	-	-	-	(2,624,000)	(2,624,000)
Balance September 30, 2016	33,703,732	$ 338,000	$ 388,618,000	$ (325,471,000)	$ 63,485,000

The accompanying notes are an integral part of these condensed consolidated statements.

Abeona Therapeutics Inc. and Subsidiaries

Condensed Consolidated Statements of Cash Flows
(unaudited)

	Nine Months ended September 30,	
	2016	2015
Cash flows from operating activities:		
Net loss	$ (14,871,000)	$ (12,186,000)
Adjustments to reconcile net loss to cash used in operating activities:		
Depreciation and amortization	577,000	401,000
Stock option compensation expense	3,894,000	2,851,000
Stock issued to directors, employees and consultants	3,120,000	2,960,000
Stock issued for services	-	333,000
Gain on contingent consideration liability	(2,591,000)	-
Change in operating assets and liabilities:		
Receivables	(491,000)	(235,000)
Prepaid expenses and other current assets	159,000	(109,000)
Other assets	(4,000)	(29,000)
Accounts payable	1,038,000	(1,081,000)
Deferred revenue	(452,000)	(451,000)
Net cash used in operating activities	(9,621,000)	(7,546,000)
Cash flows from investing activities:		
Capital expenditures	(501,000)	(41,000)
Cash from Abeona Ohio	-	3,697,000
Net cash (used in) provided by investing activities	(501,000)	3,656,000
Cash flows from financing activities:		
Proceeds from $3.00 common stock issuances net of costs	-	7,001,000
Proceeds from $8.00 common stock issuances net of costs	-	9,005,000
Proceeds from $5.50 common stock issuances net of costs	-	15,411,000
Proceeds from exercise of $5.00 warrants	-	4,635,000
Proceeds from $2.85 restricted common stock issuance	150,000	-
Proceeds from an average of $6.44 per share common stock issuances net of costs	1,019,000	-
Payment of short-term debt	-	(400,000)
Net cash provided by financing activities	1,169,000	35,652,000
Net increase (decrease) in cash and cash equivalents	(8,953,000)	31,762,000
Cash and cash equivalents at beginning of period	40,138,000	11,520,000
Cash and cash equivalents at end of period	$ 31,185,000	$ 43,282,000
Supplemental disclosure of noncash transactions:		
Shares issued to holders of Abeona Ohio for acquisition	*$ -*	*$ 31,758,000*
Contingent milestones to Abeona Ohio members	*-*	*6,489,000*
Licensed technology from Abeona Ohio	*-*	*2,156,000*
Shares issued to EB Research Partnership and Epidermolysis Bullosa Medical Research Foundation for licenses	2,452,000	-

The accompanying notes are an integral part of these condensed consolidated statements.

Abeona Therapeutics Inc. (together with our subsidiaries, "we", "our", "Abeona" or the "Company") is a Delaware corporation. We are a clinical stage biopharmaceutical company developing gene and plasma-based therapies for life-threatening rare genetic diseases. Our lead programs are ABO-102 (AAV-SGSH) and ABO-101 (AAV-NAGLU), adeno-associated virus (AAV) based gene therapies for Sanfilippo syndrome (MPS IIIA and IIIB, respectively). We are also developing EB-101 (gene-corrected skin grafts) for recessive dystrophic epidermolysis bullosa (RDEB), EB-201 for epidermolysis bullosa (EB), ABO-201 (AAV-CLN3) gene therapy for juvenile Batten disease (JNCL), ABO-202 (AAV-CLN1) gene therapy for treatment of infantile Batten disease (INCL), and ABO-301 (AAV-FANCC) for Fanconi anemia (FA) disorder and ABO-302 using a novel CRISPR/Cas9-based gene editing approach to gene therapy for rare blood diseases. In addition, we have a plasma-based protein therapy pipeline, including SDF Alpha™ (alpha-1 protease inhibitor) for inherited COPD, using our proprietary SDFTM (Salt Diafiltration) ethanol-free process.

(1) Interim Financial Statements

The condensed consolidated balance sheet as of September 30, 2016, the condensed consolidated statements of operations for the three and nine months ended September 30, 2016 and 2015, the condensed consolidated statements of stockholders' equity for the three and nine months ended September 30, 2016, and the condensed consolidated statements of cash flows for the nine months ended September 30, 2016 and 2015, were prepared by management without audit. In the opinion of management, all adjustments, consisting only of normal recurring adjustments, except as otherwise disclosed, necessary for the fair presentation of the financial position, results of operations, and changes in financial position for such periods, have been made.

Certain information and footnote disclosures normally included in financial statements prepared in accordance with accounting principles generally accepted in the United States of America (U.S. GAAP) have been condensed or omitted. It is suggested that these interim financial statements be read in conjunction with the consolidated financial statements and notes thereto included in our Annual Report on Form 10-K for the year ended December 31, 2015. The results of operations for the period ended September 30, 2016 are not necessarily indicative of the operating results which may be expected for a full year. The condensed consolidated balance sheet as of December 31, 2015 contains financial information taken from the audited Abeona financial statements as of that date.

(2) Intangible Assets

Intangible assets consist of the following (in thousands):

	September 30, 2016		December 31, 2015	
	Gross carrying value	Accumulated amortization	Gross carrying value	Accumulated Amortization
Amortizable intangible assets Licensed technology	$ 9,608	$ 1,021	$ 7,156	$ 547

Amortization expense related to intangible assets totaled $183,000 and $474,000 for the three and nine months ended September 30, 2016, respectively, and totaled $145,000 and $392,000 for the three and nine months ended September 30, 2015, respectively. The aggregate estimated amortization expense for intangible assets remaining as of September 30, 2016 is as follows (in thousands):

2016	$ 203
2017	812
2018	812
2019	812
2020	812
over 5 years	5,136
Total	$ 8,587

(3) Fair Value Measurements

We calculate the fair value of our assets and liabilities which qualify as financial instruments and include additional information in the notes to the consolidated financial statements when the fair value is different than the carrying value of these financial instruments. The estimated fair value of receivables, accounts payable, short-term notes payable and payable to Plasma Technologies, LLC approximate their carrying amounts due to the relatively short maturity of these instruments.

U.S. GAAP defines fair value as the exchange price that would be received for an asset or paid to transfer a liability (an exit price) in the principal or most advantageous market for the asset or liability in an orderly transaction between market participants at the measurement date. This guidance establishes a three-level fair value hierarchy that prioritizes the inputs used to measure fair value. The hierarchy requires entities to maximize the use of observable inputs and minimize the use of unobservable inputs. The three levels of inputs used to measure fair value are as follows:

- Level 1 – Quoted prices in active markets for identical assets or liabilities.
- Level 2 – Observable inputs other than quoted prices included in Level 1, such as quoted prices for similar assets and liabilities in active markets; quoted prices for identical or similar assets and liabilities in markets that are not active; or other inputs that are observable or can be corroborated by observable market data.

· Level 3 – Unobservable inputs that are supported by little or no market activity and that are significant to the fair value of the assets and liabilities. This includes certain pricing models, discounted cash flow methodologies and similar valuation techniques that use significant unobservable inputs.

The guidance requires an entity to maximize the use of observable inputs and minimize the use of unobservable inputs when measuring fair value.

We have segregated all financial assets and liabilities that are measured at fair value on a recurring basis (at least annually) into the most appropriate level within the fair value hierarchy based on the inputs used to determine the fair value at the measurement date in the table below.

Financial assets and liabilities measured at fair value on a recurring and non-recurring basis as of September 30, 2016 and December 31, 2015 are summarized below:

(in thousands)

Description	As of September 30, 2016	Level 1	Level 2	Level 3	Total Gains (Losses)
Non-recurring					
Assets:					
Licensed technology (net)	$ 8,587	$ -	$ -	$ 8,587	$ -
Goodwill	32,466	-	-	32,466	-
Recurring					
Liabilities:					
Contingent consideration	$ -	$ -	$ -	$ -	$ 2,591

Description	As of December 31, 2015	Level 1	Level 2	Level 3	Total Gains (Losses)
Non-recurring					
Assets:					
Licensed technology (net)	$ 6,609	$ -	$ -	$ 6,609	$ -
Goodwill	32,466	-	-	32,466	-
Recurring					
Liabilities:					
Contingent consideration	$ 2,591	$ -	$ -	$ 2,591	$ 3,898

(4) Stock Based Compensation and Restricted Stock Compensation

For the three and nine months ended September 30, 2016, we recognized stock-based compensation expense of $917,000 and $3,894,000, respectively. For the three and nine months ended September 30, 2015 we recognized stock-based compensation expense of $1,452,000 and $2,581,000, respectively.

The following table summarizes stock-based compensation for the three and nine months ended September 30, 2016 and 2015:

	Three months ended September 30,		Nine months ended September 30,	
	2016	2015	2016	2015
Research and development	$ 274,000	$ 142,000	$ 937,000	$ 246,000
General and administrative	643,000	1,310,000	2,957,000	2,335,000
Stock-based compensation expense included in operating expense	$ 917,000	$ 1,452,000	$ 3,894,000	$ 2,581,000

For the three and nine months ended September 30, 2016 we granted no options and 1,440,000 stock options, respectively, and for the three and nine months ended September 30, 2015 we granted no options and 1,815,000 stock options, respectively.

For the three and nine months ended September 30, 2016, we recognized restricted stock compensation expense of $241,000 and $3,120,000, respectively. For the three and nine months ended September 30, 2015 we recognized stock-based compensation expense of $1,892,000 and $2,960,000, respectively.

The following table summarizes restricted stock compensation expense for the three and nine months ended September 30, 2016 and 2015:

	Three months ended September 30,		Nine months ended September 30,	
	2016	2015	2016	2015
Research and development	$ -	$ 138,000	$ 200,000	$ 228,000
General and administrative	241,000	1,754,000	2,920,000	2,732,000
Restricted stock compensation expense included in operating expense	$ 241,000	$ 1,892,000	$ 3,120,000	$ 2,960,000

For the three and nine months ended September 30, 2016 no shares were granted to directors and employees. For the three and nine months ended September 30, 2015 we granted no shares and 1,360,000 shares, respectively of our common stock to directors and employees.

(5) Litigation

We are not currently subject to any material legal proceedings.

(6) Subsequent Events

On November 1, 2016, we closed an underwritten public offering of 6,000,000 shares of common stock, at a public offering price of $7.00 per share. The gross proceeds to the Company were $42,000,000, before deducting the underwriting discounts and commissions and estimated offering expenses payable by the Company.

EXHIBIT 31.1

PRINCIPAL EXECUTIVE OFFICER CERTIFICATION PURSUANT TO 18 U.S.C.
SECTION 1350, AS ADOPTED PURSUANT TO SECTION 302
OF THE SARBANES-OXLEY ACT OF 2002

I, Steven H. Rouhandeh, certify that:

1. I have reviewed this report on Form 10-Q of Abeona Therapeutics Inc.

2. Based on my knowledge, this report does not contain any untrue statement of a material fact or omit to state a material fact necessary to make the statements made, in light of the circumstances under which such statements were made, not misleading with respect to the period covered by this report;

3. Based on my knowledge, the financial statements, and other financial information included in this report, fairly present in all material respects the financial condition, results of operations and cash flows of the registrant as of, and for, the periods presented in this report;

4. The registrant's other certifying officer(s) and I are responsible for establishing and maintaining disclosure controls and procedures (as defined in Exchange Act Rules 13a-15(e) and 15d-15(e) and internal control over financial reporting (as defined in Exchange Act Rules 13a-15(f) and 15d-15(f)) for the registrant and have:

   a) Designed such disclosure controls and procedures, or caused such disclosure controls and procedures to be designed under our supervision, to ensure that material information relating to the registrant, including its consolidated subsidiaries, is made known to us by others within those entities, particularly during the period in which this report is being prepared;

   b) Designed such internal control over financial reporting, or caused such internal control over financial reporting to be designed under our supervision, to provide reasonable assurance regarding the reliability of financial reporting and the preparation of financial statements for external purposes in accordance with generally accepted accounting principles;

   c) Evaluated the effectiveness of the registrant's disclosure controls and procedures and presented in this report our conclusions about the effectiveness of the disclosure controls and procedures, as of the end of the period covered by this report based on such evaluation; and

   d) Disclosed in this report any change in the registrant's internal control over financial reporting that occurred during the registrant's most recent fiscal quarter (the registrant's first fiscal quarter in the case of an annual report) that has materially affected, or is reasonably likely to materially affect, the registrant's internal control over financial reporting; and

5. The registrant's other certifying officer(s) and I have disclosed, based on our most recent evaluation of internal control over financial reporting, to the registrant's auditors and the audit committee of the registrant's board of directors (or persons performing the equivalent functions):

   a) All significant deficiencies and material weaknesses in the design or operation of internal control over financial reporting which are reasonably likely to adversely affect the registrant's ability to record, process, summarize and report financial information; and

   b) Any fraud, whether or not material, that involves management or other employees who have a significant role in the registrant's internal control over financial reporting.

Dated: November 14, 2016

/s/ Steven H. Rouhandeh
Steven H. Rouhandeh
Executive Chairman
Principal Executive Officer

EXHIBIT 31.2

PRINCIPAL FINANCIAL OFFICER CERTIFICATION PURSUANT TO 18 U.S.C.
SECTION 1350, AS ADOPTED PURSUANT TO SECTION 302
OF THE SARBANES-OXLEY ACT OF 2002

I, Stephen B. Thompson, certify that:

1. I have reviewed this report on Form 10-Q of Abeona Therapeutics Inc.

2. Based on my knowledge, this report does not contain any untrue statement of a material fact or omit to state a material fact necessary to make the statements made, in light of the circumstances under which such statements were made, not misleading with respect to the period covered by this report;

3. Based on my knowledge, the financial statements, and other financial information included in this report, fairly present in all material respects the financial condition, results of operations and cash flows of the registrant as of, and for, the periods presented in this report;

4. The registrant's other certifying officer(s) and I are responsible for establishing and maintaining disclosure controls and procedures (as defined in Exchange Act Rules 13a-15(e) and 15d-15(e) and internal control over financial reporting (as defined in Exchange Act Rules 13a-15(f) and 15d-15(f)) for the registrant and have:

   a) Designed such disclosure controls and procedures, or caused such disclosure controls and procedures to be designed under our supervision, to ensure that material information relating to the registrant, including its consolidated subsidiaries, is made known to us by others within those entities, particularly during the period in which this report is being prepared;

   b) Designed such internal control over financial reporting, or caused such internal control over financial reporting to be designed under our supervision, to provide reasonable assurance regarding the reliability of financial reporting and the preparation of financial statements for external purposes in accordance with generally accepted accounting principles;

   c) Evaluated the effectiveness of the registrant's disclosure controls and procedures and presented in this report our conclusions about the effectiveness of the disclosure controls and procedures, as of the end of the period covered by this report based on such evaluation; and

   d) Disclosed in this report any change in the registrant's internal control over financial reporting that occurred during the registrant's most recent fiscal quarter (the registrant's first fiscal quarter in the case of an annual report) that has materially affected, or is reasonably likely to materially affect, the registrant's internal control over financial reporting; and

5. The registrant's other certifying officer(s) and I have disclosed, based on our most recent evaluation of internal control over financial reporting, to the registrant's auditors and the audit committee of the registrant's board of directors (or persons performing the equivalent functions):

   a) All significant deficiencies and material weaknesses in the design or operation of internal control over financial reporting which are reasonably likely to adversely affect the registrant's ability to record, process, summarize and report financial information; and

   b) Any fraud, whether or not material, that involves management or other employees who have a significant role in the registrant's internal control over financial reporting.

Dated: November 14, 2016

/s/ Stephen B. Thompson
Stephen B. Thompson
Vice President Finance
Principal Financial and Accounting Officer

EXHIBIT 32.1

CERTIFICATION PURSUANT TO 18 U.S.C.
SECTION 1350 AS ADOPTED PURSUANT TO SECTION 906
OF THE SARBANES-OXLEY ACT OF 2002

This certification set forth below is hereby made solely for the purposes of satisfying the requirements of Section 906 of the Sarbanes-Oxley Act of 2002 and may not be relied upon or used for any other purposes.

A signed original of this written statement required by Section 906 has been provided to Abeona Therapeutics Inc. and will be retained by Abeona Therapeutics Inc. and furnished to the SEC or its staff upon its request.

Pursuant to Section 906 of the Public Company Accounting Reform and Investor Act of 2002 (18 U.S.C. 1350, as adopted, the "Sarbanes-Oxley Act"), Steven H. Rouhandeh, Executive Chairman of Abeona Therapeutics Inc. (the "Company") hereby certifies that to his knowledge the report on Form 10-Q for the period ended September 30, 2016 of the Company filed with the Securities and Exchange Commission on the date hereof (the "Report") fully complies with the requirements of section 13(a) or 15(d) of the Securities Exchange Act of 1934 and the information contained in the Report fairly presents, in all material respects, the financial condition and results of operations of the Company for the period specified.

Signed at the City of Dallas, in the State of Texas, this 14th day of November, 2016.

/s/ Steven H. Rouhandeh
Steven H. Rouhandeh
Executive Chairman
Principal Executive Officer

EXHIBIT 32.2

CERTIFICATION PURSUANT TO 18 U.S.C.
SECTION 1350 AS ADOPTED PURSUANT TO SECTION 906
OF THE SARBANES-OXLEY ACT OF 2002

This certification set forth below is hereby made solely for the purposes of satisfying the requirements of Section 906 of the Sarbanes-Oxley Act of 2002 and may not be relied upon or used for any other purposes.

A signed original of this written statement required by Section 906 has been provided to Abeona Therapeutics Inc. and will be retained by Abeona Therapeutics Inc. and furnished to the SEC or its staff upon its request.

Pursuant to Section 906 of the Public Company Accounting Reform and Investor Act of 2002 (18 U.S.C. 1350, as adopted, the "Sarbanes-Oxley Act"), Stephen B. Thompson, Vice President Finance of the Company hereby certifies that to his knowledge the report on Form 10-Q for the period ended September 30, 2016 of the Company filed with the Securities and Exchange Commission on the date hereof (the "Report") fully complies with the requirements of section 13(a) or 15(d) of the Securities Exchange Act of 1934 and the information contained in the Report fairly presents, in all material respects, the financial condition and results of operations of the Company for the period specified.

Signed at the City of Dallas, in the State of Texas, this 14th day of November, 2016.

/s/ Stephen B. Thompson
Stephen B. Thompson
Vice President Finance
Principal Financial and Accounting Officer