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 March 31, 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 **0-9314**

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 May 16, 2016 was 32,795,703 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 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 expectation that we will continue to incur losses, our belief that we will expend substantial funds to conduct research and development programs, preclinical studies and clinical trials of potential products, 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 ability to achieve profitability on a sustained basis or at all, our expected cash burn rate, that 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, and that we intend to commercialize ProctiGard in a manner similar to the commercialization of MuGard, which may include confirmatory clinical trials, with the objective of commercialization in collaboration with marketing partners globally. 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 13.

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 focused on developing and delivering gene therapy and plasma-based products for severe and life-threatening rare diseases. Abeona's lead programs are ABO-101 (AAV NAGLU) and ABO-102 (AAV SGSH), adeno-associated virus (AAV)-based gene therapies for Sanfilippo syndrome (MPS IIIB and IIIA, respectively). We are also developing ABO-201 (AAV CLN3) gene therapy for Juvenile Neuronal Ceroid Lipofuscinoses (JNCL), also known as juvenile Batten disease, and ABO-301 (AAV FANCC) for Fanconi anemia (FA) disorder using a novel CRISPR/Cas9-based gene editing approach to gene therapy program for rare blood diseases. In addition, we are also developing rare plasma protein therapies including PTB-101 SDF Alpha™ (alpha-1 protease inhibitor) for inherited COPD using our proprietary SDF™ (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 January 11, 2016 we announced initial regulatory approval for Phase 1/2 gene therapy clinical studies for patients with Sanfilippo syndrome types A and B. The Interministerial Council of Genetically Modified Organisms has approved the Genetically Modified Organism (GMO) Voluntary Release regulatory filings for both Phase 1/2 Gene Therapy Clinical Studies to treat patients with ABO-101 (AAV NAGLU) and ABO-102 (AAV SGSH) for patients with Sanfilippo syndrome type A (MPS IIIA) or type B (MPS IIIB). Additionally, the Comite Etico De Investigacion Clinica de Euskadi (CEIC-E) has approved the ethical committee regulatory filings for both ABO-101 and ABO-102. Abeona plans to file Clinical Trial Authorization’s (CTAs) for both programs shortly for the upcoming clinical studies to be conducted at Cruces University Hospital (Bilbao, Spain).

On February 29, 2016 we announced that the FDA cleared the Investigational New Drug Application for ABO-102 (AAV-SGSH), a single treatment strategy for Mucopolysaccharidosis Type IIIA (MPS IIIA). The ABO-102 IND application is now active and enables Nationwide Children’s Hospital (Columbus, OH) to initiate a Phase 1/2 clinical study designed to assess the safety, tolerability and potential efficacy of ABO-102 in children with MPS III A.

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—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), which we believe make them attractive for addressing lysosomal storage diseases which have severe CNS manifestations of the disease.

Lysosomal storage diseases (LSD) 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, we believe that 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. Also known as Sanfilippo syndromes type A and type B, MPS III 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 expected to be given only once.

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

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 called heparan sulfate, which is essential in breaking down 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, which will involve a one-time delivery of a normal copy of the defective gene to cells of the CNS with the goal 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 ABO-101 or ABO-102 are well tolerated with minimal side effects.

ABO-201 for Juvenile Neuronal Ceroid Lipofuscinoses (JNCL) (or Juvenile Batten Disease (JBD))

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 CNS with the goal 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 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.

JNCL 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 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.

ABO-301 for Fanconi Anemia (FA)

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 goal 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 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 for more precise gene modification.

Plasma-based Therapeutics using the SDF™ technology platform

Abeona's proprietary patented 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 SDF 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. SDF 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 such individuals have been diagnosed as symptoms caused by this deficiency are very similar to those of 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 aveolii.

PTB-101 SDF Alpha™ (alpha1-proteinase inhibitor) for Alpha-1 Antitrypsin Deficiency (Alpha-1)

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, received marketing clearance from the FDA in the US 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.

ProctiGard™ (mucoadhesive oral wound rinse) approved for rectal mucositis and radiation proctitis

ProctiGard™ received 510(K) marketing clearance from the FDA on July 22, 2014 for the treatment of symptomatic management of rectal mucositis. ProctiGard is our product for the treatment of radiation proctitis, a frequent side effect of radiation treatment to the pelvic region. Radiation proctitis, or RP, is the inflammation and damage to the lower portion of the colon after exposure to x-rays or ionizing radiation as part of radiation therapy. RP is most common after treatments for cancer, such as cervical, colon and prostate cancer. RP can be acute, occurring within weeks of initiation of therapy, or can occur months or years after treatment. We intend to commercialize ProctiGard in a manner similar to the commercialization of MuGard, which may include confirmatory clinical trials, with the objective of commercialization in collaboration with marketing partners globally.

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 March 31, 2016. As of March 31, 2016, our cash and cash equivalents were $37,395,000.

As of March 31, 2016, our working capital was $30,196,000. Our working capital at March 31, 2016 represented a decrease of $8,895,000 as compared to our working capital of $39,091,000 as of December 31, 2015. The decrease in working capital at March 31, 2016 reflects three months of net operating costs and changes in current assets and liabilities and the classification of contingent consideration liability ($2,000,000) and payable to Licensor ($4,000,000) from long-term liabilities to current liabilities.

Net cash used in operating activities for the three months ended March 31, 2016 was $2,156,000 as compared to $3,163,000 for the same period in 2015, a decrease of $647,000. The decrease was primarily due a $1.0 million license payment made in the first quarter of 2015 offset by higher research and development spending in the first quarter of 2016.

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 March 31, 2016 of $316,144,000. 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.

FIRST QUARTER 2016 COMPARED TO FIRST QUARTER 2015

Our licensing revenue for the first quarter 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 $84,000 for first quarter of 2016 and $107,000 for the same period of 2015, a decrease of $23,000. 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 quarter of 2016 was $1,855,000, as compared to $453,000 for the same period of 2015, an increase of $1,402,000. The increase in expenses was primarily due to:

- increased development work on the preparation of our gene therapy products for clinical trials ($387,000);
- increased salary and related costs ($358,000) from the hiring of scientific staff;
- increased stock based compensation expense for granted stock options ($323,000) and granted stock ($138,000);
- increased rent for our new laboratory ($71,000) and
- other net increases in research spending ($125,000).

Total general and administrative expenses were $4,366,000 for the first quarter of 2016, as compared to $1,689,000 for the same period of 2015, an increase of $2,677,000. The increase in expenses was due primarily to the following:

- increased stock based compensation expense for granted stock options ($992,000) and granted stock ($1,755,000);
- increased legal fees ($70,000);
- increased in net other general and administrative expenses ($136,000);
- offset by decreased investor relations fees ($187,000); and
- offset by lower salary and related costs ($89,000).

Depreciation and amortization was $174,000 for the first quarter of 2016 as compared to $118,000 for the same period in 2015, an increase of $56,000. We are amortizing the licenses for SDF Alpha and ABO-101 and ABO-201 over the life of the patents. The increase is due to amortization of licensed technology $29,000 and depreciation $27,000.

Total operating expenses for the first quarter of 2016 were $6,395,000 as compared to total operating expenses of $2,260,000 for the same period of 2015, an increase of $4,135,000 for the reasons listed above.

Interest and miscellaneous income was $618,000 for the first quarter of 2016 as compared to $3,000 for the same period of 2015, an increase of $615,000. 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 ($591,000) related to the acquisition of Abeona Therapeutics LLC and interest income ($24,000).

Interest and other expense was $2,000 for the first quarter of 2016 as compared to $1,000 for the same period in 2015.

Net loss for the first quarter of 2016 was $5,544,000, or a $0.17 basic and diluted loss per common share as compared to a net loss of $2,000,000, or a $0.10 basic and diluted loss per common share, for the same period in 2015, an increased loss of $3,544,000.

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 March 31, 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.

None.

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 March 31, 2016, formatted in XBRL (Extensible Business Reporting Language): (i) Condensed Consolidated Balance Sheets at March 31, 2016 and December 31, 2015, (ii) Condensed Consolidated Statements of Operations for the months ended March 31, 2016 and March 31, 2015, (iii) Condensed Consolidated Statements of Stockholders' Equity for the three months ended March 31, 2016, (iv) Condensed Consolidated Statements of Cash Flows for the three months ended March 31, 2016 and March 31, 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: May 16, 2016

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

Date: May 16, 2016

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

Abeona Therapeutics Inc. and Subsidiaries

Condensed Consolidated Balance Sheets

	March 31, 2016	December 31, 2015
ASSETS	(unaudited)	
Current assets		
Cash and cash equivalents	$ 37,395,000	$ 40,138,000
Receivables	244,000	115,000
Prepaid expenses and other current assets	201,000	315,000
Total current assets	37,840,000	40,568,000
Property and equipment, net	548,000	350,000
Licensed technology, net	6,464,000	6,609,000
Goodwill	32,466,000	32,466,000
Other assets	102,000	62,000
Total assets	$ 77,420,000	$ 80,055,000
LIABILITIES AND STOCKHOLDERS' EQUITY		
Current liabilities		
Accounts payable	$ 1,042,000	$ 875,000
Current portion of deferred revenue	602,000	602,000
Contingent consideration liability	2,000,000	-
Payable due licensor	4,000,000	-
Total current liabilities	7,644,000	1,477,000
Contingent consideration liability	-	2,591,000
Payable due licensor	-	4,000,000
Long-term deferred revenue	4,115,000	4,266,000
Total liabilities	11,759,000	12,334,000
Commitments and contingencies		
Stockholders' equity		
Common stock - $.01 par value; authorized 200,000,000 shares; issued, 32,743,013 at March 31, 2016 and December 31, 2015	328,000	328,000
Additional paid-in capital	381,477,000	377,993,000
Accumulated deficit	(316,144,000)	(310,600,000)
Total stockholders' equity	65,661,000	67,721,000
Total liabilities and stockholders' equity	$ 77,420,000	$ 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 March 31,	
	2016	2015
Revenues		
License revenues	$ 151,000	$ 151,000
Royalties	84,000	107,000
Total revenues	235,000	258,000
Expenses		
Research and development	1,855,000	453,000
General and administrative	4,366,000	1,689,000
Depreciation and amortization	174,000	118,000
Total expenses	6,395,000	2,260,000
Loss from operations	(6,160,000)	(2,002,000)
Interest and miscellaneous income	618,000	3,000
Interest and other expense	(2,000)	(1,000)
	616,000	2,000
Net loss	$ (5,544,000)	$ (2,000,000)
Basic and diluted loss per common share	$ (0.17)	$ (0.10)
Weighted average number of common shares outstanding	32,743,013	19,983,751

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 capital	Accumulated deficit	Total stockholders' equity
	Shares	Amount			
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 based 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

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

Abeona Therapeutics Inc. and Subsidiaries
Condensed Consolidated Statements of Cash Flows
(unaudited)

	Three Months ended March 31,	
	2016	2015
Cash flows from operating activities:		
Net loss	$ (5,544,000)	$ (2,000,000)
Adjustments to reconcile net loss to cash used in operating activities:		
Depreciation and amortization	174,000	118,000
Stock option compensation expense	1,592,000	224,000
Stock issued to directors, employees and consultants	1,892,000	32,000
Stock issued for services	-	87,000
Change in operating assets and liabilities:		
Receivables	(129,000)	(109,000)
Prepaid expenses and other current assets	114,000	(98,000)
Other assets	(40,000)	(9,000)
Accounts payable	167,000	(1,258,000)
Contingent consideration liability	(591,000)	-
Deferred revenue	(151,000)	(150,000)
Net cash used in operating activities	(2,516,000)	(3,163,000)
Cash flows from investing activities:		
Capital expenditures	(227,000)	(9,000)
Net cash used in investing activities	(227,000)	(9,000)
Cash flows from financing activities:		
Payment of short-term debt	-	(400,000)
Net cash used in financing activities	-	(400,000)
Net decrease in cash and cash equivalents	(2,743,000)	(3,572,000)
Cash and cash equivalents at beginning of period	40,138,000	11,520,000
Cash and cash equivalents at end of period	$ 37,395,000	$ 7,948,000

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 focused on developing and delivering gene therapy and plasma-based products for severe and life-threatening rare diseases. Abeona's lead programs are ABO-101 (AAV NAGLU) and ABO-102 (AAV SGSH), adeno-associated virus (AAV)-based gene therapies for Sanfilippo syndrome (MPS IIIB and IIIA, respectively). We are also developing ABO-201 (AAV CLN3) gene therapy for Juvenile Neuronal Ceroid Lipofuscinoses (JNCL), also known as juvenile Batten disease, and ABO-301 (AAV FANCC) for Fanconi anemia (FA) disorder using a novel CRISPR/Cas9-based gene editing approach to gene therapy program for rare blood diseases. In addition, we are also developing rare plasma protein therapies including PTB-101 SDF Alpha™ (alpha-1 protease inhibitor) for inherited COPD using our proprietary SDF™ (Salt Diafiltration) ethanol-free process. Our efforts have been principally devoted to research and development, resulting in significant losses.

(1) Interim Financial Statements

The condensed consolidated balance sheet as of March 31, 2016, the condensed consolidated statements of operations for the three months ended March 31, 2016 and 2015, the condensed consolidated statements of stockholders’ equity for the three months ended March 31, 2016, and the condensed consolidated statements of cash flows for the three months ended March 31, 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 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 March 31, 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):

	March 31, 2016		December 31, 2015	
	Gross carrying value	Accumulated amortization	Gross carrying value	Accumulated Amortization
Amortizable intangible assets				
Licensed technology	$ 7,156	$ 692	$ 7,156	$ 547

Amortization expense related to intangible assets totaled $145,000 for the three months ended March 31, 2016 and totaled $116,000 for the three ended March 31, 2015. The aggregate estimated amortization expense for intangible assets remaining as of March 31, 2016 is as follows (in thousands):

2016	$ 436
2017	582
2018	582
2019	582
2020	582
over 5 years	3,700
Total	$ 6,464

(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 licensor approximate their carrying amounts due to the relatively short maturity of these instruments.

U.S. GAAP define's 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 basis as of March 31, 2016 and December 31, 2015 are summarized below:

(in thousands)

Description	As of March 31, 2016	Level 1	Level 2	Level 3	Total Gains (Losses)
Assets:					
Licensed technology (net)	$ 6,464	$ -	$ -	$ 6,464	$ -
Goodwill	$ 32,466	$ -	$ -	$ 32,466	$ -
Liabilities:					
Contingent consideration	$ 2,000	$ -	$ -	$ 2,000	$ 591

(in thousands)

Description	As of December 31, 2015	Level 1	Level 2	Level 3	Total Gains (Losses)
Liabilities:					
Contingent consideration	$ 2,591	$ -	$ -	$ 2,591	$ 3,898

(4) Stock Based Compensation and Restricted Stock Compensation

For the three months ended March 31, 2016, we recognized stock-based compensation expense of $1,592,000 for granted options. For the three months ended March 31, 2015 we recognized stock-based compensation expense of $224,000.

The following table summarizes stock-based compensation for the three months ended March 31, 2016 and 2015:

	Three months ended March 31,	
	2016	2015
Research and development	$ 341,000	$ 18,000
General and administrative	1,251,000	206,000
Stock-based compensation expense included in operating expense	$ 1,592,000	$ 224,000

For the three months ended March 31, 2016 we granted 1,315,000 stock options and for the three months ended March 31, 2015 we granted 120,000 stock options.

For the three months ended March 31, 2016, we recognized restricted stock compensation expense of $1,892,000 for granted stock. For the three months ended March 31, 2015 there was no restricted stock compensation expense recognized.

The following table summarizes restricted stock compensation expense for the three months ended March 31, 2016 and 2015:

	Three months ended March 31,	
	2016	2015
Research and development	$ 62,000	$ -
General and administrative	1,830,000	-
Stock-based compensation expense included in operating expense	$ 1,892,000	$ -

For the three months ended March 31, 2016 and 2015 no stock granted to directors or officers.

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: May 16, 2016

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

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: May 16, 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 March 31, 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 16th day of May, 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 March 31, 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 16th day of May, 2016.

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