Aldeyra Therapeutics (ALDX)
Rating: Buy

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Preparing for a Game of Aldehyde-and-Seek; Initiate with a Buy Rating

Stock Data	11/17/2014
Price	$7.65
Exchange	NASDAQ
Price Target	$13.00
52-Week High	$11.99
52-Week Low	$3.00
Enterprise Value (MM)	$39
Market Cap (MM)	$48
Shares Outstanding (MM)	5.6
3 Month Avg Volume	19,125
Short Interest (MM)	0.02
Balance Sheet Metrics	
Cash (MM)	$10.1
Total Debt (MM)	$1.4
Total Cash/Share	$1.82
Book Value/Share	$1.42

EPS Diluted			
Full Year - Dec	2013A	2014E	2015E
1Q	(13.03)	(4.00)A	(0.44)
2Q	(5.47)	(1.56)A	(0.45)
3Q	(9.81)	(0.36)A	(0.45)
4Q	(2.52)	(0.43)	(0.45)
FY	(30.84)	(6.35)	(1.78)

Revenue ($M) Diluted			
Full Year - Dec	2013A	2014E	2015E
1Q	0.00	0.00A	0.00
2Q	0.00	0.00A	0.00
3Q	0.00	0.00A	0.00
4Q	0.00	0.00	0.00
FY	0.00	0.00	0.00

Initiating coverage with a Buy rating and a price target of $13.00 per share. Aldeyra is embarking on a strategy to develop a therapy to trap free aldehydes, which have a high propensity to bind to macromolecules such as DNA and protein, causing irreparable damage. Free aldehydes, believed to accumulate in certain conditions, have been implicated in various inflammatory and autoimmune diseases that represent large multi-billion dollar markets. The company is developing NS2, a small molecule, that has shown efficacy in lowering free aldehydes in preclinical studies. Management is planning to initiate clinical studies to test NS2 in two rare indications: Sjögren-Larsson syndrome (SLS) and acute anterior uveitis (AAU), in 2015. If successful, we expect NS2 to enter the market as a SLS treatment in 2017 and gain the follow-on AAU indication in 2018. Management could enter into partnerships to further investigate the utility of NS2 in larger inflammatory and autoimmune indications. Considering the market potential of NS2 in SLS and AAU, upside from potential partnerships and the potential for positive operating income by 2019, we believe the shares of Aldeyra are undervalued.

Going where no one has gone before. Though free aldehydes have been implicated in many diseases, there has been no known attempts to lower their accumulation in the target tissue. With positive preclinical data with NS2, Aldeyra is planning to conduct clinical studies in SLS and AAU to prove that aldehyde burden is a major factor in various inflammatory conditions. If successful, NS2 could become a therapy for multiple inflammatory and immune conditions, such as mucositis, dermatitis, and allergies that make up nearly a $10 billion market. Positive data from a Phase 2/3 study in SLS and a Phase 2 study in AAU, expected in 2H15, could aid in generating a foothold for NS2 as an anti-inflammatory therapy.

SLS is an ultra-orphan indication. SLS is a rare disease with an estimated 2,000 patients worldwide. NS2 is believed to alleviate the most apparent feature of the disease - the dry, scaly skin and associated pruritis, which is believed to result from accumulation of lipids and a dysfunctional water-barrier in the skin. We estimate SLS is a $400 million market worldwide. Currently, we assume only US commercialization, and conservatively project risk-adjusted NS2 sales of $37 million by 2025. Once successful in the US, we expect management to commercialize NS2 in ex-US geographies, which could provide upside to our current estimates.

Success in AAU could open multiple opportunities. AAU, a rare but severe eye condition that is the cause of nearly 1% blindness in the US, is believed to result from inflammation. Positive data with NS2 from a planned Phase 2 study could solidify its utility as an anti-inflammatory therapy. This could attract major players in inflammation and autoimmune diseases, such as AbbVie (ABBV; not rated) and Amgen (AMGN; not rated) as partners in developing NS2 further. We believe AAU could be a $1.6 billion opportunity worlwide. Considering the early stage of the development program, and assuming only US commercialization, we estimate risk-adjusted sales of $40 million by 2025.

Valuation. We derived a 12-month price target of $13.00 per share based on the average of two valuation methods: 1) price-sales multiple using 8x 2025 sales estimate discounted 15%; and 2) price-earnings multiple using 25x 2025 earnings estimate discounted 15%.

Investment Summary

Aldeyra Therapeutics, headquartered in Burlington, MA, is a biopharmaceutical company specializing in the development of products for the treatment of immune-mediated, inflammatory, orphan, and other diseases that potentially are caused by free aldehydes. The company is developing a novel small molecule drug, NS2, which it discovered to trap and dispose free aldehydes. Management initially plans to evaluate NS2 in two rare diseases: Sjögren-Larsson syndrome (SLS) and acute anterior uveitis (AAU) that together have an addressable market of nearly $2 billion. Free aldehydes have been implicated in various inflammatory and autoimmune conditions. The company has conducted preclinical studies to show that NS2 has efficacy in trapping free aldehydes, diminishing inflammation, and lowering cytokine assault from inflammatory processes. A Phase 1 clinical study in healthy volunteers determined NS2 to be safe and well tolerated. The company is planning to file investigational new drug applications to initiate a Phase 2/3 study in SLS and a Phase 2 study in AAU, and expects to initiate the studies in 2015. As both diseases are considered orphan indications, these small patient studies could be conducted quickly; and if the results are successful, the drug could be launched for SLS and AAU in 2017 and 2018, respectively. We believe if the studies in AAU are successful, NS2 could potentially be evaluated in other inflammatory and auto-immune indications, such as dermatitis, allergy and mucositis, that could collectively address a nearly $10 billion market. We expect management to seek partnership with either a large biotechnology or pharmaceutical company to evaluate NS2 in these larger indications, as the company is not sufficiently funded to conduct the required development programs. Management plans to commercialize NS2 for the two orphan indications, SLS and AAU, without entering into partnerships. However, we believe, as the current development programs with NS2 proceed; management could either acquire or license complementary products to establish a stronger commercial operation. Considering the success of companies that have built operations solely on orphan indications, such as Genzyme, a subsidiary of Sanofi (SAN; not rated) or Alexion (ALXN; not rated), we believe, if NS2 enters market, the company could be a potential acquisition target. However, with Aldeyra depending on a single product and no clinical efficacy data with NS2 to date, an investment in the company has a high risk-reward ratio. Considering the market potential for NS2 in SLS and AAU, the potential to become operating income positive by 2019, and its potential to become an acquisition target, we believe the shares of Aldeyra are undervalued. Our 12-month price target of $13.00 per diluted share represents nearly 70% upside to November 17 close of $7.65 per diluted share.

Valuation

We value the shares of Aldeyra using discounted price-sales and price-earnings multiples. Considering the following: 1) NS2 yet to be studied in SLS and AAU patients; 2) the studies could take longer than expected to complete; 3) execution risk with no specialized sales force in place; and 4) NS2 adoption could be slower than expected, we are assuming a 15% discount rate; the high end of the industry range of 12-15% in both price-sales multiple analysis and price-earnings multiple analysis. Hence, considering that the company is pursuing orphan indications that have a longer market life, we are assuming an 8x multiple, higher-end of the industry average 6-8x, to our 2025 sales and an 15% discount rate in our price-sales multiple analysis, we arrive at a fair value of $13.13 per diluted share. In our price-earnings multiple analysis, considering that we are uncertain of some of the pushes and pulls on operating margins at present time, we are assuming conservatively a 25x multiple, low end of the industry average 25-28x, to our 2025 EPS estimate of $1.77 and a 15% discount rate, we obtain a fair value of $13.07 per diluted share. Averaging the results of these two methods, we obtain a 12-month price target of $13.10, which we round to $13.00 per share.

Catalysts and Milestones for 2014-2015

- File IND to initiate Phase 2/3 study in SLS (4Q14/1Q15)
- File IND to initiate Phase 2 study in AAU (4Q14/1Q15)
- Publication of data from Phase 2/3 study in SLS (2H15)
- Publication of data from Phase 2 study in AAU(2H15)

Risk Analysis

In addition to risks associated with emerging biotechnology companies, specific risks to be considered are:

Clinical risk. Management is planning to initiate clinical studies with NS2 as a treatment for Sjögren-Larsson syndrome and acute anterior uveitis in 2015. Outside of a 45-patient Phase 1 study with NS2 in an eye drop formulation, the drug has yet to show efficacy in humans. If the clinical studies are unable to show efficacy with statistical significance, in either of the conditions, the market potential of NS2 could be lower than expected.

The skin symptoms are thought to be the result of mutations in the enzyme, Fatty Aldehyde Dehydrogenase (FALDH) that converts long-chain aldehydes to fatty acids. In addition, SLS patients also have elevated levels of fatty alcohols and diminished levels of fatty acids. NS2 is proposed to trap only aldehydes and is not expected to have any impact on either fatty alcohols or fatty acids. NS2 may fail in the clinic if either fatty alcohols or fatty acids have a greater impact on the condition.

Aldehydes are believed to be mediators of inflammation. However, to date, there has been no approved product that has shown clinical efficacy in trapping or lowering aldehydes. Hence, NS2 may not be clinically effective in lowering or eliminating the SLS symptoms.

Commercial risk. The management is planning to commercialize NS2 for SLS and uveitis without engaging a commercial partner. Though there may not be a need for an extensive commercial infrastructure as the indications are rare, yet might require specialized salesforce for detailing. Management, with its limited experience, may not be able to produce the desired commercial results.

NS2 is expected to be used as a chronic treatment for SLS. However, the long-term safety data of NS2 is not available neither in animals nor humans. If the company is not able to show long-term safety of NS2, the product may not achieve its full commercial potential.

Regulatory risk. NS2 has a unique mechanism of action that has not been proven before in a clinical setting. The FDA might require extensive safety and efficacy data before approving the drug.

Management plans to use a visual ichthyosis scale to determine the efficacy of NS2 in lowering the skin symptoms in SLS. However, the scale has not been validated. The FDA may not accept the efficacy data based on the visual ichthyosis scale alone.

Financial risk. The company's revenue potential is dependent on the success of NS2. If the product is unsuccessful either in the clinic or during the regulatory process, the company may not be able to be generate any revenues in the near-term.

Aldeyra has not achieved profitability to date and could potentially continue to incur operating losses in the future. We believe any additional capital raises to maintain operations could potentially dilute ownership interest.

Legal and intellectual property risk. At present, there are compositions of matter patents issued in the United States and other countries to protect NS2. Management could potentially fail to protect their intellectual property and/or may infringe on the proprietary rights of third parties in order to operate. Such an event could adversely affect company's business.

Investment Highlights

An unique technology to trap aldehydes. Free aldehydes are intermediary products of biochemical pathways. Due to their highly reactive nature, these intermediaries react with any macromolecules (DNA or protein) in close proximity. When free aldehydes accumulate, they promote inflammation and become mediators of many immune-mediated inflammatory diseases. Free aldehydes are thought to be partially involved in many immune-related or inflammation-related diseases, such as systemic lupus erythematosus, uveitis, multiple sclerosis, atherosclerosis and diabetic nephropathy. Aldeyra is developing a small molecule technology that could trap these free radicals in an irreversible manner and lower their impact on the downstream inflammatory and immune processes. Considering the above mentioned diseases, the technology could potentially address a large multi-billion dollar market. The company is developing NS2, a small molecule aldehyde trap and is planning to initially test in two indications: Sjögren-Larsson syndrome and acute anterior uveitis.

Sjögren-Larsson syndrome and acute anterior uveitis are orphan indications. Sjögren-Larsson syndrome (SLS) is a rare condition that is believed to affect about 2,000 patients in the United States and Europe. SLS is a congenital disease and patients suffer from ichthyosis, a severe scaly dry skin condition, mental delay, and spasticity. The disease is believed to be the result of mutations of an enzyme called fatty aldehyde dehydrogenase. Fatty aldehydes, the substrate for the enzyme, accumulate to high levels in the skin as a result of an ineffective enzyme and are thought to be mediators of ichthyosis in SLS. Aldeyra believes, by trapping the free aldehydes with NS2, the drug can lower the primary day-to-day complaint of pruritus in SLS patients.

Acute anterior uveitis is an eye disease in which patients suffer from a rapid onset of pain, sensitivity to light and could eventually lead to loss of vision. The annual incidence of AAU in US is nearly 25,000. About a third of the patients experience at least one episode per year. Those with recurrent episodes develop cataracts, glaucoma and retinal dysfunction. It is believed that approximately 10% of blindness in the US is caused by uveitis. A higher amount of free aldehydes have been noted in AAU patients and could be the potential cause for the inflammatory conditions that are seen in these patients. At present, corticosteroids are prescribed for treating AAU patients. However, protracted use of corticosteroids increases the probability of cataracts, glaucoma, infection, and corneal ulceration. The company company expects to initiate a Phase 2 study in AAU patients in 2015.

We project a risk-adjusted sales of $77 million for the two indications by 2025.

NS2 might require a short runway. As orphan indications, SLS and AAU do not require large clinical studies and could receive approval in a relatively short period. We expect NS2 to potentially receive marketing approval for the treatment of SLS from the FDA in late 2016 or early 2017. We anticipate that management could price the drug around $180,000 per year for treating SLS patients and at a lower $30,000 per year for a AAU patient.

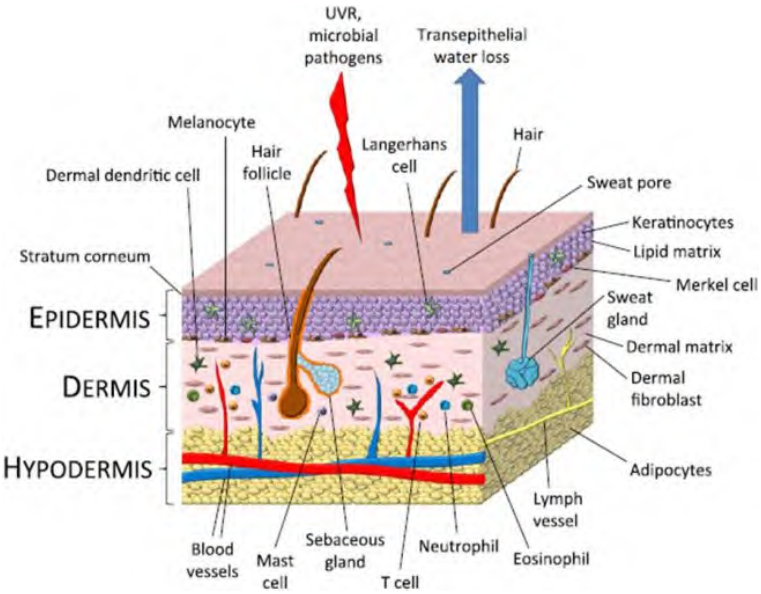
NS2 has potential to enter large anti-inflammatory and autoimmune disease markets. AAU is thought to be the result of autoimmune-related inflammatory processes in the eye. We believe success in the initial clinical study in AAU patients could show a utility for NS2 in other inflammatory and autoimmune indications. Although, Aldeyra may not be able to fund all potential development programs, it could enter into partnerships to develop NS2 for these other multi-billion dollar indications.

High acquisition target potential. NS2, if successful in SLS and AAU, could generate a long-term revenue stream, as orphan indications have limited competition. Additionally, as the platform has the potential to expand into other large disease markets, we expect the company to become an acquisition target. Note that Genzyme was acquired by Sanofi (SNY; not rated) in 2011 at 4.7x 2010 sales and Alexion (ALXN; not rated) is currently trading at 18x sales (trailing twelve months).

Scientific Overview and Background

Skin is the first line of defense. The skin is the largest organ as it covers the entire surface area of the body. As it is the organ that first comes in contact with every conceivable threat to the body, it's most important function is to defend the body. The skin provides protection by: 1) providing a barrier against mechanical, thermal, or physical injury, and hazardous substances; 2) preventing loss of moisture; 3) reducing harmful effects of UV radiation; 4) acting as a sensory organ to touch and temperature; 5) helping to regulate temperature; and 6) acting as an immune organ against infections.

Exhibit 1: Structure of Skin



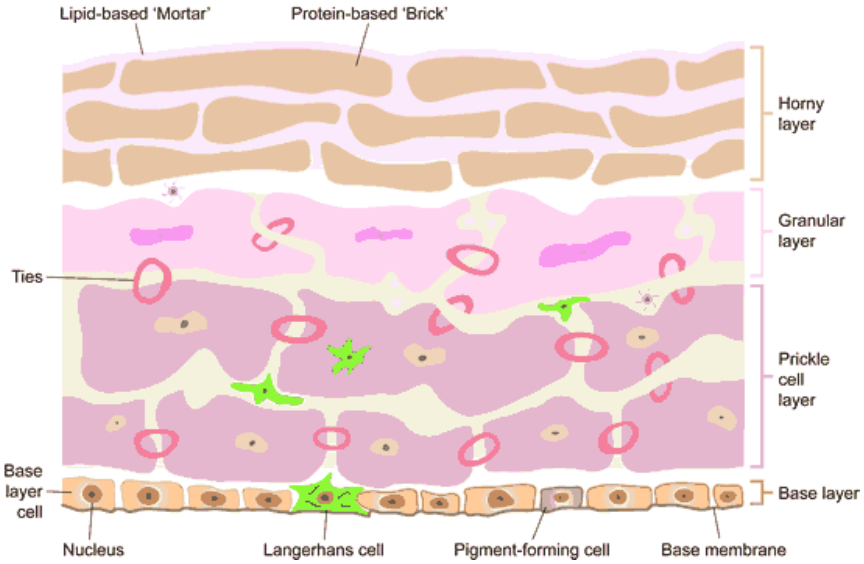
Source: http://cdn.intechopen.com/pdfs-wm/43810.pdf

The skin has three main layers: 1) epidermis; 2) dermis; and 3) subcutaneous layer. The epidermis is the outermost layer that is elastic in nature and includes three different cell types: 1) keratinocytes; 2) corneocytes; and 3) melanocytes. Keratinocytes are the main cells of the epidermis that form the base and they are continually generated through cell division. These cells gradually die as they move up to the surface and become flattened. The flattened dead keratinocytes are called corneocytes that make up the outer layer of the epidermis called the stratum corneum or horny layer. This outermost protective layer is continually worn away and shed. Melanocytes are cells that produce the pigment, melanin, which provides the color to the skin and also protects from UV radiation. The second layer, dermis, helps in regulating the temperature of the body through sweat glands, hair follicles, and sebaceous glands. Sweat glands produce sweat that travels to the surface of the skin through sweat ducts to openings in the epidermis called pores. Along with sweat, the sebum (oil) from the sebaceous glands makes up a film on the skin to regulate body temperature. The sebum also keeps the hair free from dust and bacteria. Hair follicles are pits through which the hair grows. The subcutaneous layer is the third innermost layer of the skin, made of connective tissue and fat, and it acts as a good insulator.

Skin needs to maintain water content to be flexible. The elasticity of the skin is dependent on the water content in the stratum corneum, the outermost layer of the epidermis. The need to maintain a certain amount of water also makes the skin an efficient water barrier. The corneocytes of the epidermis form a brick-like structure that are held together by a layer of lipids and water, and together act like mortar. The lipid layer is made of crystal-like structures that are impermeable to water and other type of lipids that allow water to percolate through, thus making the barrier semi-permeable. The skin also produces natural moisturizing factors (NMFs) that help in retaining water in the skin. If the skin is kept moist for a long time, the production of

NMFs stops. Whereas when the skin is low on water, the corneocytes do not shed as they do normally, and the skin becomes rough, thick and flaky, eventually causing the skin to lose elasticity and crack.

Exhibit 2: Skin as a Water Barrier



Source: http://www.hse.gov.uk/skin/professional/causes/understand.htm

Lipids are necessary components of skin's water barrier. The stratum corneum that houses the water barrier is made of equimolar amounts of three kinds of lipids: ceramides; cholesterol; and free fatty acids. The lipid composition of the stratum corneum is important for a functional water barrier. This was demonstrated in an experimental model by topically administering inhibitors of the enzymatic pathways that synthesized these lipids which resulted in a compromised water barrier. Similarly, genetic defects that result in errors in lipid metabolism also cause a chronic water barrier defect and the skin manifests a scaly appearance which is known as icthyosis.

Free aldehydes cause multiple toxicities. Free aldehydes, products of multiple metabolic processes, are believed to initiate downstream inflammatory reactions. As the levels of free aldehydes increase, they bind to proteins, lipids, carbohydrates, and DNA. Some of these adducts, being indigestible, aggregate initiating inflammatory reactions which lead to cellular dysfunction. To reduce these toxicities, all living organisms have enzymes, called aldehyde dehydrogenases, which detoxify aldehydes. If the aldehyde dehydrogenase activity is diminished, the levels of free aldehydes are higher, as seen with certain inflammatory diseases, including: autoimmune disease, neurodegenerative disease, and cardiovascular diseases.

Sjögren-Larsson Syndrome is a rare genetic disorder. Nearly sixty years ago, a Swedish psychiatrist and geneticist first described a combination of symptoms in patients from North Sweden, which included: ichthyosis (scaly skin); mental retardation; and spastic diplegia (constant stiffness of muscles of lower extremities) or tetraplegia (total loss of all limbs).[1] A year later, Sjögren, along with a biostatistician, described their clinical findings of 28 patients, establishing it as an autosomal recessive disease, Sjögren Larsson Syndrome (SLS).[2] It is believed that the incidence of SLS is approximately one in 250,000 people or approximately 1,000 people in the US. It is believed that the worldwide prevalence is about 2,000 patients.

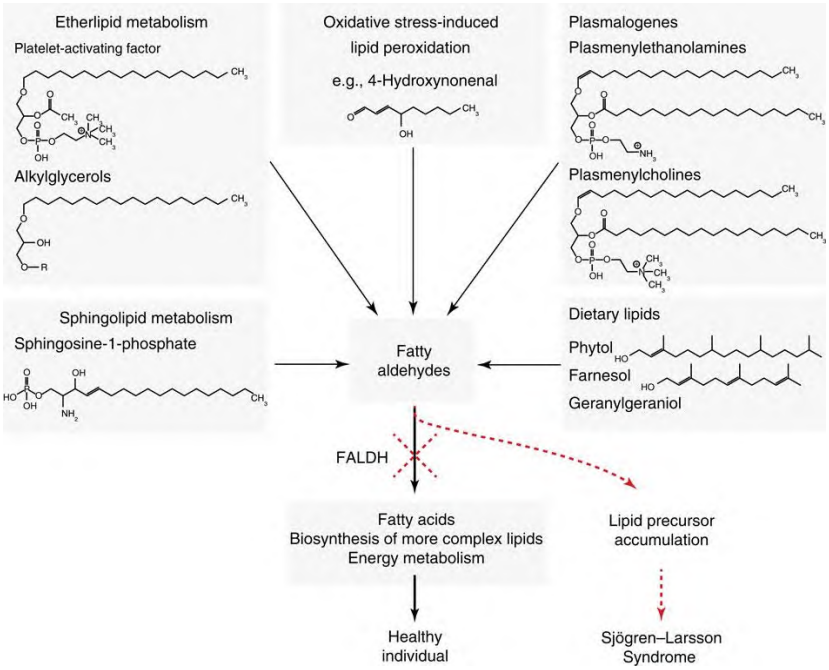
[1] Sjögren T. *Acta Genetica.* 1956;6:80–91

[2] Sjögren T, Larsson T. *Acta Psychiatr Neuro. Scand*. 1957; 32: 1–113

Symptoms are noticed at birth. As in the case of any congenital disease, children suffering from SLS are born with abnormalities. The skin conditions are believed to start as early as 23 weeks of gestation.[3] Patients tend to be born pre-term. The disease is manifested at birth by icthyosis, which is normally noticed at the nape of the neck, trunk and extremities. At the age of two, neurological symptoms, such as movement disorders due to spastic diplegia or spastic tetraplegia, are evident. Nearly half of the patients require crutches or braces to walk. These patients also have cognitive deficits and it is rare to find a patient with normal cognitive functions. Most SLS patients live into adulthood.[4]

Faulty FALDH. SLS is believed to be the result of various mutations in the, Fatty Aldehyde Dehydrogenase (FALDH) enzyme, a key component in the detoxification pathway of aldehydes. With a non-functional FALDH, aldehydes accumulate, causing the dermatological and neurological symptoms. Both patients and caregivers seek relief from Icthyosis, which is their major concern. It is believed that with the accumulation of aldehydes the lipids, that form the water barrier in the skin, are modified. This modification of lipids and hence the water barrier eventually causes water loss from the skin. To compensate, the skin thickens, which causes further dryness. It is hypothesized that by lowering the accumulation of aldehydes in the skin, lipid modification and moisture barrier dysfunction can be inhibited. At this time, moisturizers and some non-specific topical creams, including keratinolytics (acids that soften skin), are used for temporary relief.

Exhibit 3: Role of FALDH in Metabolic Pathways

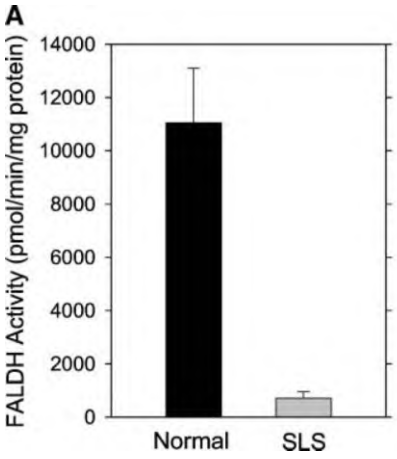


Source: Keller MA. et al., Nat Commun. 2014 (5):4439

SLS patients lack FALDH activity. To understand the impact of FALDH in generating the skin symptoms of SLS, lipid metabolism was investigated in cultured keratinocytes derived from SLS patients.[5] The investigators noted that less than 10% of FALDH activity in the SLS keratinocytes compared to normal controls. The data indicated that the FALDH deficiency in SLS keratinocytes causes fatty alcohol accumulation. Additionally, it was noted that lipid abnormalities in cultured SLS keratinocytes may be related to stratum corneum dysfunction and ichthyosis in SLS.

[3] Kousseff BG et.al. *J Pediatr*. 1982; 101: 998–1001
[4] Rizzo WB Mol. *Genet Metab*. 2007; 90:1-9
[5] Rizzo WL et. al*., J Lipid Res*. 2008; (49): 410–419

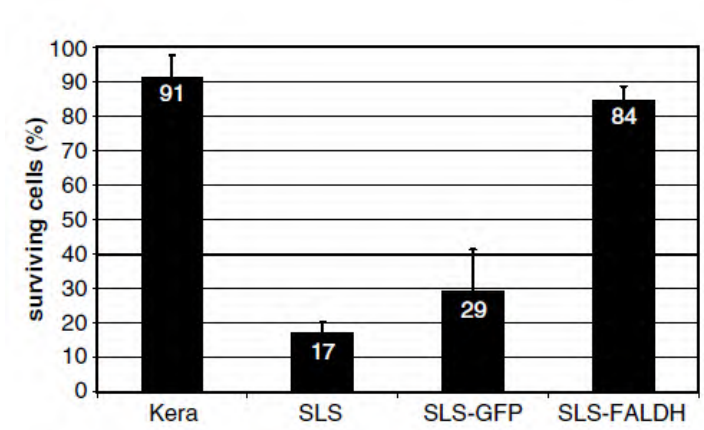
Exhibit 4: FALDH Activity in Keratinocytes from SLS Patients



Source: Rizzo WL et. al., J Lipid Res. 2008 (49): 410–419.

Restoration of FALDH activity aids in higher resistance to free aldehydes. In an attempt to identify different ways to restore FALDH activity in SLS, investigators transferred a functional gene into cultured keratinocytes from an SLS patient.[6] Normally, if keratinocytes lack FALDH, treatment with long chain aldehydes, such as octadecanal, could result in death of the cells. As seen in Exhibit 5, only 17% of cells survived treatment with octadecanal, whereas 84% of FALDH-transduced cells survived, These data underscores the impact of loss of FALDH activity in SLS.

Exhibit 5: FALDH Restoration Leads to Higher Resistance to Long-chain Aldehyde Insult



Source: Haug S and Braun-Falco M, Gene Therapy 2006 (13): 1021-1026

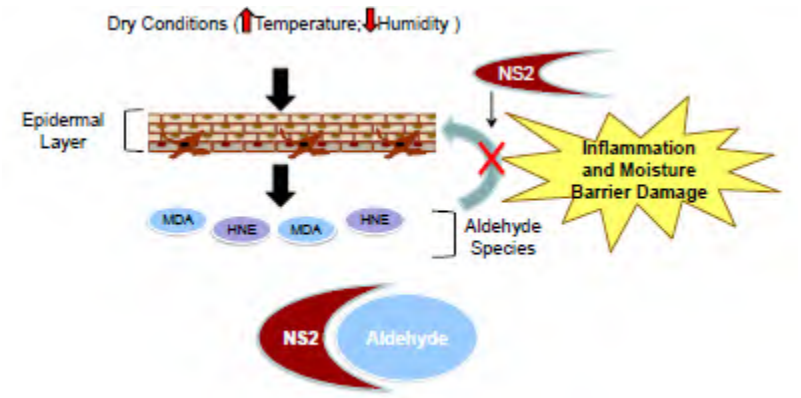
Uveitis is an ocular inflammatory condition. The uvea is the middle portion of the eye is made of three structures: iris, ciliary body, and choroid. These structures aid in many functions, such as adjusting for light or different distances of objects. Uveitis is an inflammation of any one of these structures. Uveitis can be caused by autoimmune disorders such as rheumatoid arthritis or ankylosing spondylitis, infection, or exposure to toxins. The disease is classified as anterior, intermediate or posterior, depending on the part of the uvea affected by inflammation, where the most common form is anterior uveitis. It is believed that the incidence of uveitis is nearly 25,000 in the US. The spectrum of the disease can range froma transient to a recurrent condition, which can eventually lead to retinal dysfunction. Uveitis is believed to occur when the eye is immunocompromised, either through infection or by autoimmune conditions.

[6] Haug S and Braun-Falco M. *Gene Therapy*. 2006; (13): 1021-1026

Free aldehydes accumulate in uveitis. Free aldehydes have been postulated as a causative agent that could initiate inflammation in the anterior uveitis. In an animal model of endotoxin-derived uveitis, when the aqueous humor (thin watery fluid that fills space between cornea and iris) was assayed for malondialdehyde (MDA, a marker for free aldehyde presence), after eye treatment with an endotoxin, the amount of MDA was significantly higher compared to controls.[7] In an 89-patient study (45 acute anterior uveitis and 43 healthy) measurement of serum MDA levels in the uveitis patients demonstrated a statistically significant, higher amount of aldehyde compared to control subjects.[8] These results indicate that there is accumulation of aldehydes in acute anterior uveitis. At present, patients are treated with corticosteroids. However, long-term use of corticosteroids could potentially increase the risk of glaucoma; and hence, there is a need for new therapies.

Aldeyra's NS2 traps aldehydes. The company is developing a small molecule drug called NS2 that is designed to trap free aldehydes. NS2 binds to free aldehydes at a rapid rate compared to cellular proteins. It has also been noted that when NS2-aldehyde adducts form, they are stable and irreversible. The bound NS2-aldehyde is believed to be transported into lysosomes for degradation. Thus, NS2 is believed to aid in lowering aldehyde levels.

Exhibit 6: NS2 Mechanism of Action

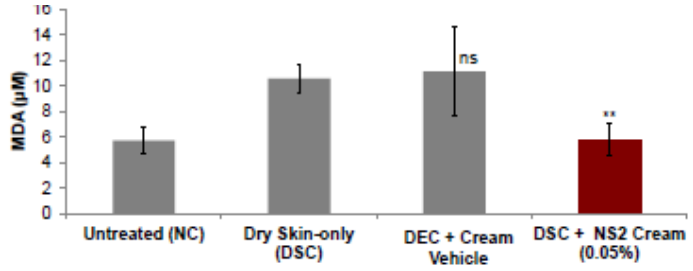


Source: Company reports

Preclinical data with NS2 shows reduction in aldehyde burden. A reconstructed human epidermis was used to test the utility of a topical dermatologic formulation of NS2 (0.05% w/v) under normal and dry skin conditions (DSC). The tissues were acclimated before treatment for 24 hours and then treated topically with or without NS2. Tissues were incubated under normal conditions (37°C; >40% relative humidity) or DSC (40°C; <40% relative humidity) for 72 hours. MDA is a lipid that is elevated in inflammatory conditions, such as psoriasis, atopic dermatitis, anterior uveitis and rosacea, and is considered as a marker of inflammations. As seen in Exhibit 7, tissue samples grown in DSC had higher amounts of MDA compared to untreated tissue. Upon treatment with NS2, the tissue levels of MDA were reduced significantly ($p<0.01$).

[7] Satici A et al., Eur. *J Opthalmol*. 2003 (13): 779-783
[8] Turk A et. al., *Ocul Immunol Inflamm*. 2014 (22): 127-132

Exhibit 7: Treatment with NS2 lowers MDA Levels



Source: Company Reports

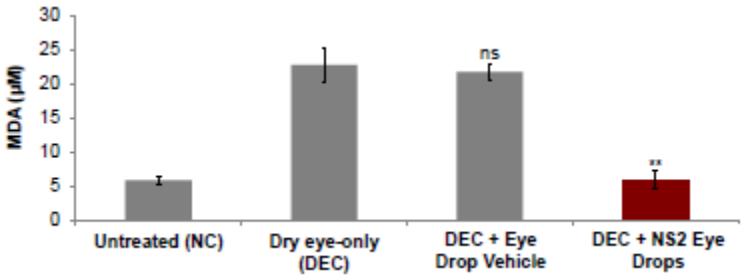
Phase 1 study with NS2 shows the drug is tolerated. The company conducted a double-blind, placebo-controlled Phase 1 clinical trial with 0.25% and 0.5% NS2 administered as an eye drop in 48 healthy volunteers. Up to four doses per day were administered per volunteer for seven days for both concentrations. No NS2 was detectable in the plasma, and was well tolerated in all subjects throughout the duration of the study.

Phase 2/3 study to evaluate NS2 as an SLS treatment is expected to initiate in the near-term. Management is planning to initiate a 12-patient Phase 2/3 study with NS2 (0.05% w/v) to evaluate as a treatment for SLS. This placebo-controlled study is expected to test an 8-week treatment of NS2 and utilize a visual rating for assessment. We expect further details regarding the design and the time-line of the study in the near future. Due to the short-term nature of the study, we expect data from the trial could be published in 2H15. Conservatively, we assume a potential pivotal study could be required by the FDA for approval. Hence, we assume the drug, if successful, could to be launched in 2017.

First drug to be tested for SLS. Currently there is no approved treatment for SLS. Parents and caregivers perceive ichthyosis as the immediate concern that requires constant attention. Topical moisturizing creams are generally used to keep the skin hydrated. Though systemic retinoids seem to show benefit, their potential adverse effects, such as growth delay, does not allow them to be used in children. NS2, if successful, could become the first approved drug for SLS.

NS2 has the potential to lower aldehydes in eye. In a preclinical study, reconstructed human cornea-like tissue was treated with an eye drop formulation of NS2 (0.5% w/v) was administered to test the drug's utility in lowering aldehyde burden. The ocular tissues were incubated under normal (37^{o}C; >40% relative humidity) or under dry eye conditions (DEC; 40^{o}C; <40% RH) for 72 hours and levels of MDA were assayed. The results showed that on NS2 treatment, the tissue incubated under DEC condition had significantly lower levels of MDA compared to control treatment.

Exhibit 8: Treatment with NS2 lowers MDA Levels



Source: Company Reports

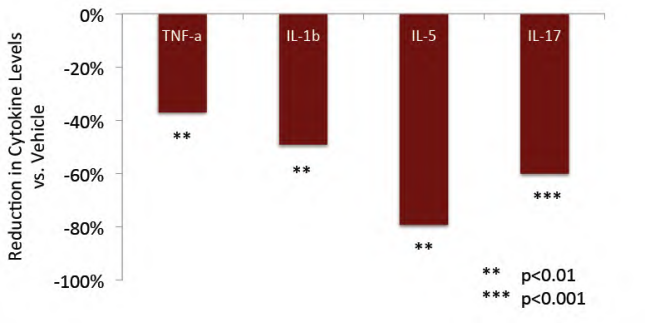
Phase 2 study with NS2 as a treatment for uveitis. The management is planning to initiate a Phase 2 study to evaluate NS2 as a treatment of uveitis in 4Q14/1Q15. The trial is expected to be a 45-patient placebo-controlled study, designed to test a 0.5% NS2 as an eye drop over an 8-week treatment period. Endpoints for

the study could be anterior chamber cell score, pain and visual acuity. We believe data from the study could be available by the end of 2015. Success in the Phase 2 study could help elucidate the potential for NS2 as a therapy for other inflammatory conditions.

Steroids provide some symptomatic relief. Though corticosteroids are prescribed to provide immediate relief from inflammation, long-term treatment is avoided due to their side-effect profile: nausea, heartburn, weight gain, and fluid retention. In refractory cases, biologicals, such as Humira are prescribed as AAU is thought to be related to autoimmune disorders. If successful, NS2 could be the first drug approved for AAU.

Preclinical data shows NS2 as a potential anti-inflammatory therapy. Multiple preclinical studies have been conducted to investigate NS2's potential as an anti-inflammatory therapy. In order to test NS2 to reduce the release of pro-inflammatory cytokines as a result of an autoimmune reaction, mice were treated with NS2 or a placebo about 30 minutes before endotoxin exposure. Cytokine levels were measured two hours post exposure. The results showed that there were statistically significant, lower levels of TNFα, IL-1β, IL-5, and IL-7 when treated with NS2 compared to placebo.

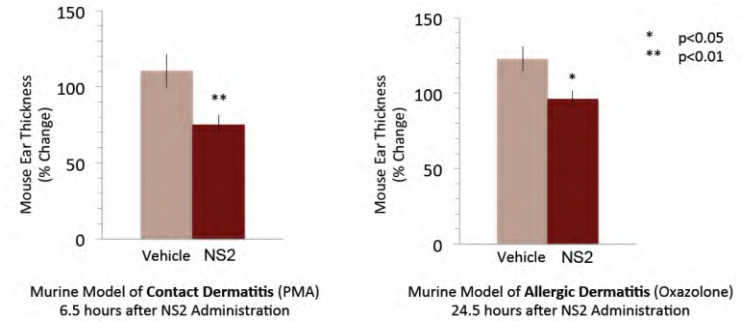
Exhibit 9: Treatment with NS2 lowers MDA Levels



Source: Company Reports

NS2 also seems to have potential in contact dermatitis and allergic dermatitis. The drug was tested in a murine model for these two indications. The model correlates the efficacy of a test drug with the amount of decline in skin inflammation caused by a known irritant, such as phorbol myristate acetate (PMA), applied to the animal. When PMA was applied to the the skin of the experimental mouse and then treated with NS2, there was statistically significant, lower skin inflammation compared to vehicle. Similarly, in a mouse model of allergic dermatitis, which used oxazolone, a known respiratory allergen, NS2 treatment significantly lowered skin inflammation compared to placebo treatment. We believe management could initiate clinical programs in these inflammatory conditions if the anticipated Phase 2 uveitis study is successful.

Exhibit 10: Treatment with NS2 lowers MDA Levels



Source: Company Reports

Financials

Revenues. In the first nine months of FY14 Aldeyra did not record revenues. We believe Aldeyra could gain approval to market NS2 for the treatment of SLS in 2017. The drug is also being evaluated as a treatment for AAU. We expect the drug to be approved for AAU in 2018. At present time, we are considering only the US market in our financial model. Management's long-term plan is to commercialize the drug outside the United States as well. We believe, if successful, ex-US revenues could provide upside to our current estimates.

SLS (US market): In our current financial model, we are assuming revenues from marketing NS2 for treating SLS patients in the US. We believe approximately 1,300 patients could be diagnosed with SLS during 2017, the year when NS2 could receive marketing approval. We believe management could potentially price NS2 at $180,000 per year per patient. With Phase 2 studies yet to start, we assume a 25% probability for the launch of the drug in 2017. Accordingly, we project SLS-related US NS2 risk-adjusted revenues to grow from $1 million in 2017 to $37 million in 2025.

Exhibit 11: NS2 Revenues, 2017E-2025E

Sjögren Larsson Syndrome	2017E	2018E	2019E	2020E	2021E	2022E	2023E	2024E	2025E
Sjögren Larsson Syndrome patients	**1,305**	**1,314**	**1,324**	**1,333**	**1,342**	**1,352**	**1,361**	**1,371**	**1,381**
NS2 penetration	2%	5%	8%	10%	20%	30%	40%	50%	60%
US SLS revenues, risk-adjusted (million)	**$1**	**$3**	**$5**	**$6**	**$12**	**$18**	**$25**	**$31**	**$37**

US Acute Anterior Uveitis	2017E	2018E	2019E	2020E	2021E	2022E	2023E	2024E	2025E
US Acute Anterior Uveitis population	25,127	25,304	25,482	25,662	25,842	26,024	26,207	26,392	26,578
NS2 penetration	0%	2%	5%	8%	10%	12%	15%	18%	20%
US Acute Anterior Uveitis revenues, risk-adjusted (million)	**$0**	**$4**	**$10**	**$15**	**$19**	**$23**	**$29**	**$36**	**$40**

Source: H.C. Wainwright Estimates

AAU (US market): We believe management could receive FDA approval to market NS2 for the treatment of AAU in 2018. We conservatively estimate that the product could achieve a peak market penetration of 20% by the end of 2025. Estimating that the cost of the drug is $30,000 per patient per year and assuming a 25% probability of launch, we expect the risk-adjusted US sales to achieve nearly $40 million by the end of 2025.

Net income and EPS. Aldeyra announced a net loss of $2.0 million and an EPS of ($0.36) at the end of 3Q14 compared to a net loss of $1.7 million and EPS of ($9.81) in 3Q13. We estimate a net loss of approximately $9.8 million and an diluted EPS of ($6.35) for full year 2014 compared to a net loss of $20.5 million and an EPS of ($1.11) in 2013. We believe 2019 could be the first year of a positive net income of approximately $100,000 resulting in EPS of $0.01. Refer to the detailed income statement for our net income and EPS estimates for the forecast period, 2014E-2025E.

Cash. Aldeyra ended its 3Q14 with $10.1 million in cash and short-term investments. We expect the company to close 2014 with approximately $7.8 million in cash and short-term investments. We estimate operating cash use of nearly $10.4 million in 2015. Although Aldeyra could fund its operations for the next 12 months, we believe management could potentially raise $30 million during 3Q15 to maintain operations as they progress towards profitability, which is expected in 2019.

Valuation

We value the shares of Aldeyra using discounted price-sales and price-earnings multiples. Considering the following: 1) NS2 yet to be studied in SLS and AAU patients; 2) the studies could take longer than expected to complete; 3) execution risk with no specialized sales force in place; and 4) NS2 adoption could be slower than expected, we are assuming a 15% discount rate; the high end of the industry range of 12-15% in both price-sales multiple analysis and price-earnings multiple analysis. Hence, considering that the company is pursuing orphan indications that have a longer market life, we are assuming an 8x multiple, higher-end of the industry average 6-8x, to our 2025 sales and an 15% discount rate in our price-sales multiple analysis, we arrive at a

fair value of $13.13 per diluted share. In our price-earnings multiple analysis, considering that we are uncertain of some of the pushes and pulls on operating margins at present time, we are assuming conservatively a 25x multiple, low end of the industry average 25-28x, to our 2025 EPS estimate of $1.77 and a 15% discount rate, we obtain a fair value of $13.07 per diluted share. Averaging the results of these two methods, we obtain a 12-month price target of $13.10, which we round to $13.00 per share.

Exhibit 12: Price-Sales Multiple and Price-Earnings Multiple Analyses

Price-Sales multiple

Discount factor	7	8	9
11%	16.36	18.70	21.04
13%	13.69	15.64	17.60
15%	11.49	13.13	14.77
17%	9.67	11.05	12.43
19%	8.16	9.32	10.49

Price-Earnings multiple

Discount factor	24	25	26
11%	17.88	18.63	19.37
13%	14.96	15.58	16.20
15%	12.55	13.07	13.60
17%	10.56	11.00	11.44
19%	8.92	9.29	9.66

Source: H.C. Wainwright Estimates

Summary of Executives

Todd C. Brady, MD Ph.D., President, Chief Executive Officer and Director

Dr. Brady has more than 18 years of pharmaceutical clinical and business development experience. Prior to joining Aldeyra as president and CEO in 2011, Dr. Brady was an entrepreneur-in-residence and principal at Domain Associates, where he led institutional financing in numerous biotechnology companies from 2004 to 2013. Prior to joining Domain, Dr. Brady was a co-founder and CEO of Phenome Sciences, a biotechnology firm he merged with Xanthus Pharmaceuticals (acquired by Antisoma), where he was later executive vice president of Strategic Development and Planning. Dr. Brady also worked as head of business development and medical director at Aderis Pharmaceuticals (acquired by Schwarz Pharma, now part of UCB[UCB.BR; not rated]). While at Xanthus and Aderis, Dr. Brady was a medical consultant on numerous pre-clinical programs and clinical programs in Phases 1 through 4, including Neupro®, a drug now marketed for Parkinson's Disease. Dr. Brady holds an M.D. from Duke University Medical School, a Ph.D. from Duke University Graduate School, and an A.B. from Dartmouth College.

Stephen Tulipano, CPA, Chief Operating Officer

Mr. Tulipano has more than 27 years of accounting and financial experience, of which 12 years were focused on the pharmaceutical industry. Prior to joining Aldeyra as Chief Financial Officer in June 2014, Mr. Tulipano held positions at Three Tulips Inc., an accounting and management advisory services firm, where he provided full-time accounting services and financial management counsel. Prior to joining Three Tulips Inc., he served as Chief Financial Officer of Javelin Pharmaceuticals where he helped lead the company through its acquisition by Hospira in 2010. Mr. Tulipano also served as the Director of Corporate Accounting at Biogen Idec, and has held several accounting roles both within companies and accounting firms. Mr. Tulipano holds a B.S. in Business Administration and Accounting from Salem State College and an M.B.A. in Finance from Suffolk University. He is also a Certified Public Account.

Scott L. Young, Chief Operating Officer

Mr. Young has more than 25 years of pharmaceutical pre-clinical and clinical development experience. Prior to joining Aldeyra Therapeutics in December 2011 as chief operating officer (COO), he was the COO at Link Medicine, Inc., a biotechnology company developing novel pharmaceuticals to treat neurodegenerative diseases such as Alzheimer's disease and Parkinson's disease. While at Link Medicine, Mr. Young and colleagues successfully raised more than $40 million in financing, advanced the lead program to clinical development, and subsequently out-licensed the technology to AstraZeneca (AZN; not rated). Mr. Young also served as COO for OXiGENE, Inc. (OXGN; Buy), a publicly traded NASDAQ oncology therapeutics development company, where, during the eight years of his tenure, he was instrumental in advancing a pharmaceutical candidate from laboratory testing into Phase 3 clinical trials and led the development of a compound in an orphan ophthalmology indication. Mr. Young has also held positions in clinical and regulatory affairs, GMP manufacturing operations and R&D and process development at Genzyme Corporation, RepliGen Corporation (RGEN; not rated) and Genetics Institute (now Pfizer [PFE; not rated]). He holds a B.S. in biochemistry from the University of Massachusetts, Amherst.

Aldeyra Historical Income Statement and Financial Projections

$ ('000) Except Per Share Data	FY 2013A	FY 2014E	FY 2015E	FY 2016E	FY 2017E	FY 2018E	FY 2019E	FY 2020E	FY 2021E	FY 2022E	FY 2023E	FY 2024E	FY 2025E
Revenues	-	-	-	-	**1,175**	**6,753**	**14,321**	**21,396**	**31,464**	**41,672**	**53,989**	**66,476**	**77,144**
COGS	-	-	-	-	76	405	788	1,070	1,573	2,084	2,699	3,324	3,857
Gross Income	-	-	-	-	**1,098**	**6,348**	**13,534**	**20,326**	**29,890**	**39,589**	**51,289**	**63,153**	**73,287**
Research & Development	1,542	3,739	6,255	6,621	6,890	7,169	7,460	7,763	7,685	7,382	9,730	12,024	14,011
G&A	2,135	3,521	4,117	4,458	4,895	5,298	5,735	6,208	8,953	11,821	15,353	18,884	21,857
Operating Income (Loss)	**(3,676)**	**(7,260)**	**(10,372)**	**(11,078)**	**(10,686)**	**(6,119)**	**338**	**6,355**	**13,253**	**20,385**	**26,206**	**32,244**	**37,418**
Pretax Income (Loss)	**1,110**	**(9,774)**	**(10,539)**	**(11,245)**	**(10,853)**	**(6,286)**	**172**	**6,188**	**13,086**	**20,219**	**26,040**	**32,077**	**37,251**
Income Taxes (Benefit)	-	-	-	-	-	-	-	2,104	4,449	6,874	8,853	10,906	12,665
Net Income	**1,110**	**(9,774)**	**(10,539)**	**(11,245)**	**(10,853)**	**(6,286)**	**172**	**4,084**	**8,637**	**13,344**	**17,186**	**21,171**	**24,586**
Basic EPS	**3.49**	**(2.25)**	**(1.78)**	**(1.78)**	**(1.62)**	**(0.89)**	**0.02**	**0.51**	**1.04**	**1.53**	**1.88**	**2.22**	**2.48**
Diluted EPS	**0.45**	**(6.35)**	**(1.78)**	**(1.78)**	**(1.62)**	**(0.89)**	**(0.06)**	**0.34**	**0.70**	**1.05**	**1.31**	**1.57**	**1.77**
Basic Shares Outstanding	318	3,824	5,915	6,315	6,715	7,115	7,515	7,915	8,315	8,715	9,115	9,515	9,915
Diluted Shares Outstanding	857	3,861	5,915	6,315	6,715	7,115	9,494	11,872	12,272	12,672	13,072	13,472	13,872

Source: Company Reports and H.C. Wainwright Estimates

Aldeyra Historical Balance Sheet

S ('000)	Q1 2014A	Q2 2014A	Q3 2014A
Assets			
Cash & cash equivalents	2,146	11,536	10,142
Preferred stock issuance receivable	-	-	-
Prepaid expenses and other	10	270	203
Total Current Assets	**2,155**	**11,805**	**10,345**
Deferred offering costs	735	-	-
Total Assets	**2,891**	**11,805**	**10,351**
Liabilities & Shareholders' Equity			
Accounts payable	166	556	482
Convertible notes payable	155	-	-
Accrued interest on convertible notes payable	5	-	-
Total Current Liabilities	**692**	**1,175**	**846**
Credit facility, net of current portion of debt	972	816	1,241
Accrued deffered offering costs	604	-	-
Total Liabilities	**4,027**	**1,990**	**2,087**
Preferred stock (series A and B)	38,509	-	-
Common stock	0	6	6
Additional paid-in capital	1,277	51,867	52,325
Accumulated deficit	(40,923)	(42,058)	(44,067)
Total Shareholders' Equity	**(39,646)**	**9,815**	**8,264**
Total Liabilities & Shareholders' Equity	**2,891**	**11,805**	**10,351**

Source: Company Reports and H.C. Wainwright Estimates

Aldeyra Historical Cash Flow Statement

S ('000)	Q1 2014A	Q2 2014A	Q3 2014A
Cash Flow Operating Activities			
Net Income/ (loss)	(13)	(5,330)	(2,009)
Adjustments to reconcile net loss	(415)	(4,195)	0
Net Income/ (loss)	**402**	**(1,135)**	**(2,009)**
Stock based compensation	366	748	458
Interest converted to preferred stock	-	-	-
President and CEO contriubuted services	-	-	-
Amortization of debt discount - non cash interest expense	88	33	18
Change in fair value of warrant liability, purchase right and w	(1,760)	(568)	-
Value provided in excess of issuance price of series B redeer	-	-	-
Depreciation	-	-	0
Change in assets and liabilities	(159)	27	322
Net cash used for operating activities	**(1,064)**	**(894)**	**(1,211)**
Investing Activities			
Acquisition of PPE	-	-	(6)
Purchase of intangible assets	-	-	-
Net cash used for investing activities	-	-	**(6)**
Financing Activities			
Proceeds from convertible notes payable - related parties	-	-	-
Proceeeds from issuance of common stock	-	10,232	(177)
Cash paid for deferred offering costs	(53)	53	-
Net Financing Cash Flow	**(53)**	**10,285**	**(177)**
Net change in cash and cash equivalents	(1,117)	9,390	(1,394)
Cash and cash equivalents - beginning of the period	3,262	2,146	11,536
Cash and cash equivalents - end of the period	**2,146**	**11,536**	**10,142**

Source: Company Reports and H.C. Wainwright Estimates

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Market Outperform (Buy): The common stock of the company is expected to outperform a passive index comprised of all the common stock of companies within the same sector.

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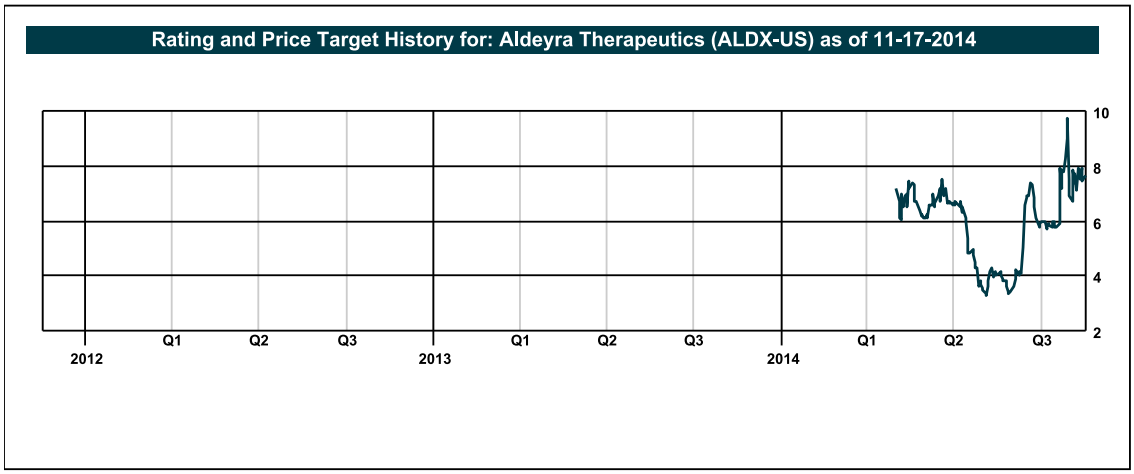

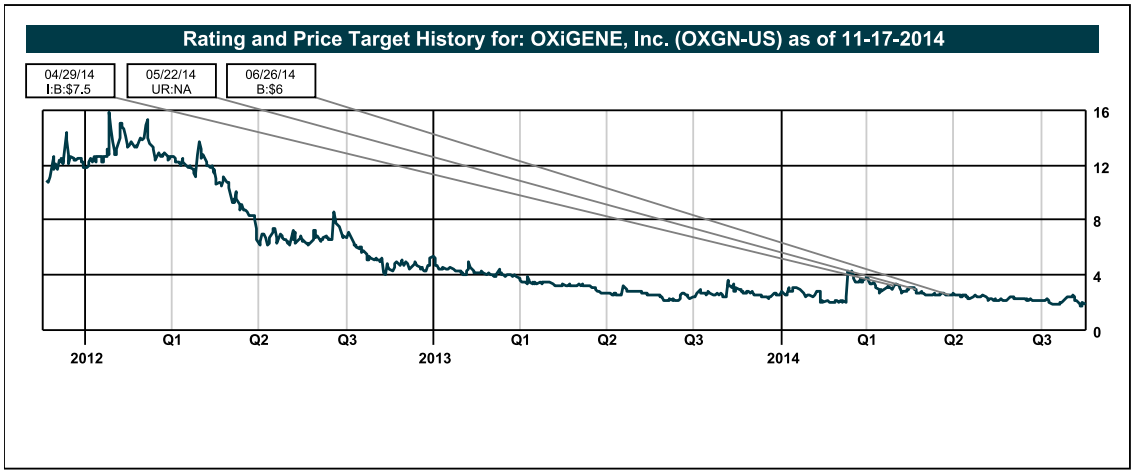

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Distribution of Ratings Table				
			IB Service/Past 12 Months	
Ratings	Count	Percent	Count	Percent
Buy	90	91.84%	37	41.11%
Neutral	7	7.14%	1	14.29%
Sell	0	0.00%	0	0.00%
Under Review	1	1.02%	0	0.00%
Total	98	100%	38	38.78%

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