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Edesa Biotech, Inc. (EDSA-NASDAQ)

EDSA: Initiating Coverage of Edesa Biotech, Inc.; Dermatology and GI Focused Drug Development…

Based on our probability adjusted DCF model that takes into account potential future revenues of EB01 and EB02, EDSA is valued at $5.00/share. This model is highly dependent upon continued clinical success of the company's pipeline and will be adjusted accordingly based on future clinical results.

Current Price (04/02/20)	$2.18
Valuation	**$5.00**

OUTLOOK

We are initiating coverage of Edesa Biotech, Inc. (EDSA) with a valuation of $5.00. Edesa is a biopharmaceutical company developing treatments for dermatological and gastrointestinal (GI) indications where effective therapies are needed. The company's lead development compound, EB01, is an $sPLA_2$ inhibitor for the topical treatment of chronic allergic contact dermatitis (ACD). A Phase 2b clinical trial initiated in October 2019 and we anticipate interim results in mid-2020. ACD is one of the most prevalent skin conditions in the U.S., with over one million patients suffering from chronic ACD.

The company will be evaluating the use of $sPLA_2$ inhibitors in a number of other indications, including through a Phase 2a trial in hemorrhoids disease that is anticipated to initiate in the first half of 2020. Edesa recently completed a $4.4 million direct offering and we estimate the current cash will fund operations for at least the next 12 months.

SUMMARY DATA

52-Week High	**$11.70**
52-Week Low	**$1.64**
One-Year Return (%)	**-68.40**
Beta	**-0.15**
Average Daily Volume (sh)	**23,838**
Shares Outstanding (mil)	**9**
Market Capitalization ($mil)	**$19**
Short Interest Ratio (days)	**N/A**
Institutional Ownership (%)	**0**
Insider Ownership (%)	**66**
Annual Cash Dividend	**$0.00**
Dividend Yield (%)	**0.00**
5-Yr. Historical Growth Rates	
Sales (%)	**N/A**
Earnings Per Share (%)	**N/A**
Dividend (%)	**N/A**
P/E using TTM EPS	**N/A**
P/E using 2019 Estimate	**-0.3**
P/E using 2020 Estimate	**-0.4**

Risk Level	**Average**
Type of Stock	**Small-Growth**
Industry	**Med-Biomed/Gene**

ZACKS ESTIMATES

Revenue
(in millions of $)

	Q1 (Dec)	Q2 (Mar)	Q3 (Jun)	Q4 (Sep)	Year (Sep)
2019	0.1 A	0.1 A	0.1 A	0.1 A	0.4 A
2020	0.1 A	0.1 E	0.1 E	0.1 E	0.4 E
2021					0.4 E
2022					0.4 E

Earnings per Share

	Q1 (Dec)	Q2 (Mar)	Q3 (Jun)	Q4 (Sep)	Year (Sep)
2019	-$0.26 A	-$0.31 A	-$0.30 A	-$0.15 A	-$0.55 A
2020	-$0.15 A	-$0.12 E	-$0.18 E	-$0.15 E	-$0.62 E
2021					-$0.53 E
2022					-$0.52 E

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WHAT'S NEW

Initiating Coverage



Source: Edesa Biotech, Inc.

We are initiating coverage of Edesa Biotech, Inc. (EDSA) with a valuation of $5.00. Edesa is a biopharmaceutical company focused on developing therapies for dermatological and gastrointestinal (GI) conditions where effective therapies are needed. The company's lead development products are based on inhibition of a family of enzymes called secretory phospholipase 2 ($sPLA_2$), which are responsible for hydrolyzing phospholipids to produce arachidonic acid. This process is an initiator of a broad inflammatory cascade. Inhibition of the inflammatory signaling pathway through targeting of $sPLA_2$ should lead to a superior anti-inflammatory effect since the inflammatory signal will be inhibited at its inception rather than after inflammation has occurred. Edesa is developing EB01 as a treatment for allergic contact dermatitis (ACD), which is one of the most common occupational and work-related skin conditions in the U.S., and EB02 as a treatment for hemorrhoids disease (HD), which is a common disorder characterized by itching, pain, bleeding, and difficulty defecating.

Novel Approach to Treating Inflammation

Edesa is developing treatments for dermatological and GI inflammatory conditions through inhibition of $sPLA_2$, which sits at the apex of the inflammatory signaling cascade that exerts its effects through the generation of arachidonic acid. By inhibiting the generation of arachidonic acid the inflammatory signal is stopped at the source, similar to the mechanism of action for corticosteroids, but with a better safety and tolerability profile. In addition, $sPLA_2$ is upstream from where non-steroidal anti-inflammatory drugs (NSAIDs) exert their effects.

ACD is One of the Most Prevalent Skin Conditions in the U.S.

ACD is a very common workplace and occupational skin condition and leads to approximately $2 billion in lost work, productivity, medical care, and disability payments each year in the U.S. An estimated 2.5 million individuals in the U.S. suffer from ACD and as many as one million may have chronic ACD. Current treatment options have low efficacy rates and corticosteroids have a number of significant side effects that preclude their extended use.

Near Term Interim Efficacy Analysis

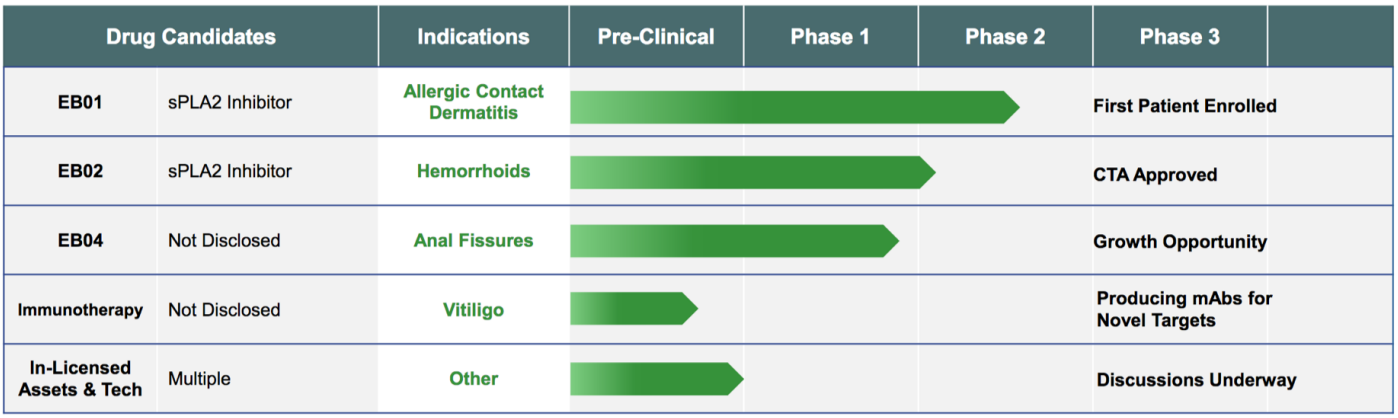
Edesa is currently conducting a Phase 2b clinical trial of EB01 in patients with ACD. The first cohort of the study will be comprised of approximately 46 patients with a primary outcome examining the mean percent change from baseline in contact dermatitis severity index (CDSI) at Day 29 following 28 days of treatment with varying strengths of EB01 cream. Results from a blinded interim analysis of the first cohort of patients is anticipated in mid-2020, at which point the study will proceed to an 80 subject second cohort, a 120 subject second cohort, or be stopped for futility.

HD Offers Additional Upside

A proof-of-concept Phase 2a clinical trial will be initiated in patients with HD to determine the efficacy of EB02. A blinded interim analysis will take place after the first approximately 48 patients are treated, with similar outcomes to the ACD trial possible. With approximately 12.5 million U.S. adults affected by HD and no FDA approved treatments, this indication could represent a large opportunity.

INVESTMENT THESIS

Edesa Biotech, Inc. (EDSA) is a biopharmaceutical company focused on the development of dermatological and gastrointestinal (GI) treatments for conditions with unmet medical needs. The company has two lead development products that both exert their anti-inflammatory activity through inhibition of secretory phospholipase A_2 ($sPLA_2$) – EB01, which is being developed as a treatment for allergic contact dermatitis (ACD); and EB02, which is being developed as a treatment for hemorrhoids disease (HD).



Source: Edesa Biotech, Inc.

EB01

EB01 is a topical vanishing cream that contains a novel, non-steroidal anti-inflammatory compound that inhibits $sPLA_2$ to provide an anti-inflammatory effect. The compound was obtained from an exclusive license agreement with Yissum Research Development Company, the technology transfer company of Hebrew University of Jerusalem Ltd. EB01 is currently being tested in a Phase 2b clinical trial of patients with ACD.

ACD Overview

Contact dermatitis is one of the most common conditions seen in dermatology clinics and accounts for the majority of occupation skin disorders in the Western world (Zack *et al*., 2017). It can be divided into two subsets; irritant contact dermatitis (ICD), which accounts for approximately 80% of cases, and allergic contact dermatitis (ACD), which makes up the other 20% of contact dermatitis cases (Nelson *et al*., 2010). Both forms of contact dermatitis involve inflammatory pathways, however ICD does not involve the immune system and may affect any person at any time depending on the concentration of the irritant and the duration of contact with the skin. In contrast, ACD is a hypersensitivity reaction that tends to only affect genetically predisposed individuals who have been previously sensitized by allergen exposure.

Hypersensitivity reactions are exaggerated immune responses to an antigen and are divided into four types. Types I – III are mediated by antibodies while Type IV is mediated by T lymphocytes. ACD is an example of a Type IV hypersensitivity reaction.

Type IV hypersensitivity has two phases: sensitization and elicitation. In sensitization, the initiating antigen (typically a hapten, or a molecule that only elicits an immune response when attached to a larger carrier such as a protein) binds to a carrier protein, and this complex is then engulfed by Langerhans cells, the antigen-presenting cells of the skin. The processed hapten/peptide complexes are presented to naïve Th1 cells in the context of major histocompatibility complex II (MHC II), and it is only in genetically susceptible individuals that have proper T cell receptors and MHC II molecules in which a reaction can occur. Successful presentation results in secretion of interleukin (IL)-1 and IL-2 that initiates proliferation of the sensitized Th1 cells. The number of sensitized Th1 cells is insufficient to lead to a reaction upon the first exposure, however memory Th1 cells are released into the circulation and are primed for the next exposure. Elicitation begins with exposure to the same hapten, with

presentation by Langerhans cells occurring to the primed Th1 cells in the epidermis, dermis, and regional draining lymph nodes. Following migration of the Th1 cells to the initial allergen contact site, inflammatory cytokines are released that causes the intercellular edema and dermal infiltrate that are common features of ACD.

It is estimated that approximately 2.5 million individuals in the U.S. suffer from ACD and approximately 1 million of those have chronic ACD, which is when someone is affected for three months or greater. Since the two types of contact dermatitis are difficult to distinguish there may exist an even larger pool of patients with undiagnosed ACD. The hands and face are the most common areas affected, but other areas include the eyelids (typical when the allergen is in cosmetics), chest, or scattered over the body.

Treatment of ACD

The simplest solution to treating ACD is to avoid contact with the inciting allergen. Unfortunately, this is not always possible as 1) the allergen is not identified in approximately 50% of patients, or 2) the allergen is unavoidable since it is present at work. In addition, once a compound is identified as an allergen that recognition is permanent, thus an inability to avoid the allergen can mean prolonged disease.

First line treatment of acute ACD is typically mid- to high-potency topical corticosteroids that are gradually tapered over a course of two weeks. Co-administration of diphenhydramine to control itching is not recommended as it can lead to cutaneous sensitization.

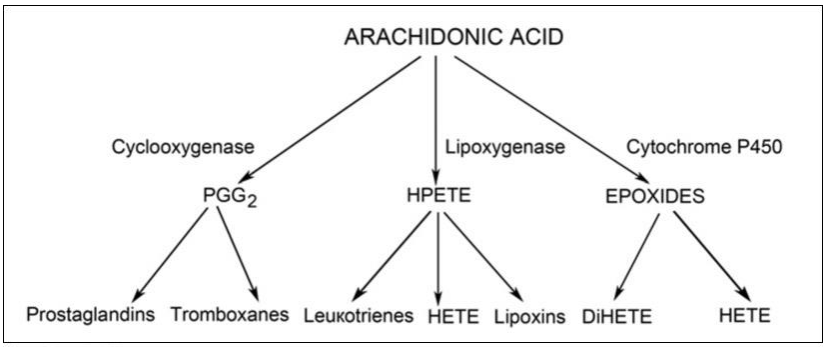
For chronic ACD, systemic corticosteroids are typically not used as the treatment may last for an extended period of time, and long-term use of corticosteroids has a number of potential side effects, including skin thinning, acne, and hair growth. Instead, low-potency topical corticosteroids are preferred due to the long-time course for treatment. Calcineurin inhibitors are not approved for use in ACD, however they are used off-label for chronic cases and in particular around sensitive areas (e.g., eyelids).

In addition to side effects, another potential issue with topical corticosteroids is the potential to develop ACD to the corticosteroid itself in a small percentage of patients (Zmudzinska *et al*., 2008). If ACD does develop to a corticosteroid it is possible to develop cross-reactivity to different corticosteroids of the same group (based on chemical structure).

Thus, while topical corticosteroids can provide relief to ACD patients, their use is associated with a number of side effects. What would be most beneficial is a treatment that is easy to use like topical corticosteroids but does not have side effects that preclude its long-term use.

Targeting PLA_2 for Treating ACD

PLA_2 is a family of enzymes that are responsible for catalyzing the cleavage of phospholipids to produce lyso-phospholipids and free fatty acids (e.g., arachidonic acid) (Burke *et al*., 2009). There are at least fifteen separate groups and five main categories of PLA_2: secreted PLA_2 ($sPLA_2$), cytosolic Ca^{2+}-dependent ($cPLA_2$), Ca^{2+}-independent intracellular PLA_2 ($iPLA_2$), the PAF acetylhydrolases, and lysosomal PLA_2. The products of PLA_2 activity initiate signaling cascades for a number of pathological conditions, including inflammatory and allergic responses. Arachidonic acid is utilized in a number of signaling pathways, including the cyclooxygenase (COX), lipoxygenase (LOX), and cytochrome P450 (CYP) pathways, to produce an array of compounds, including prostaglandins, thromboxanes, leucotrines, hydroxyeicosatetraenoic acides (HETE), and lipoxines, as shown in the following figure (Dan *et al*., 2012).



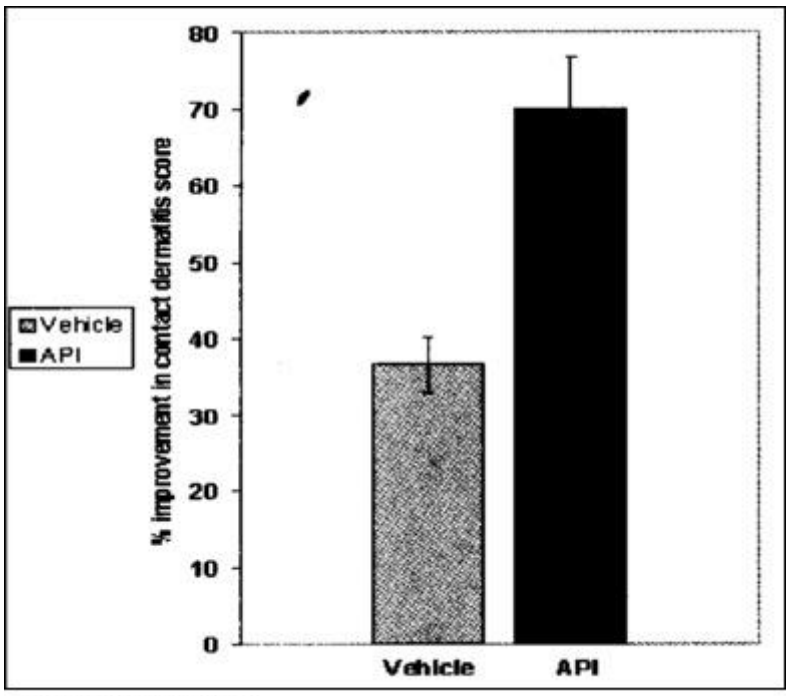
Source: Dan *et al.*, 2012

Since arachidonic acid is involved in multiple pathways that trigger inflammatory responses, the inhibition of PLA_2, and thus the production of arachidonic acid, is seen as a novel means of preventing an inflammatory response. $sPLA_2$ is constitutively expressed in skin (Haas *et al*., 2005), with some $sPLA_2$ enzymes expressed in healthy skin while others are expressed only in diseased skin. In addition, many skin inflammatory conditions (including ACD) are associated with increased eicosanoids, which implies increased PLA_2 activity. Intracellular PLA_2's are involved in a number of physiologic/homeostatic roles, thus making them inappropriate treatment targets. Therefore, $sPLA_2$ is a validated target for decreasing inflammation in ACD with an agent that cannot penetrate the cell to interact with intracellular PLA_2.

EB01 Clinical Data

In 2007, a small study was conducted in 11 ACD patients with a 1.0% EB01 topical cream (Ingber *et al*., 2007). Each participant in the study suffered from hand eczema resulting from ACD distributed symmetrically between their hands. One hand for each patient was treated twice daily with EB01-containing cream while the other hand was treated with placebo cream. Disease severity was assessed by the Contact Dermatitis Severity Index (CDSI) before treatment (Day 0), on Day 14, and Day 30. CDSI uses physician's visual assessment of dryness, scaling, redness, pruritis, and fissures, with each scored from 0 (none) to 3 (severe).

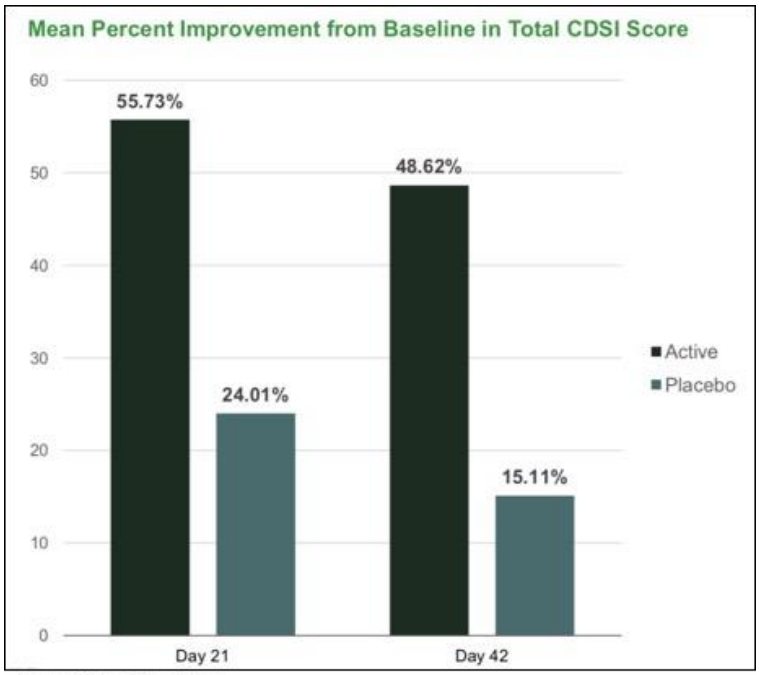
The following figure shows that the average visual score for the EB01-treated side was reduced by 69.9% while the placebo-treated side the score was reduced by 36.5% (*P*=0.0024). The vehicle was described as a "cooling cream", thus it was not unexpected to see some type of a therapeutic response. Regardless, the data showed that inhibition of PLA_2 could prove to be effective in treating ACD.



Source: Ingber *et al.*, 2007

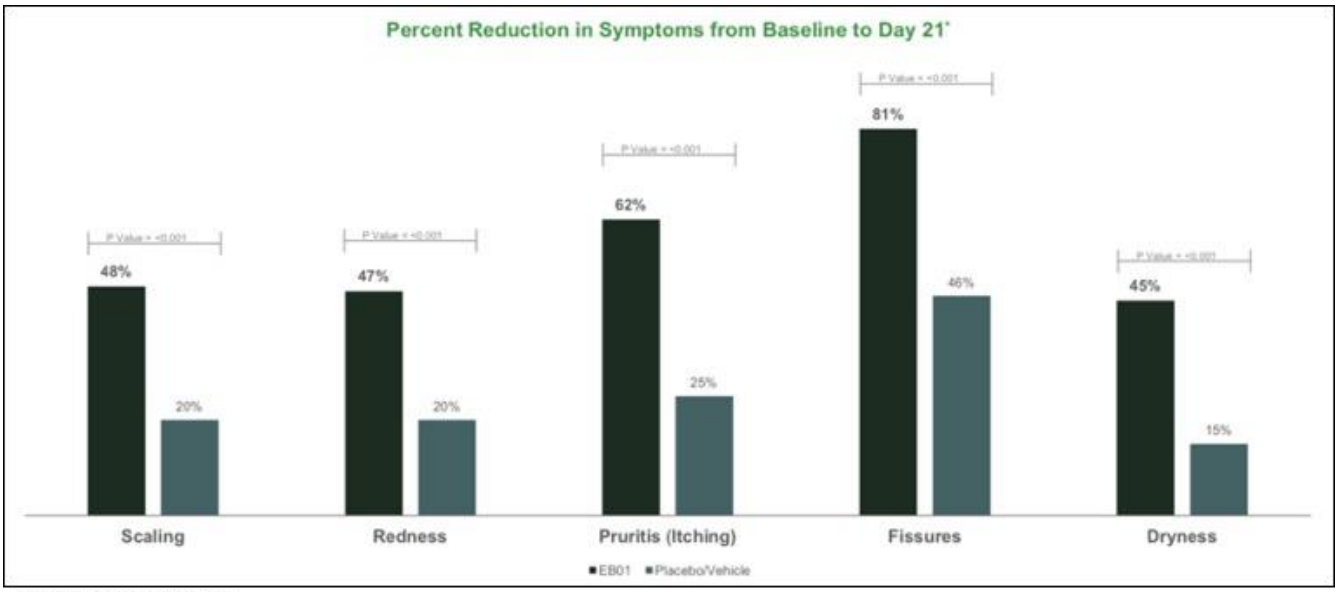
EB01 Phase 2 Clinical Trial

A larger clinical study was performed in 30 patients who had bilateral ACD with a CDSI score of at least 10 on each side and no more than a 1-point difference between lesions. A 2.0% EBI-containing cream or placebo-cream was applied twice daily for 21 days. Efficacy was measured by the change in CDSI at Day 21 (to test for efficacy) and Day 42 (to test for durability). The following image shows that on Day 21 patients treated with EB01 had a mean improvement from baseline of 55.7% compared to 24.0% for those treated with placebo cream (P<0.001). The results were durable, as shown by 48.6% decrease in total CDSI score on Day 42 for EB01-treated patients compared to a 15.1% decrease for placebo-treated patients (P=0.003).



Source: Edesa Biotech, Inc.

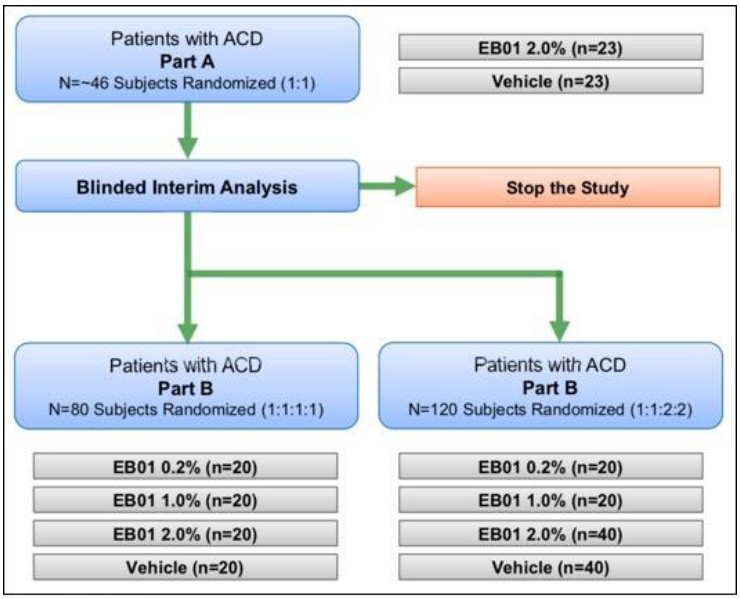
EB01 treatment significantly improved each of the components of the CDSI (dryness, scaling, redness, pruritis, and fissures) compared to placebo, as shown in the following figure.



Source: Edesa Biotech, Inc.

EB01 Phase 2b Clinical Trial

Edesa is currently conducting a Phase 2b clinical trial of EB01 2.0% cream in patients with ACD. The randomized, double blind, placebo controlled, sample size adaptive design trial is expected to enroll approximately 46 patients in Part A randomized 1:1 between EB01 and placebo for 28 days of treatment. Following the enrollment of the first cohort, a blinded interim analysis will be conducted that can have the following outcomes: 1) stop the study for futility; 2) continue to the dose ranging portion of the trial with 80 additional subjects; or 3) continue to the dose ranging portion of the trial with 120 additional subjects. The primary endpoint of the trial will measure the mean percent change from baseline in CDSI at Day 29, with secondary endpoints examining symptom reduction, dose-response relationships, and safety. An outline of the trial is shown below.



Source: Edesa Biotech, Inc.

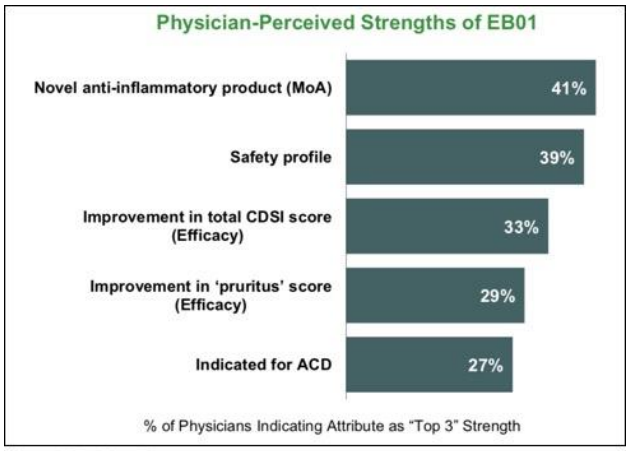
EB01 Market Opportunity

ACD is a very common condition, with some studies suggesting a prevalence as high as 20% of the general population (Alinaghi *et al*., 2019). If one member of a family develops ACD then others in the same family have a higher prevalence of developing the condition, which would suggest a genetic predisposition, although environmental exposure most likely contributes as well. Women tend to develop ACD more than men, although this is likely due to a higher prevalence of wearing jewelry, which increases contact with nickel, a known trigger of ACD.

Contact dermatitis is responsible for the majority of occupational skin disorders, with hairdressers, health care workers, food handlers, construction workers, and metal workers having high rates of ACD due to their constant and repeated contact with common allergens (Friis *et al*., 2014). The development of ACD has a negative impact on productivity and expenses, including missing work, altering work conditions or practices, and the potential to lose employees to other careers.

We estimate there are approximately 2.5 million individuals in the U.S. that suffer from ACD and that approximately 1.0 million suffer from the condition chronically. However, given how difficult it is to distinguish ACD from ICD we may be underestimating the potential number of patients.

Physicians appear to want additional treatment options for ACD. A survey conducted by Edesa showed that doctors who treat ACD patients would like a non-steroidal treatment option and that a high degree of efficacy with limited side effects would make EB01 an attractive therapeutic option.



Source: Edesa Biotech, Inc.

EB02

EB02 represents an opportunity for expansion of the $sPLA_2$ technology and is being tested for the topical treatment of hemorrhoid disease.

Hemorrhoid Disease Overview

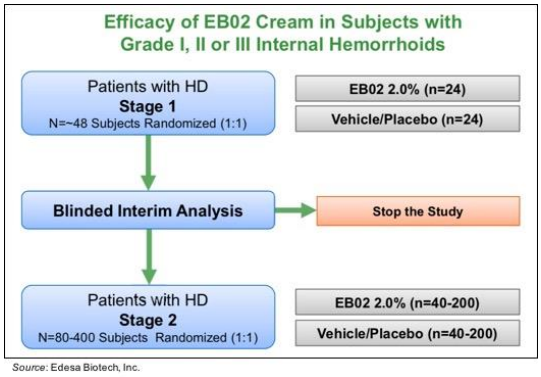
Hemorrhoid disease (HD) is the result of swelling of the veins or blood vessels in and around the anus and lower rectum that typically involves an inflammatory reaction in the vascular wall and surrounding connective tissue . The term 'hemorrhoid' is often used to describe the pathologic structure associated with HD. However, hemorrhoids are the highly vascular submucosal cushions that lie along the anal canal in three columns – the left lateral, right anterior, and right posterior positions. They are made up of elastic connective tissue and smooth muscle. Their role is to protect the anal sphincter muscles and help with closure of the anal canal.

Pathologic hemorrhoids are classified as either internal, in which the hemorrhoid is inside the anus, or external, in which it is under the skin around the anus. Internal hemorrhoids are classified according to their degree of prolapse: Grade I – protrude into the lumen of the anal canal but do not prolapse; Grade II – protrude with a bowel movement but spontaneously return when straining stops; Grade III – protrude either spontaneously or with a bowel movement and can be manually reduced; Grade IV – an irreducible prolapse.

Treatment of HD usually involves changes in diet to soften the stool, decreasing inflammation, and treating any pain. Stool softeners and a change in diet to increase the consumption of fiber help the stool to be passed more easily. The most common treatments to control inflammation are sold over the counter, with some containing low-dose topical anesthetics to alleviate discomfort or astringents to shrink swollen tissue. Low-dose steroids are typically included to decrease inflammation, with some patients receiving prescription strength steroids if their symptoms warrant. For grades III and IV, treatment includes rubber band ligation, which is used to block off blood flow to the symptomatic hemorrhoid, and conventional hemorrhoidectomy. Thus far, there are no products approved for the treatment of hemorrhoids by the FDA through the NDA process.

EB02 Phase 2 Clinical Trial

Edesa will be initiating a Phase 2a clinical trial of EB02 in patients with Grades I-III internal hemorrhoids. It will be a randomized, double blind, placebo controlled, two stage trial with an expected enrollment of approximately 48 patients in Stage 1, in which patients will be randomized 1:1 between EB02 2.0% cream or placebo cream. Patients will be treated for 14 days with a 7-day follow-up exam. Assessment will occur at baseline and Day 2, Day 7, Day 14, and Day 21 following initiation of treatment. The primary endpoint will evaluate efficacy while secondary endpoints will examine safety and tolerability. Following enrollment of the Stage 1 cohort a blinded interim analysis will be conducted in which the study can be halted for futility or move on to Stage 2 in which an additional 80-400 subjects will be enrolled. An outline of the trial is shown below.



Source: Edesa Biotech, Inc.

HD Market Opportunity

It is estimated that approximately 1 in 20 individuals and 50% of adults in the U.S. experience the symptoms of hemorrhoids (Fox *et al*., 2014). There are approximately 20 million over the counter treatments sold every year in the U.S., however there is limited evidence of their efficacy. In addition, one study estimated that at total of $88 million is spent annually on prescription hemorrhoid medications (Yang *et al*., 2019) and estimated worldwide sales of Preparation H, an over-the-counter hemorrhoid treatment, were $144 million in 2019 (EvaluatePharma).

Financials and Capital Structure

On Feb. 13, 2020, Edesa announced financial results for the first quarter of fiscal year 2020 that ended Dec. 31, 2019. The company reported revenues of $0.11 million for the three months ending Dec. 31, 2019 compared to no revenues for the three months ending Dec. 31, 2018. The revenue reflected sale of product inventory obtained in the reverse merger completed in June 2019. R&D expenses for the first quarter of fiscal year 2020 were $0.53 million compared to $0.26 million for the first quarter of fiscal year 2019. The increase was primarily due to increased expenses associated with the initiation of the EB01 clinical studies. G&A expenses for the first quarter of fiscal year 2020 were $0.68 million compared to $0.15 million for the same period last year. The increase was primarily due to increased salaries, legal, and professional fees.

As of Dec. 31, 2019, Edesa had approximately $4.3 million in cash, cash equivalents, and short-term investments. On January 8, 2020, the company closed a registered direct offering that resulted in gross proceeds of approximately $4.4 million. As of Feb. 13, 2020, the company had approximately 8.9 million common shares and when factoring in stock options and warrants the fully diluted share count was approximately 10.9 million.

Risks to Consider

Clinical Risks: Edesa is currently testing EB01 in a Phase 2b clinical trial in ACD and will be initiating a Phase 2a clinical trial of EB02 in HD. While EB01 has shown positive results in previous clinical trials of ACD patients, those trials did not have a high number of subjects and thus those results may not be replicated in the ongoing Phase 2b trial. EB02 has never been tested in HD before, thus there is a chance that it will not be successful in that indication.

Development Risk: While there are currently no FDA approved therapies for ACD or HD, even if EB01 or EB02 were approved they may not be viewed favorably by physicians or payors compared to other therapies. In addition, other companies may develop more effective therapies and render Edesa's products obsolete.

Financing Risk: While Edesa currently has sufficient funding to finance operations for at least the next 12 months, the company will require substantial additional funding to perform Phase 3 trials and commercialize any of the drug candidates currently in development.

Stock Risk: Edesa's shares currently trade on the Nasdaq under the ticker symbol EDSA. However, the stock has limited liquidity with a low average trading volume (3-month average volume of ~53,000 shares/day). Thus, large block purchases of shares could have a significant effect on the stock price and the lack of liquidity may make it difficult to exit a position.

MANAGEMENT PROFILES

Par Nijhawan, MD – Chief Executive Officer

Dr. Nijhawan is the Chief Executive Officer and Secretary of Edesa, positions he has held since forming the company in 2015. Dr. Nijhawan has 19 years of experience in cross-functional roles including finance, marketing, and business development. Prior to Edesa, Dr. Nijhawan was the founder and CEO of Medical Futures Inc, which was sold to Tribute Pharmaceuticals. Dr. Nijhawan founded and led Digestive Health Clinic into becoming Canada's largest provider of private endoscopy services. He is also the founder of Exzell Pharma. Dr. Nijhawan was Inducted into the CHLA BD Hall of fame in 2018 and Awarded Business Development Deal of the Year 2017. Dr. Nijhawan received his MD (University of Ottawa), Internship (Yale), Residency IM (Mayo), Fellowship GI (Mayo).

Michael Brooks, PhD – President

Dr. Brooks was appointed President of Edesa in January 2019. He initially joined Edesa in September 2015 as Vice President, Corporate Development and Strategy. Prior to joining Edesa, Dr. Brooks held positions of increasing responsibility at Cipher Pharmaceuticals Inc (TSX:CPH) from 2010 to 2015 and served most recently as Director of Business Development. Prior to joining Cipher, Dr. Brooks was a Post-Doctoral fellow at the University of Toronto. Dr. Brooks holds a Hons B.Sc. degree in Microbiology and a PhD in Molecular Genetics from the University of Toronto. Dr. Brooks also holds a Master of Business Administration from the Rotman School of Management where he was a Canadian Institute for Health Research (CIHR) Science to Business Scholar.

Kathi Niffenegger – Chief Financial Officer

Ms. Niffenegger was appointed Chief Financial Officer in June 2019, having previously served as CFO and Corporate Secretary of Edesa's legacy business since 2013. Ms. Niffenegger has more than 30 years of experience in accounting and finance in a range of industries. She held positions of increasing responsibility in the audit division of Glenn Burdette CPAs from 1988 to 2012 and served as technical partner. She obtained CFO experience at Martin Aviation, and began her career at Peat, Marwick, Mitchell & Co. (now KPMG LLP). Ms. Niffenegger has held leadership roles for audits of manufacturing, aquaculture, pharmaceutical and governmental grant clients, and developed specific expertise in cost accounting systems and internal controls.

Blair Gordon – Vice President, Research and Development

Dr. Gordon is responsible for the design, management, and execution of Edesa's clinical programs. He completed his PhD studies at the University of Toronto in the Department of Molecular Genetics where he was an NSERC Alexander Graham Bell Fellow. His previous roles include Business Development at Cipher Pharmaceuticals (TSX:CPH) and Medical Affairs at ArcticDX.

Gary Koppenjan – Vice President, Investor Relations and Communications

Mr. Koppenjan has more than 20 years of experience in corporate communications, marketing, and investor relations. Most recently, he led investor relations for a public life science company that was acquired by a large conglomerate. Previously, he held various marketing and communications positions at a global crop science company. During his career, Mr. Koppenjan has received multiple advertising and public relations awards for product introduction campaigns and stakeholder outreach. He is a past member of the Biotechnology Industry Organization's communications committee and the California Department of Food and Agriculture's Technical Advisory Committee. Mr. Koppenjan is a graduate of the University of California at Los Angeles.

VALUATION

We are initiating coverage of Edesa Biotech, Inc. (EDSA) with a valuation of $5.00. Edesa is a biopharmaceutical company focused on developing therapies for dermatological and gastrointestinal (GI) conditions where effective therapies are needed. The company's lead development products are based on inhibition of a family of enzymes called secretory phospholipase 2 ($sPLA_2$), which are responsible for hydrolyzing phospholipids to produce arachidonic acid. This process is an initiator of a broad inflammatory cascade. Inhibition of the inflammatory signaling pathway through targeting of $sPLA_2$ should lead to a superior anti-inflammatory effect since the inflammatory signal will be inhibited at its inception rather than after inflammation has occurred. Edesa is developing EB01 as a treatment for allergic contact dermatitis (ACD), which is one of the most common occupational and work-related skin conditions in the U.S., and EB02 as a treatment for hemorrhoids disease (HD), which is a common disorder characterized by itching, pain, bleeding, and difficulty defecating.

EB01 / EB02

EB01 and EB02 are both topical creams that contains a novel, non-steroidal anti-inflammatory compound that inhibits $sPLA_2$ to provide an anti-inflammatory effect. PLA_2 is a family of enzymes that are responsible for catalyzing the cleavage of phospholipids to produce lyso-phospholipids and free fatty acids (e.g., arachidonic acid). The products of PLA_2 activity initiate signaling cascades for a number of pathological conditions, including inflammatory and allergic responses. Since arachidonic acid is involved in multiple pathways that trigger inflammatory responses, the inhibition of PLA_2, and thus the production of arachidonic acid, is seen as a novel means of preventing an inflammatory response.

$sPLA_2$ is constitutively expressed in skin, with many skin inflammatory conditions being associated with increased eicosanoids, which implies increased PLA_2 activity. Intracellular PLA_2's are involved in a number of physiologic/homeostatic roles, thus making them inappropriate treatment targets. Therefore, $sPLA_2$ is a validated target for decreasing inflammation in ACD with an agent that cannot penetrate the cell to interact with intracellular PLA_2.

For HD, hemorrhoidal specimens can show evidence of an inflammatory reaction involving mucosal ulceration, ischemia, and thrombosis along with the presence of inflammatory cells. Thus, there are numerous processes involved in the etiology of hemorrhoids that are mediated by $sPLA_2$.

Valuation

We value Edesa using a probability adjusted discounted cash flow model that takes into account potential future revenues for EB01 and EB02. We are not including anal fissures or vitiligo in our model at this point, but these indications represent potential upside to our model.

For EB01, we model for a Phase 3 trial to initiate in ACD in 2022, an NDA filing in 2024 and approval in 2025. We estimate that the company will enter into a collaboration with a larger pharmaceutical company for marketing the drug and that Edesa will receive a 15% royalty on net sales. We forecast for peak revenue of $90 million seven years after launch. Using a 15% discount rate and a 60% probability of approval leads to a net present value for EB01 of $45 million.

For EB02, we model for a Phase 3 trial to initiate in HD in 2022, an NDA filing in 2024 and approval in 2025. As with EB01, we model for Edesa to enter into a commercialization partnership and receive a 15% royalty on net sales. We estimate a total prescription market for HD treatments of $90 million in 2020 that is growing at approximately 2% per year and we model for EB02 to eventually capture approximately 25% of this market. Using a 15% discount rate and a 50% probability of approval leads to a net present value for EB02 of $2 million.

Combining the net present value for EB01 and EB02 along with the current cash balance and potential money from exercised warrants leads to a net present value for the company of $55 million. Dividing by the fully diluted share count of 10.9 million shares leads to a valuation of $5 per share.

PROJECTED FINANCIALS

Edesa Biotech, Inc.	FY2019 A	Q1FY20 A	Q2FY20 E	Q3FY20 E	Q4FY20 E	FY2020 E	FY2021 E	FY2022 E
EB01	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0
EB02	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0
Other Income	$0.4	$0.1	$0.1	$0.1	$0.1	$0.4	$0.4	$0.4
Total Revenues	**$0.4**	**$0.1**	**$0.1**	**$0.1**	**$0.1**	**$0.4**	**$0.4**	**$0.4**
Cost of Sales	$0.1	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0
Product Gross Margin	*75%*	-	-	-	-	-	-	-
Research & Development	$1.1	$0.5	$0.6	$0.8	$1.0	$2.9	$5.0	$6.0
General & Administrative	$2.0	$0.7	$0.7	$0.8	$0.8	$3.0	$3.2	$3.5
Other (Income) Expense	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0
Operating Income	($2.8)	($1.1)	($1.2)	($1.5)	($1.7)	($5.5)	($7.8)	($9.1)
Operating Margin	-	-	-	-	-	-	-	-
Non-Operating Expenses (Net)	$0.1	($0.0)	($0.1)	($0.1)	($0.1)	($0.3)	($0.2)	($0.2)
Pre-Tax Income	($2.8)	($1.1)	($1.1)	($1.6)	($1.8)	($5.8)	($8.0)	($9.3)
Income Taxes	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0	$0
Tax Rate	*0%*	*0%*	*0%*	*0%*	*0%*	*0%*	*0%*	*0%*
Net Income	**($2.8)**	**($1.1)**	**($1.1)**	**($1.6)**	**($1.8)**	**($5.8)**	**($8.0)**	**($9.3)**
Net Margin	-	-	-	-	-	-	-	-
Reported EPS	**($0.55)**	**($0.15)**	**($0.12)**	**($0.18)**	**($0.15)**	**($0.62)**	**($0.53)**	**($0.52)**
YOY Growth	-	-	-	-	-	-	-	-
Basic Shares Outstanding	5.0	7.5	8.9	9.0	12.0	9.4	15.0	18.0

Source: Zacks Investment Research, Inc. *David Bautz, PhD*

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