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EQUITY RESEARCH
Company Report

January 26, 2015

Aldeyra Therapeutics (ALDX - $ 8.86)

Healthcare/Biotechnology

Novel Aldehyde Trapping Approach Could Potentially Afford New Treatment Modality in Multiple Diseases

Ticker:	**ALDX**
Rating:	**Buy**
Price Target:	**$ 30.00**

We are initiating coverage of Aldeyra Therapeutics with a Buy rating and a $30 12-month price target. ALDX affords a novel aldehyde trapping treatment that could potentially treat multiple inflammatory and several ultra-orphan indications associated with excess toxic free aldehyde or with direct impairments of free aldehyde regulating enzymes.

- **Aldehyde trapping is a novel therapeutic modality with the potential to treat multiple indications.** With increasing understanding and awareness of how excess toxic free aldehyde could damage the body via multiple mechanisms, such as inflammation or direct injury to the skin, aldehyde trapping is becoming a unique therapeutic modality that could enrich the armamentarium of anti-inflammation treatments. ALDX is the leading developer in aldehyde trapping.

- **Success in Sjögren-Larsson Syndrome (SLS) treatment could be a fast path for NS2to reach market possibly at more measured risk.** ALDX is developing NS2 cream as a potential treatment for SLS with Phase II/III study results potentially available in 3Q15. As an ultra-orphan indication and encouraging pre-clinical study results supporting skin symptom alleviation after direct removal of disease-causing aldehyde; possible upcoming clinical success in treating SLS could be a faster path for NS2 to reach market with the drug priced at a premium.

- **Potential clinical success of NS2 in acute anterior uveitis (AAU) could place aldehyde trapping as a potential treatment in other larger inflammatory disorders.** ALDX is also developing an NS2 eye drop as a potential treatment for AAU (an orphan indication) with data from the ongoing Phase II study expected in 4Q15. The AAU trial potentially could be a proof-of-concept (POC) clinical study not only for AAU but also for other inflammatory diseases. If successful, a robust outcome could further open opportunities for aldehyde trapping to treat other inflammatory diseases in addition to AAU – a scenario that substantially expands the value of aldehyde trapping.

- **Substantial upside remains at the current valuation.** With two leading orphan indication clinical developments in place, we believe NS2 has potential in taking a shorter time to market. Further, aldehyde trapping could potentially address unmet needs of greater disease areas. Our $30 12-month price target is based on our peer comparable and probability adjusted DCF analyses for this under-exposed, and under-valued, story.

Trading Data:

Last Price (01/23/2015)	$ 8.86
52–Week High (10/29/2014)	$ 11.99
52–Week Low (8/4/2014)	$ 3.00
Market Cap. (MM)	$ 49
Shares Out. (MM)	6

Earnings Estimates: (per share)

(Dec)	1Q	2Q	3Q	4Q	FY	P/E
FY-15E	-0.37	-0.36	-0.37	-0.37	-1.54	N.A.
FY-14E	-0.04A	-1.43A	-0.36A	-0.37	-2.47	N.A.
FY-13A	-13.03	-5.47	2.76	18.47	3.49	N.A.
FY-12A	NA	NA	NA	NA	-124.44	N.A.

Source: Laidlaw & Company estimates

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Investment Thesis

Our $30 price target is supported by peer comparable and DCF analyses.

- **We are initiating coverage of Aldeyra Therapeutics (ALDX) with a Buy rating and a 12-month price target of $30.** Aldeyra Therapeutics is a mid-clinical stage biotechnology company focusing on the development of its proprietary aldehyde trapping medications for the treatment of diseases directly associated with impairment of free aldehyde regulating enzymes as well as inflammatory diseases that excess free aldehyde have played a critical part in pathogenesis.

Excess free aldehyde could modify cellular constitutes, such as lipid, protein or carbohydrate, leading to the formation of indigestible aggregates, which have been implicated in the progressions of autoimmune, inflammatory and other diseases.

- **Aldehyde trapping is a novel treatment platform with potential treating multiple indications.** Although the negative impacts of toxic free aldehyde toward the human body have not been broadly publicized and discussed; various recent studies have provided increasing insights regarding multiple disorders that might be mediated by inappropriately-regulated aldehyde. As a summary, excess free aldehyde could modify cellular constitutes, such as lipid, protein or carbohydrate, leading to the formation of indigestible aggregates, and many of which have been implicated in the progressions of autoimmune, inflammatory and other diseases. Aldehyde trapping is a proprietary small molecule therapy developed by Aldeyra Therapeutics that could trap free aldehyde rapidly and transport the complex to lysosome for an immediate elimination (within hours). ALDX's lead drug, NS2, is currently in two forms – cream and eye drop – as a potential treatment of Sjögren-Larsson Syndrome (SLS) and (non-infectious) acute anterior uveitis. We believe the company also is developing other methods of delivery, including orally administrated NS2, potentially as a systemic treatment for other relevant indications.

Sjögren-Larsson syndrome is an ultra-orphan genetic disease caused by mutations in an enzyme called fatty aldehyde dehydrogenase (FALDH), resulting in the accumulation of high levels of toxic aldehyde.

- **NS2 in Sjögren-Larsson Syndrome (SLS) treatment potentially could represent a shortest path to market.** Sjögren-Larsson syndrome is an ultra-orphan genetic disease caused by mutations in an enzyme called fatty aldehyde dehydrogenase (FALDH), resulting in the accumulation of high levels of toxic aldehyde. Major symptoms of SLS include dry, scaly skin (ichthyosis) which is generally dispersed over the whole body, as well as other neurological and ophthalmological disabilities. Prevalence of SLS in the U.S. is approximately > 1,000 patients with approximately similar patient size in Europe. ALDX conducted several pre-clinical studies demonstrated that NS2 could prevent the increase of aldehyde levels caused by dry conditions in skin tissue – a circumstance that recapitulates the pathological environment of SLS. ALDX filed an IND in late 4Q14, with commencement of patient recruitment expected in 1Q15 for a Phase II study that evaluates NS2 cream in SLS. It is a placebo control study (n=12 with treatment duration of eight weeks) with top-line results (visual rating of skin condition improvements are primary endpoints) potentially available in 3Q15 – a major catalyst for ALDX shares, in our opinion, given it

ALDX started Phase II study that evaluates NS2 cream in SLS with top-line results potentially available in 3Q15 – a major catalyst for ALDX shares, in our opinion.

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would be the first clinical proof-of-concept study outcome. Based on a more conservative estimate of the clinical development timeline, we believe potential NS2 in SLS approval could occur in 2017 if trial outcomes are positive. We estimate potential annual peak sales of NS2 in SLS in the U.S. could exceed $120MM with global sales exceeding $200MM. In addition to SLS, our due diligence suggested other rare orphan free aldehyde associated diseases, such as succinic semialdehyde dehydrogenase deficiency (SSADH deficiency), could potentially be treated by NS2 or other aldehyde trapping drugs. If such a scenario can be realized, we believe the market potential for NS2 in the ultra-orphan arena could further expand beyond SLS.

Other rare orphan free aldehyde associated diseases, such as succinic semialdehyde dehydrogenase deficiency (SSADH deficiency), could potentially be treated by NS2 or other aldehyde trapping drugs.

- **The NS2 in non-infectious acute anterior uveitis Phase II study, if successful, could potentially expand the utility of the aldehyde trapping modality in other inflammatory disorders with substantially greater market potential.** In addition to SLS, ALDX is also developing the NS2 eye drop as a potential treatment in non-infectious acute anterior uveitis. Uveitis is an inflammation of the uveal structures, and can be classified into anterior, intermediate and posterior uveitis. Anterior uveitis (AU) refers to inflammation of the iris and ciliary body. AU can be further categorized into acute and chronic. The current treatments for acute AU (AAU) are relatively non-specific; mainly with topical corticosteroids which incur serious side effects, such as increase the propensity for the formation of cataracts and glaucoma. Further, there are high recurrence rate in AAU with 66% patients developed at least one and 36% with three or more relapses. Prevalence for AU in the U.S. is estimated to be 38,000 incidences per year; while AAU is approximately 26,000 – also an orphan disease. ALDX conducted pre-clinical studies demonstrated that NS2 could prevent the increase of aldehyde levels caused by dry conditions in ocular tissue. Further, a Phase I safety study evaluating the NS2 eye drop in 48 healthy volunteers demonstrated that the drug was well tolerated in all treatment groups; and without plasma exposure detected. Supported by encouraging results, ALDX recently (4Q14) filed an IND for a Phase II study evaluating the NS2 eye drop in non-infectious acute anterior uveitis patients. Patient recruitment commencement is expected in 1Q15 and top-line results potentially available in 4Q15. We believe the data reporting of NS2 in the AAU Phase II trial could be another major catalyst for ALDX shares, given 1) it would be the first clinical proof-of-concept study outcome of this indication; and 2) it also would be the first clinical proof-of-concept trial potentially to illustrate the anti-inflammatory activities of NS2, possibly expanding it as a treatment for several other inflammatory disorders. In addition, pre-clinical data of NS2 in murine inflammation models will be reported at the American Academy of Allergy Asthma & Immunology Annual Meeting (AAAAI) in February 24, 2015; and we believe this would be another opportunity for investor to gain more insights on anti-inflammatory activities of NS2.

A Phase II study evaluating NS2 eye drop in non-infectious acute anterior uveitis patients is underway with top-line results potentially be available in 4Q15.

The AAU Phase II trial would be the first clinical proof-of-concept study for 1) acute anterior uveitis and 2) potentially to illustrate anti-inflammatory activities of NS2, possibly expanding it as a treatment for several other inflammatory disorders.

- **Valuation is favorable.** We believe ALDX shares are undervalued, based on the substantial near term market potential that NS2 could be realized from two orphan indication as well as the possibilities that NS2 could be used in treating broader inflammatory and other indications with potentially much large market potential. With several critical

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catalyst events, some in 2015 and beyond; we believe ALDX shares could materially appreciate should the outcome of these events be positive. Accordingly, our $30 price target is supported by peer comparable and DCF analyses. We are recommending ALDX shares to long-term oriented investors with high risk tolerance.

Company Description

Aldeyra Therapeutics is a mid-clinical stage biotech company focused on exploring its proprietary aldehyde trapping platform as a novel treatment modality for inflammatory and other disorders with substantial unmet medical needs. Aldehyde trapping is based on the concept that by removing excess free aldehydes accumulated in the body, patients with inflammatory or other free aldehyde-causing disorders could be treated. The two initial indications addressed by the company's lead product, NS2 are Sjögren-Larsson Syndrome (SLS) and acute anterior uveitis. The company filed two INDs in late 4Q14 for Phase II studies in SLS and in non-infectious acute anterior uveitis with data potentially available in 2H15. Both indications are orphan indications with potential of shorter time to market and the option of premium pricing. Should the acute anterior uveitis clinical study successful, NS2 could potentially have therapeutic utility in several other inflammatory diseases.

Anticipated milestones in 2015 and beyond and pipeline

Product	Indication	Event	Timing	Importance
NS2 cream	Sjögren-Larsson Syndrome	Potentially enroll first patient for Phase II study	**1Q15**	***
		Potentially report Phase II study top-line results	**3Q15**	****
		Potentially report pre-clinical results	**2015**	***
NS2 eyedrop	Acute anterior uveitis	Potentially enroll first patient for Phase II study	**1Q15**	***
		Presentation of pre-clinical anti-inflammation data at the 2015 American Academy of Allergy Asthma & Immunology Annual Meeting	**Feb. 24, 2015**	***
		Potentially report Phase II study top-line results	**4Q15**	****

***** / ***** Major catalyst event that could impact share price very significantly while *** event is more informative*

Source: Laidlaw & Company and company presentation.

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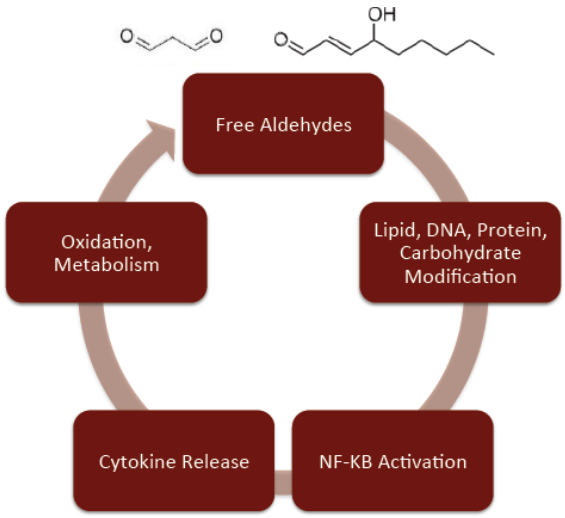
Aldehyde Trapping and NS2

What are fatty aldehydes and their implication in disease progression

Mechanistically, excess free aldehyde could bind to cellular constituents, such as protein, lipid, DNA and carbohydrate to form indigestible adducts and aggregates.

Medium to long-chain aliphatic aldehydes are organic metabolic products derived from metabolism of corresponding fatty acids. Since the majority of aldehydes 1) are rapidly metabolized with only relatively minor accumulation in the body in healthy individual; and 2) are not essential components of cellular membranes; the research in this area has been relatively limited so far. The free fatty aldehydes are formed endogenously by lipid peroxidation (LPO) or carbohydrate metabolism. Under the circumstances when high level of free aldehyde accumulates in the body, it could cause illness such as disorders associated with inflammation. Mechanistically, excess free aldehyde could bind to cellular constituents, such as protein, lipid, DNA and carbohydrate to form indigestible adducts and aggregates. As such, these abnormal macromolecules could act as inflammation cascade triggering mediators to potentially cause various inflammatory diseases (Figure 1). Under normal circumstances, excess free aldehyde can be removed by enzymes, called aldehyde dehydrogenases, to detoxify aldehydes. One example that demonstrates most directly the causal relationship between excess free aldehyde and a disease is in Sjögren-Larsson Syndrome (SLS), of which patients carry various mutations in their fatty aldehyde dehydrogenase (FALDH) gene, resulting in accumulation of free aldehyde with symptom manifestations of dry, scaly skin (ichthyosis); neurological (such as leukoencephalopathy) and eye (like myopia) symptoms.

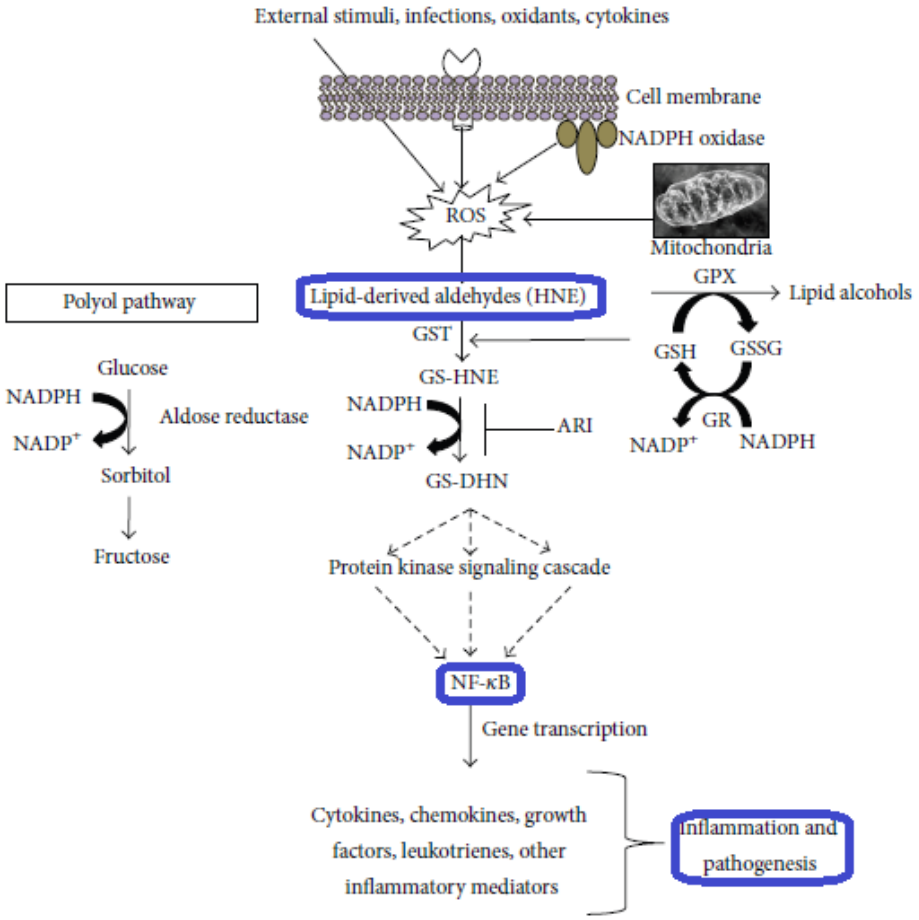
Figure 1: Aldehyde and relationship with various disorders



Source: Company presentation

The connection between free aldehyde and anti-inflammatory effects. Although the number of scientific and clinical studies on the relationship between free aldehydes and inflammatory diseases remains modest, a recently published review by Yadav and Ramana affords solid support for the potential use of the aldehyde trapping approach for treating inflammatory disorders[1]. Specifically, toxic lipid aldehyde species generated from oxidative stress could activate redox sensitive transcription factors, such as NF-κB and AP-1, which in turn stimulate the expression of an array of inflammatory cytokines and chemokines genes (Figure 2), potentially leading to various inflammatory disorders.

Figure 2:Regulation of lipid aldehyde-induced inflammatory signalling



Source: Yadav, U.S.C. and Ramana, K.V, Oxidative Medicine and Cellular Longevity, 2013

The basic function of aldehyde trapping is to remove excess free aldehydes from the sites of disease in order to treat the disorder caused by toxic lipid aldehyde species.

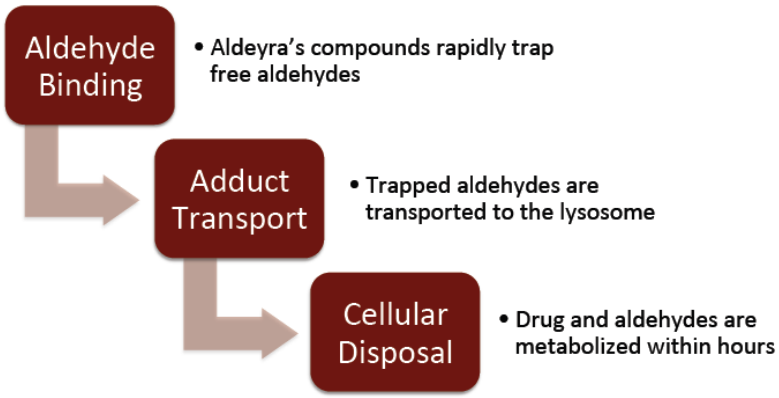
Aldehyde trapping's mechanism of action. The basic function of aldehyde trapping is to remove excess free aldehydes from the sites of disease in order to treat the disorder caused by toxic lipid aldehyde species. The company's lead product, NS2, in topical and eye drop formats, currently is undergoing clinical studies in two different indications. In addition, an orally administrated NS2 is also in development. The principal mechanism of action of NS2 in trapping

[1] *Yadav, U.S.C. and Ramana, K.V, Oxidative Medicine and Cellular Longevity, 2013*

Free aldehydes could bind more rapidly to NS2 … resulting in the

aldehyde is that the free aldehydes could bind more rapidly to NS2 than binding to cellular constituents, and resulting in the formation of an irreversible complex, called NS2-aldehyde adducts. Such adducts remain stable intracellularly and only can be degraded after they have been transported to cellular lysosomes. Inside the lysosome, this adduct could be degraded within hours. Given that it can bind free aldehydes irreversibly and rapidly transport the aldehydes to lysosomes for degradation, NS2 therefore has the potential to lower aldehyde levels substantially (Figure 3).

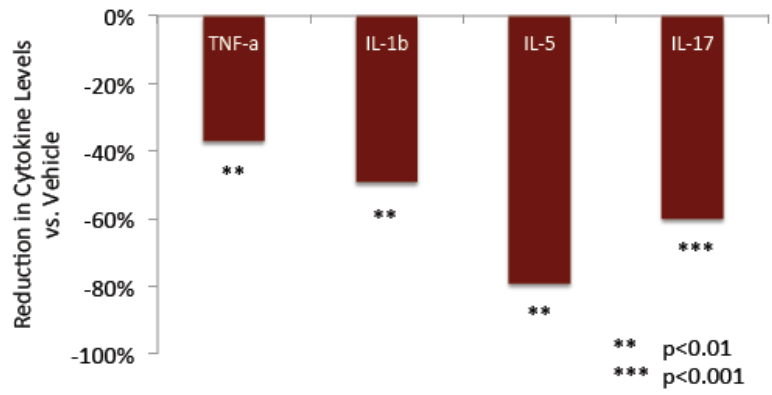
Figure 3: The overall scheme of aldehyde trapping



Source: Company presentation

Given the causal relationship between free aldehydes and inflammatory disorders, ALDX has demonstrated pre-clinically that NS2 has the capability to reduce multiple pro-inflammatory cytokines as a potential treatment for inflammatory disorders. In this study, mice were treated either with NS2 or placebo for 30 minutes prior to endotoxin exposure for the stimulation of multiple pro-inflammatory cytokines. Cytokine profiles were measured two hours after endotoxin exposure and the results indicated that NS2 could reduce several major pro-inflammatory cytokines, such as TNF-α, IL-1β, IL-5 and Il-17 (Figure 4).

Figure 4: NS2 demonstrated reduction of multiple pro-inflammatory cytokine from pre-clincial studies



Source: Company presentation

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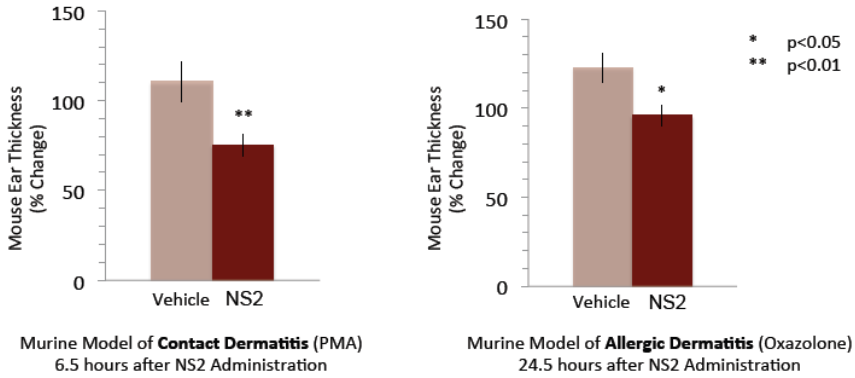
ALDX also conducted several pre-clinical studies evaluating NS2 in various inflammatory disease models. Potential indications tested in these models include dermal inflammations, such as contact dermatitis and allergic dermatitis, oral mucositis, dry eye and dry skin conditions, and SLS mutant analyses. More details regarding the latter two analyses will be presented at the ensuing session of relevant indications.

In the mouse model for contact dermatitis, PMA (phorbol 12-myristate 13-acetate) was applied topically to the surface of the ear to induce edema. NS2 treatment has demonstrated reduction of inflammation induced swelling (measured by ear thickness) vs. vehicle (Figure 5, left) with statistical significance 6.5 hours after treatment. For testing allergic dermatitis, mice were initially sensitized with oxazolone and subsequently challenged with oxazolone at the surface of the ears to induce edema. NS2 treatment has demonstrated reduction of inflammation induced swelling (measured by ear thickness) vs. vehicle with statistical significance 24.5 hours after treatment (Figure 5, right).

Figure 5: NS2 demonstrated reduction of inflammation in different derma pre-clincial models



Source: Company presentation

In a hamster cheek pouch radiation-induced oral mucositis model, NS2 treatment has demonstrated an increase of lesion healing based on the mucositis score (Figure 6, left) with statistical significance; and reduction of scarring (based on the fibrosis score) with a positive trend vs. placebo (Figure 6, right).

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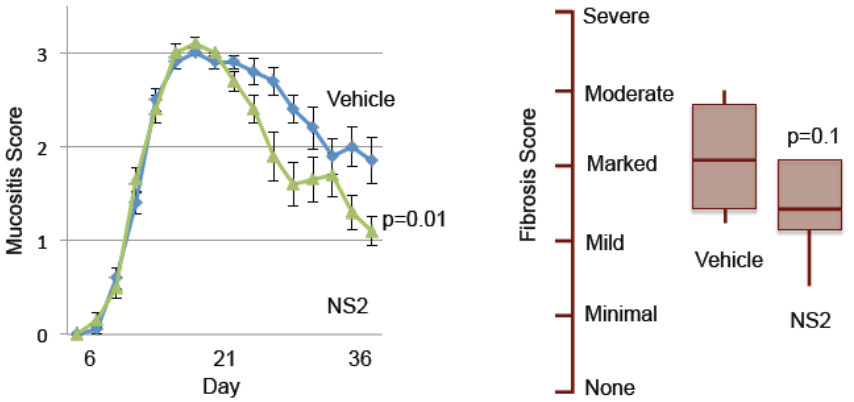
Figure 6: NS2 demonstrated speedy healing and reduced scarring in different pre-clincial model



Source: Company presentation

Together, these pre-clinical studies have demonstrated that NS2 could potentially have therapeutic impact on several dermatological disorders.

Solid intellectual property support for NS2. NS2 is covered in the U.S. by one issued patent (composition of matter) and four global pending patent applications. ALDX reported that IP protection could extend to late 2020s worldwide; and into 2033 in the U.S. under the Hatch-Waxman Act.

NS2 in Sjögren-Larsson Syndrome (SLS) Development Could be a Faster Path to Reach the Market

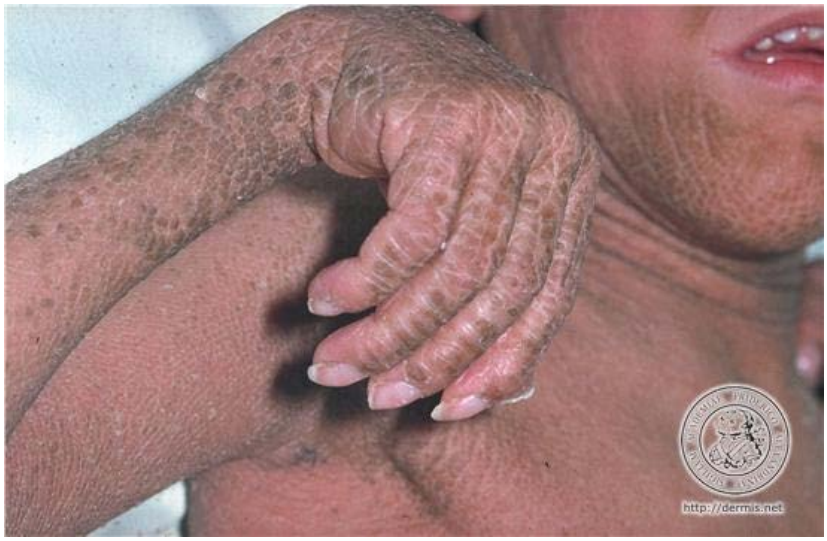
Sjögren-Larsson Syndrome (SLS) is an orphan indication with substantial un-met medical needs.

Sjögren-Larsson syndrome (SLS) is a genetic disease caused by mutations in an enzyme, fatty aldehyde dehydrogenase (FALDH), resulting in accumulation of high level of toxic aldehyde.

Sjögren-Larsson syndrome is a genetic disease caused by mutations in an enzyme, fatty aldehyde dehydrogenase (FALDH), resulting in accumulation of high level of toxic aldehyde. On the dermatological side, excess aldehydes lead to the generation of aldehyde-mediated damage of lipids that contribute to the formation of the dermal moisture barrier and increased trans-epidermal water losses. Major symptoms of SLS include dry, scaly skin (ichthyosis) which generally dispersed over the whole body. Major manifestations of ichthyosis include plaques and scales, thickening, redness, inflammation and pruritus (itching) (Figure 7). In addition, SLS also causes neurological problems, such as leukoencephalopathy as well as other disabilities, including intellectual disability, speech difficulties (dysarthria) and delayed speech. Ophthalmological disabilities include impaired retina leading to symptoms like myopia or increased light sensitivity or photophobia. Many symptoms are apparent by early childhood and many affected infants tend to be born prematurely.

Major symptoms of SLS include dry, scaly skin (ichthyosis) which generally dispersed over the whole body. Major manifestations of ichthyosis include plaques and scales, thickening, redness, inflammation and pruritus (itching).

Figure 7: Ichthyosis in Sjögren-Larsson syndrome patient



Source: Dermatology information system (www.dermis.net)

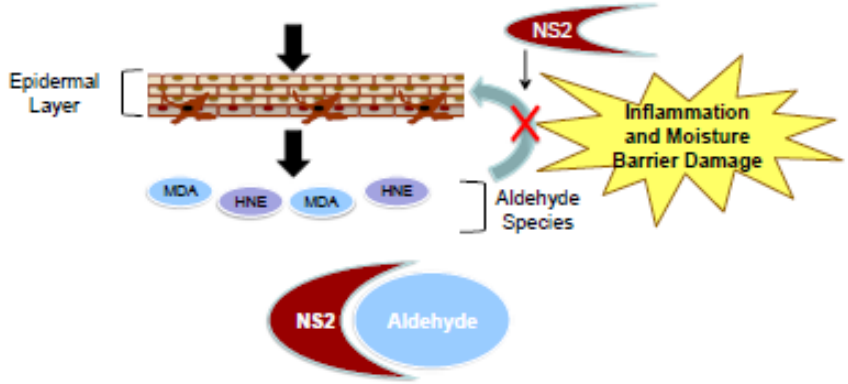
Currently, there is no approved treatment for SLS and current management of the disease is palliative in order to alleviate the symptoms. For example, ichthyosis is managed by applying topical moisturizing creams and keratolytic

agents, such as alpha-hydroxyacid (e.g. lactic acid, glycolic acid), salicylic acid, and urea. Systemic retinoids are used except in children due to potential adverse effects.

Sjögren-Larsson syndrome is a rare disease. It is estimated that the prevalence of SLS is 1 per 250,000 individuals and with an approximately > 1,000 patients in the U.S. We estimate the prevalence in Europe is similar to that of the U.S. with projected patient size is slightly larger than 1,200.

Given accumulated toxic free aldehydes are suggested to be the major pathophysiologic mechanisms causing symptom manifestations, NS2 could potentially treat SLS by removing excess free aldehydes (Figure 8).

Figure 8: Schematic presentation of how NS2 treat SLS

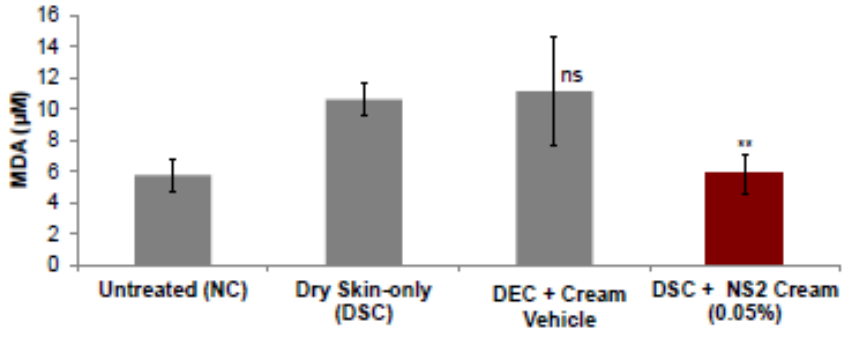


Source: Company presentation

In a pre-clinical study with reconstructed human epidermis under normal and dry skin conditions (DSC), NS2 (0.05% w/v) treatment has exhibited reduction of malondialdehyde (MDA) vs. vehicle.

ALDX conducted several pre-clinical studies demonstrated that NS2 could prevent the increase of aldehyde levels caused by dry conditions in skin tissue – a condition similar to SLS. In a pre-clinical study with reconstructed human epidermis under normal and dry skin conditions (DSC), NS2 (0.05% w/v) treatment has exhibited reduction of malondialdehyde (MDA) vs. vehicle only or DSC measured by TBARS assay ($p<0.01$) (Figure 9).

Figure 9: NS2 demonstrated reduction of MDA in human epidermis model under dry skin condition (DSC).



Source: Young, S.L., et. al., Abstract LB793, Society for Investigative Dermatology 2014 Annual Meeting

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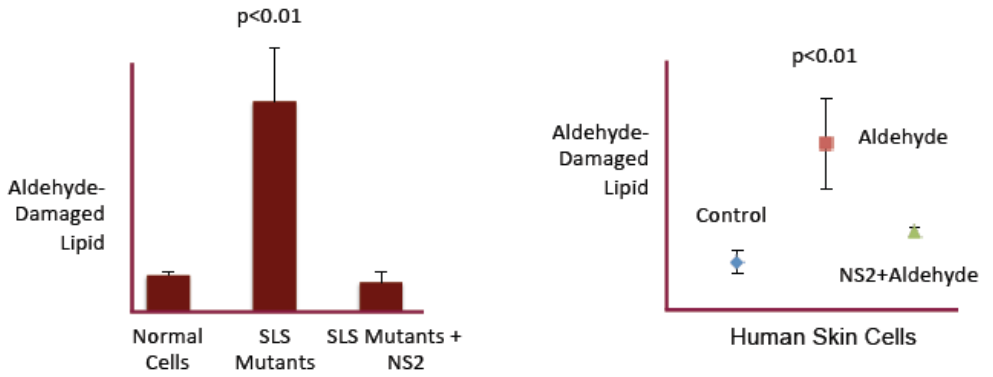
ALDX also demonstrated in an in vivo model that NS2 could reduce aldehyde damaged lipids in SLS mutant cells back to the level similar to that of normal cells.

Further, ALDX also demonstrated in an *in vivo* model that NS2 could reduce aldehyde damaged lipids in SLS mutant cells back to the level similar to that of normal cells (Figure 10, left). From a different analysis, after adding aldehydes to healthy human skin cells, cells have generated substantial level of aldehyde damaged lipids. By adding aldehyde and NS2 together, the level of aldehyde damaged lipids reduced back to the control level (Figure 10, right).

Figure 10: NS2 demonstrated reduction of aldehyde-damanged lipid in genetic mutated SLS human epidermis model (left) and in normal human skin cells (right).



Source: Company presentation

NS2 in SLS Phase II study is a 12-patient and eight week treatment trial. In addition to safety assessment, primary endpoint is visual rating of skin condition improvements. Top-line results expected in 3Q15.

Current development. Based on encouraging pre-clinical study results from dry skin and SLS mutant models and the recognition that major etiology of SLS is the lack of functional FALDH with excess of toxic free aldehyde, ALDX filed an IND in late 2014 with the expectation to start patient enrollment in 1Q15 in a Phase II study evaluating topical NS2 in Sjögren-Larsson syndrome patients. It is a placebo control study expected to enroll 12 patients with treatment duration of eight weeks. In addition to safety assessment, a primary endpoint is visual rating of skin condition improvements. Given SLS is an ultra-orphan disease with only a few treatment centers in the U.S., we believe the Phase II study would be carried out in one major clinical site with a significant portion of the patients seeking treatment. As such, we estimate the top-line results could be available in 3Q15 – a major catalyst for ALDX shares, in our opinion, given it would be the first clinical proof-of-concept study outcome. ALDX believes it is possible that Phase II study results, if robust, could potentially be sufficient for a discussion with the FDA for accelerated approval. We view this possibility to be a bullish scenario; and we more conservatively assume that the company might need to conduct another pivotal clinical study before filing for approval. As such, we assume NS2 in SLS approval could potentially be slated for late 2017, assuming it takes approximately one year for conducting second trial and a fast track approval process.

In addition, we view a potential success of developing orally administrated NS2 could be an important step for the advancement of a next generation SLS treatment. Given SLS patients also present neurological and ophthalmological disabilities in addition to dermatological disorders, an oral treatment agent could potentially provide systemic treatment effects beyond topical local therapy.

We assume NS2 in SLS launch in U.S. in 2017 and ex-U.S. in 2018 and together, we project the annual global peak sales could exceed $200MM.

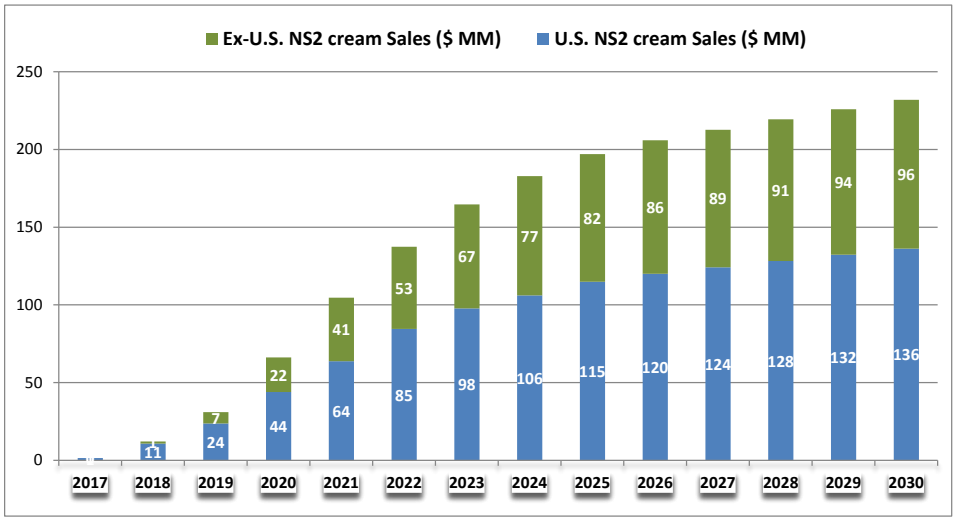
NS2 in SLS market model assumptions. We assume NS2 dermatological topical treatment could potentially receive approval in late 2017 after a positive Phase III pivotal study with fast track regulatory approval designation. We model a $140k annual treatment costs per patients in the U.S., which is in-line with the price range of many ultra-orphan medications and Targretin gel (currently sold by Valeant Pharmaceuticals), a topical product for cutaneous T cell lymphoma, which is also a rare dermatologic disease. We assume product launch in ex-U.S. could start in 2018 and together, we project the annual global peak sales could exceed $200MM.

Figure 11a: NS2 in Sjögren-Larsson Syndrome (SLS) revenue model

NS2 in Sjögren-Larsson Syndrome (SLS) Revenue Model	2017	2018	2019	2020	2021	2022	2023	2024	2025	2026	2027	2028	2029	2030
Total U.S. Sjögren-Larsson Syndrome prevalence	1,066	1,071	1,077	1,082	1,087	1,093	1,098	1,104	1,109	1,115	1,120	1,126	1,132	1,137
% treated by NS2	1%	7%	15%	27%	38%	49%	55%	58%	61%	62%	62%	62%	63%	63%
SLS patients treated with NS2	11	75	161	292	413	535	604	640	677	690	697	703	707	711
NS2 cream Price ($)	140,000	143,416	146,915	150,500	154,172	157,934	161,788	165,735	169,779	173,922	178,166	182,513	186,966	191,528
U.S. NS2 cream Sales ($ MM)	**1**	**11**	**24**	**44**	**64**	**85**	**98**	**106**	**115**	**120**	**124**	**128**	**132**	**136**
Total ex-U.S. Sjögren-Larsson Syndrome prevalence	1,218	1,224	1,230	1,236	1,243	1,249	1,255	1,261	1,268	1,274	1,280	1,287	1,293	1,300
% treated by NS2	0%	1%	5%	15%	27%	34%	42%	47%	49%	50%	50%	51%	51%	51%
SLS patients treated with NS2		12	62	185	336	425	527	593	621	637	644	650	654	656
NS2 cream Price ($)		115,020	117,320	119,666	122,060	124,501	126,991	129,531	132,121	134,764	137,459	140,208	143,012	145,873
Ex-U.S. NS2 cream Sales ($ MM)	**0**	**1**	**7**	**22**	**41**	**53**	**67**	**77**	**82**	**86**	**89**	**91**	**94**	**96**
Total NS2 in SLS Sales ($ MM)	**1**	**12**	**31**	**66**	**105**	**137**	**165**	**183**	**197**	**206**	**213**	**219**	**226**	**232**

Source: Laidlaw & Company estimates

Figure 11b: NS2 in Sjögren-Larsson Syndrome (SLS) revenue model



Source: Laidlaw & Company estimates

In addition to SLS, our due diligence suggested that NS2 or other aldehyde trapping drugs could potentially be used to treat another ultra-orphan indication – succinic semialdehyde dehydrogenase (SSADH) deficiency. Due to the lack of the SSADH enzyme, which converts inhibitory neurotransmitter γ-aminobutyric acid (GABA) to succinic acid – a molecule that can then be

utilized for energy production, an abnormal level of succinic semialdehyde (a last intermediate) is accumulated in the body. Consequently, an alternative metabolic pathway would take over and converts GABA to 4-hydroxybutyric acid or GHB (γ-hydroxybutyric acid) by gamma-hydroxybutyric dehydrogenase. GHB is believed to cause a wide range of effects within the nervous system. SSADH deficiency is a more recently identified (1981) inherited recessive disorder. Neurological manifestations of SSADH deficiency include developmental delay, seizures, hypotonia, mental retardation, and hyporeflexia. Patient size for SSADH deficiency is rather small as it is estimated that the disease inflicts on 350 to 450 patients; although we believe this could be an under-diagnosed disorder.

Given SSADH deficiency affects multiple areas of the central nerve system; we believe an orally administrated drug with systemic therapeutic effect would be more appropriate to treat patients.

Given the accumulation of succinic semialdehyde is the key pathological event leading to the formation of GHB, potential removal succinic semialdehyde by an aldehyde trapping drug, such as NS2 or other derivatives, could be a potential treatment of SSADH deficiency. Given that this disorder affects multiple areas of the central nerve system; we believe an orally administrated drug with systemic therapeutic effect would be more appropriate to treat patients. Our discussions with management indicated that the company is developing an orally administrated NS2. If such scenario can be realized, we believe the market potential for NS2 or other derivatives in the ultra-orphan arena could further expand beyond SLS.

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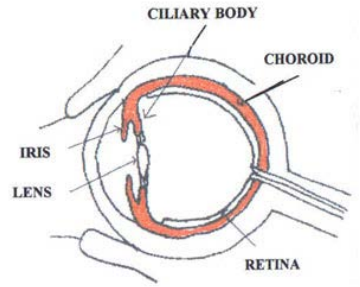
NS2 in Non-infectious Acute Anterior Uveitis Clinical Development, If Successful, Could Afford a Broader Treatment Potential in Inflammatory Disorders

Acute anterior uveitis is a rare inflammatory ocular disorder

Uveitis is inflammation of the uveal structures, which is comprised of choroid, ciliary body and iris (Figure 12).

Figure 12: Structure of uveal track of the eye

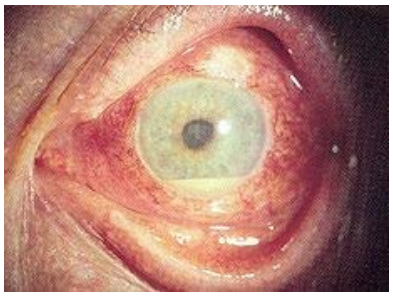


Source: www.the-eye-clinic.co.uk/common-disorders-of-the-eye-iritis-anterior-uveitis.htm

Anterior uveitis (AU) refers to inflammation of the iris and ciliary body. AU can be further categorized into acute (inflammation lasts less than six weeks) and chronic (inflammation lasts longer).

Uveitis is classified into anterior, intermediate and posterior uveitis according to the diseased location. Anterior uveitis (AU) refers to inflammation of the iris and ciliary body. AU can be further categorized into acute (inflammation lasts less than six weeks) and chronic (inflammation lasts longer). The etiologies of AU are multiple, which include systemic auto-immune/inflammatory diseases (such as rheumatoid arthritis or ankylosing spondylitis, and some of them are believed associated with HLA-B27, a specific genotype on chromosome 6), infection, external injury (trauma) and idiopathic, just names a few. Major symptoms of AU include pain, photophobia, circumlimbal redness, blurred vision and presences of anterior chamber cells (Figure 13).

Figure 13: Acute anterior uveitis



Source: Company presentation

Major complications associated with AU include cataracts, glaucoma, band keratopathy, and cystoid macular edema. Without appropriate treatment or if not properly managed, sight can be threatened.

The current treatments for AU are relatively non-specific; mainly with topical with corticosteroids and cycloplegic agents. Frequent topical corticosteroids use could lead to the formation of cataracts and glaucoma.

The current treatments for AU are relatively non-specific; mainly with topical corticosteroids and cycloplegic agents. Occasionally oral steroids or nonsteroidal anti-inflammatory drugs (NSAIDs) may be prescribed. One major shortcoming of topical corticosteroids are frequent use could lead to the formation of cataracts and glaucoma.

The recurrence rate of having more episodes of acute anterior uveitis is high. According to analysis by Natkunarajah[2] and colleagues, 66% of acute anterior uveitis patients developed at least one relapse; while 36% developed three or more relapses. Median interval between initial disease onset and the first relapse was 24 months; while median interval between the first and second relapse was 14 months.

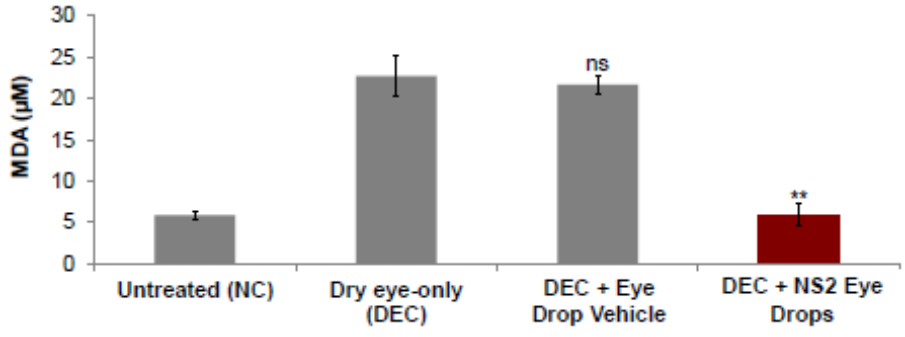
For anterior uveitis, prevalence in the U.S. occurs in approximately 38,000 incidences per year.

It has estimated that uveitis prevalence in the U.S. is approximately 38 per 100,000, or estimated 121,000 incidences per year. For anterior uveitis, prevalence in the U.S. occurs in approximately 8-12 per 100,000[3], or an estimated 38,000 incidences per year. The highest uveitis incidences occur in individual between 20-50 years old. We estimate the incidences of acute anterior uveitis are approximately 26,400 per year.

In a pre-clinical study with reconstructed human cornea-like tissues under normal and dry skin conditions (DEC), NS2 (0.05% w/v) treatment has exhibited reduction of malondialdehyde (MDA) vs. vehicle only.

ALDX conducted pre-clinical studies demonstrating that NS2 could prevent the aldehyde level increases caused by dry conditions in ocular tissue. In a pre-clinical study with reconstructed human cornea-like tissues under normal and dry skin conditions (DEC), NS2 (0.05% w/v) treatment has exhibited reduction of malondialdehyde (MDA) vs. vehicle only or DEC measured by TBARS assay ($p<0.01$) (Figure 14). In addition, a recent publication also indicated that serum MDA level [and total oxidant capacity (TOC), oxidative stress index (OSI)] of acute anterior uveitis patients were higher than that of healthy individuals[4].

Figure 14: NS2 demonstrated reduction of MDA in human ocular model under dry condition.



Source: Young, S.L., et. al., Abstract LB793, Society for Investigative Dermatology 2014 Annual Meeting

[2] *M Natkunarajah, M. et al., Br J Ophthalmol. 2007; 91:330–334*
[3] *Rothova A., e. al., Am J Ophthalmol 1987; 103:137-45*
[4] *Turk, A., et al., Ocul Immunol Inflamm. 2014 (22): 127-132*

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Positive NS2 eye drop Phase I study outcome. ALDX conducted a Phase I study evaluating the NS2 eye drop in 48 healthy volunteers for safety. Two dosings have been analyzed: 1) single day 0.25% and 0.5% bid & qid; and 2) single day 0.25% and 0.5% qid. The study indicated that NS2 eye drops were well tolerated in all treatment groups; and without plasma exposure detected.

NS2 in AAU Phase II study expects patient recruitment to start in 1Q15 with top-line results expected in 4Q15. The study compares NS2 against topical ophthalmic steroid and NS2 /steroid combination. Primary endpoints are changes of inflammatory cells of the anterior chamber of the eye as a measurement of change of level of intraocular inflammation; and examination of symptoms.

Current developments. ALDX recently (4Q14) filed an IND to start a Phase II study evaluating NS2 eye drop in acute anterior uveitis patients with patient recruitment commencement expected in 1Q15. It is an active control study expects to enroll 45 patients with eight week treatment. Although the company has not provided greater details, we believe the study is designed to compare NS2 against topical ophthalmic steroid (prednisolone acetate) and a combination of NS2 and steroid under a 1:1:1 randomization. Primary endpoints are changes of inflammatory cells of the anterior chamber of the eye as a measurement of change of level of intraocular inflammation; and examination of symptoms, like pain and visual acuity.

We estimate the top-line results could be available in 4Q15 – another major catalyst for ALDX shares, in our opinion, given 1) it would be the first clinical proof-of-concept study outcome of this indication; and 2) it also could be the first clinical proof-of-concept potentially to illustrate anti-inflammatory activities of NS2 with possibilities of treatment utilities in other inflammatory disorders.

Further, pre-clinical data of NS2 in murine models of inflammation will be reported at the American Academy of Allergy Asthma & Immunology Annual Meeting (AAAAI) in February 24, 2015; and we believe this would be another opportunity for investor to gain more insights on anti-inflammatory activities of NS2. The presentation entitled "The small molecule aldehyde trap NS2 exhibits potent anti-inflammatory activity in three murine models of inflammation" has been accepted in a late-breaking poster presentation to be presented February 24, 2015 at 9:45am at General Poster Session: 5211 (Poster: L11).

We assume NS2 eye drop treatment could potentially receive approval in 2019 after a positive second Phase III pivotal study with potential fast track regulatory approval designation. We forecast potential annual peak sales in the U.S. could exceed $300MM.

NS2 in non-infectious acute anterior uveitis market model assumptions. We assume NS2 eye drop treatment could potentially receive approval in 2019 after a positive second Phase III pivotal study with potential for a fast track regulatory approval designation. We model a $22.5k annual treatment cost per patient in the U.S., in which we assume NS2 would be prescribed to non-infectious acute anterior uveitis patients who failed steroid treatment or patients suffering from relapse. We assume NS2 in non-infectious acute anterior uveitis could potentially reach annual peak sales in the U.S. could exceed $300MM.

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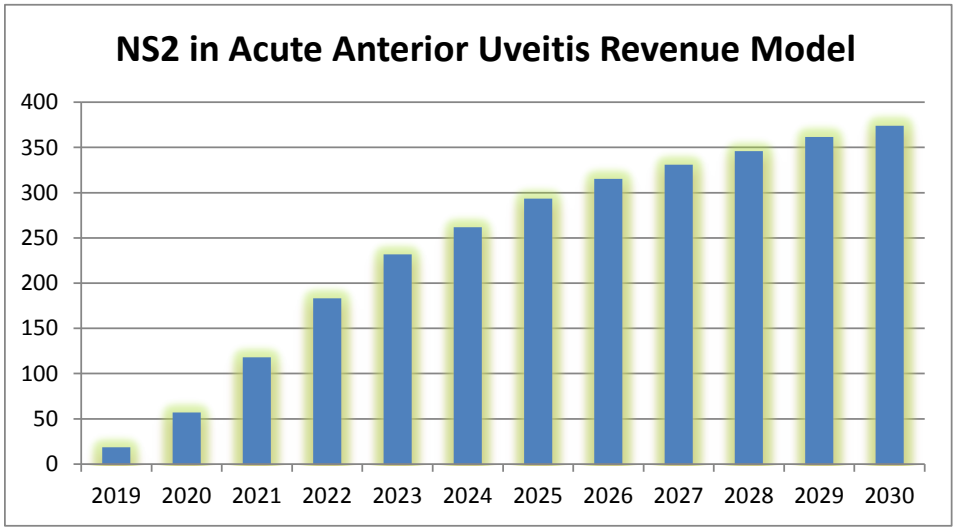
Figure 17a: NS2 in non-infectious acute anterior uveitis revenue model

NS2 in Acute Anterior Uveitis Revenue Model	2019	2020	2021	2022	2023	2024	2025	2026	2027	2028	2029	2030
Total U.S. uveitis incidences	67,933	68,748	69,573	70,408	71,253	72,108	72,974	73,776	74,588	75,408	76,238	77,076
Total U.S. anterior uveitis incidences	40,760	41,249	41,744	42,245	42,752	43,265	43,784	44,266	44,753	45,245	45,743	46,246
Total U.S. acute anterior uveitis incidences	26,494	26,812	27,134	27,459	27,789	28,122	28,460	28,773	29,089	29,409	29,733	30,060
% treated by NS2	2.0%	6.0%	12.0%	18.0%	22.0%	24.0%	26.0%	27.0%	27.4%	27.7%	28.0%	28.0%
Anterior uveitis patients treated with NS2	815	2,475	5,009	7,604	9,405	10,384	11,384	11,952	12,262	12,533	12,808	12,949
NS2 Eye Drop Price ($)	22,506	23,024	23,553	24,095	24,649	25,216	25,796	26,389	26,996	27,617	28,252	28,902
NS2 Eye Drop Sales ($ MM)	**18**	**57**	**118**	**183**	**232**	**262**	**294**	**315**	**331**	**346**	**362**	**374**

Source: Laidlaw & Company estimates

Figure 17b: NS2 in non-infectious acute anterior uveitis revenue model



Source: Laidlaw & Company estimates

Financial projections and valuation

With the recent equity offerings (January 15 and 20, 2015) of ~$9.8MM, we estimate the company has cash of ~$18MM (pro forma) based on recently reported cash and cash equivalent of approximately $10MM as of end of 3Q14. In addition, we estimate the company could potentially receive more cash should investors excise their warrants, potentially exceed $10MM. Together, we believe the cash should support the company's operations deep into 2016, by our estimate.

Our probability-adjusted DCF analysis suggested a one-year target value for ALDX of $30.07 based on cash flow until 2025 with an assumed terminal value multiple of three and a probability adjustment of 46%.

Probability-adjusted DCF analysis

Cash driven NPV	2016	2017	2018	2019	2020	2021	2022	2023	2024	2025
Revenue	0	1,492	12,162	49,287	123,139	222,637	320,649	396,499	444,719	490,603
R&D	10,348	11,694	12,746	13,893	15,005	16,055	17,179	18,381	19,668	21,045
SG&A	3,488	18,976	20,675	29,958	31,456	33,029	34,680	36,415	38,235	40,147
Operating income	(13,836)	(29,312)	(22,354)	1,000	65,596	153,516	239,931	306,018	346,791	419,530
Net income	(14,004)	(29,480)	(22,522)	524	41,220	96,609	151,051	192,686	218,372	264,198
Period	0.9	1.9	2.9	3.9	4.9	5.9	6.9	7.9	8.9	9.9
NPV	(12,335)	(22,679)	(15,132)	308	21,124	43,241	59,046	65,783	65,111	68,799

Total DCF	273,265
Terminal value	233,916
Cash (4Q15)	9,000
Total valuation ($ '000)	516,180
Probability adjustment	46%
Value per share	**$30.07**
Share outstanding (2015)	7,965
Discount rate	15%
Terminal value multiple	3

Source: Laidlaw & Company estimates

For the peer comparable analysis, we have chosen a group of companies mainly developing orphan drugs as comparable peers. As such, our peer comparable analysis suggested a 12-month target price for ALDX of $31.

In addition, our probability-adjusted-PV-driven, sum-of-the-parts analysis illustrates a breakdown of value for each potential value driver, with NS2 in Sjögren-Larsson Syndrome accounting for 43% of the total value, while NS2 in non-infectious acute anterior uveitis and the aldehyde trapping platform for future indications account for 33% and 20%, respectively. As such, our supplemented probability-adjusted-PV-driven, sum-of-the-part analysis suggested a 12-month target price of $30.88.

Comparable analysis

Company	Ticker	Rating	Target Price ($)	Price ($) (1/16/15)	Shares Outstanding (MM)	Market Cap ($ MM)	Cash ($ MM)	Debt ($ MM)	Tech Value ($ MM)	Most Advanced Development Stage	Major Indication
Sarepta Therapeutics	SRPT	NR	NA	12.11	41	500	240	4	264	Phase II	DMD
Mast Therapeutics	MSTX	Buy	2.50	0.49	129	63	57	0	6	Phase III	SCD, CV
GlycoMimetics	GLYC	NR	NA	7.05	19	133	61	0	72	Phase III	SCD, CV
QLT	QLTI	NR	NA	4.09	51	209	138	0	72	Phase I	Leber Congenital Amaurosis (LCA)
Catalyst Pharmaceutical	CPRX	NR	NA	2.94	69	203	38	0	165	Phase III	Lambert-Eaton Myasthenic Syndrome
Amicus Therapeutics	FOLD	NR	NA	8.16	79	647	85	0	562	Phase III	Pompe and Fabry diseases
Average						475	*105*	*3*	190		
Aldeyra Therapeutics	ALDX	Buy	30.00	10.17	7	68	17	0	51	Phase II	SLS, AU

RNN share fair value matching its Phase I/II oncology peers = $31.01

Potential upside = 205%

Source: Company reports and Laidlaw & Company estimates

NPV driven sum-of-the-parts analysis

NS2 cream	SLS		
	Adjusted NPV =	$106.7	
	PV per share =	$13.40	43%
NS2 eye-drop	Acute anterior uveitis		
	Adjusted NPV =	$82.2	
	PV per share =	$10.33	33%
Aldehyde trapping			
	Adjusted NPV =	$48.0	
	PV per share =	$6.03	20%
Cash			
	Adjusted NVP =	$9.0	
	NVP per share =	$1.13	4%
	Total =	$30.88	100%

Source: Laidlaw & Company estimates

Together, we assigned our blended 12-month target price for ALDX of **$30**.

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Major risks

Risks of clinical study failure could have a major impact on ALDX share value. Despite promising aspects of the company's lead products, NS2 in the two indications under clinical trials, it remains too early to predict the safety and efficacy from the two ongoing Phase II studies. Given clinical validation for these programs has not been established, it would be critical for one or both studies to demonstrate a positive outcome in order to increase the assets and shareholder value. Negative results of either Phase II studies could potentially impair their value and have a materially negative impact on shareholder value especially since success of the each study could illustrate NS2 treatment potential of separated disease areas. Further, it remains too early to predict any potential success of clinical trials should these programs further advance into later clinical stage development. In SLS, although it is possible that elevated fatty alcohol, instead of elevated aldehyde, could impact the progression of the disease. If so, NS2 potentially might not have the therapeutic effect affecting the change of elevated fatty alcohol levels. We, however, view this to be a very modest risk.

Product may not be approved or reach anticipated sales. Although Aldeyra's current pipeline products have exhibited the potential to generate positive clinical outcomes from current and future trials; it remains too early to project whether any of these products would be approved by regulatory agencies. Even if the products were to enter the market, sales could be significantly below projections due to the specific product label under approval, physician consensus for prescribing the drug, changes of treatment paradigms, entrance of competitors, and possibly the changes in pricing flexibility and payer reimbursement. A revenue outlook below expectations could also negatively affect ALDX shareholder value.

Limited product offering and further validation of technology represent limited diversification to investors. The major technology platform of ALDX is aldehyde trapping and the company currently has only one drug, NS2, in two different delivery forms, in clinical studies. As such, ALDX has a very concentrated product offering portfolio and hence, exhibits limited diversification for investors. In addition, although aldehyde trapping is a novel and logical approach in drug development, it remains too early to gain greater buy-in within medical and investor communities since clinical validation remains very limited.

Additional financings could dilute shareholder value. Although the company currently has ~$17MM (pro forma) cash after recent financing, ALDX could need more financial resources going forward if they want to expand and further develop its pipeline. Should the product not receive FDA approval, or product revenue does not reach expectations; the company might need to issue new

equity to raise additional cash. Under such a scenario, the share value of existing shareholders could be diluted.

Limited trading liquidity limits shareholder options. Given ALDX shares only entered the public market recently; daily trading volume and name recognition are relatively modest. With relatively illiquid trading volume, shareholders wanting to increase or reduce their positions in a volatile stock market may face constraints.

Management

Todd C. Brady, M.D., Ph.D. is Aldeyra Therapeutics CEO since 2012 and board of directors since 2005. Prior joining Aldeyra, he has served as Entrepreneur in Residence from April to December 2013 and as Principal from 2004 to 2013 at Domain Associates. Prior to Domain Associates from 2002 to 2004, he served as Senior Director of business development at Aderis Pharmaceuticals. Prior, he was Executive Vice President of Corporate Development and Strategy at Xanthus Life Sciences from 2001 to 2002; and before that, Dr. Brady was Chief Executive Officer of Phenome Sciences from 2000 to 2001. Prior, he was Dr. Brady was a senior associate at CB Health Ventures, LLC. Dr. Brady holds a Ph.D. degree in pathology from Duke University and a M.D. from Duke University Medical School.

Stephen Tulipano has served as CFO of Aldeyra Therapeutics since June 2014. Prior to joining Aldeyra Mr. Tulipano held positions at Three Tulips providing accounting services and financial management counsel from 2011 to 2014. Prior to joining Three Tulips, he served as Chief Financial Officer of Javelin Pharmaceuticals from 2006 to 2010. Prior, he served as the Director of Corporate Accounting at Biogen Idec from 1998 to 2006. Prior he has held several accounting roles both within companies and accounting firms. Mr. Tulipano holds an M.B.A. in Finance from Suffolk University.

Scott L. Young has served as Chief Operating Officer of Aldeyra Therapeutics since December 2011. Prior joining Aldeyra from 2006 to 2011, Mr. Young has served as Chief Operating Officer for Link Medicine Corporation. Prior, he served as Chief Operating Officer of OXiGENE from 1999 through 2006. Prior, Mr. Young has also held various positions at Genzyme Corporation, RepliGen Corporation and Genetics Institute. Mr. Young holds a BS degree in biochemistry from the University of Massachusetts.

Aldeyra Therapeutics – Income Statement

($'000)	2012	2013	1Q14	2Q14	3Q14	4Q14E	2014E	1Q15E	2Q15E	3Q15E	4Q15E	2015E	2016E	2017E	2018E	2019E	2020E
Revenue																	
Product revenue	0	0	-	-	-	-	0	-	-	-	-	0	0	1,492	12,162	49,287	123,139
Other revenue	0	0	-	-	-	-	0	-	-	-	-	0	0	0	0	0	0
Total revenue	0	0	-	-	-	-	0	-	-	-	-	0	0	1,492	12,162	49,287	123,139
Costs of goods														134	1,095	4,436	11,083
Gross sales														1,358	11,067	44,851	112,057
Research and development	469	1,542	444	664	1,196	1,327	3,631	1,778	1,956	2,132	2,218	8,085	10,348	11,694	12,746	13,893	15,005
General and administrative	645	2,135	801	983	772	780	3,336	788	796	804	812	3,200	3,488	3,976	4,175	4,383	4,602
Marketing and sales														15,000	16,500	25,575	26,854
Total Operating Expenses	1,114	3,676	1,245	1,646	1,968	2,107	6,967	2,566	2,752	2,936	3,030	11,284	13,836	30,669	33,421	43,852	46,461
Operating Incomes (losses)	(1,114)	(3,676)	(1,245)	(1,646)	(1,968)	(2,107)	(6,967)	(2,566)	(2,752)	(2,936)	(3,030)	(11,284)	(13,836)	(29,312)	(22,354)	1,000	65,596
Change in fair value of preferred stock warrant liabilities	(9)	721	1,760	568	-	-	2,328	-	180	200	110	490	500	500	500	500	500
Change in fair value of convertible preferred stock rights and rig	(126)	16,175	-	-	-	-	0	-	-	-	-	0	0	0	0	0	0
Value provided in excess of issuance price of Series B convert	(21,485)						0	-	-	-	-	0	0	0	0	0	0
Interest income	0	0	-	-	-	-	0	-	-	-	-	0	0	0	0	0	0
Other expenses	1	0						-	-	-	-	0	0	0	0	0	0
Interest expense	(342)	(159)	(113)	(56)	(41)	(42)	(253)	(42)	(42)	(42)	(42)	(168)	(168)	(168)	(168)	(168)	(168)
Total Other Income (Expense)	(21,951)	16,737	1,647	511	(41)	(42)	2,075	(42)	138	158	68	(168)	(168)	(168)	(168)	(168)	(168)
Net loss and comprehensive loss	(23,075)	13,060	402	(1,135)	(2,009)	(2,149)	(4,892)	(2,608)	(2,614)	(2,778)	(2,962)	(11,452)	(14,004)	(29,480)	(22,522)	832	65,428
Accretion of preferred stock	(389)	(823)	(192)	(142)	-	-	(333)	-	-	-	-	0	0	0	0	0	0
Allocation of undistributed earnings to preferred stockholders		(11,128)	(223)	-	-	-	(223)	-	-	-	-	0	0	0	0	0	0
Deemed dividend	(15,662)	0	-	(4,054)	-	-	(4,054)	-	-	-	-	0	0	0	0	0	0
Tax	0	0	-	-	-	-	0	-	-	-	-	0	0	0	0	(308)	(24,208)
Net Income (Loss)	(39,126)	1,110	(13)	(5,330)	(2,009)	(2,149)	(9,502)	(2,608)	(2,614)	(2,778)	(2,962)	(11,452)	(14,004)	(29,480)	(22,522)	524	41,220
Net Income (Loss) Applicable to Common Shareholders	(39,126)	1,110	(13)	(5,330)	(2,009)	(2,149)	(9,502)	(2,608)	(2,614)	(2,778)	(2,962)	(11,452)	(14,004)	(29,480)	(22,522)	524	41,220
Net Earnings (Losses) Per Share—Basic	($124.44)	$3.49	($0.04)	($1.43)	($0.36)	($0.37)	($2.47)	($0.37)	($0.36)	($0.37)	($0.37)	($1.54)	($1.49)	($2.83)	($1.97)	$0.04	$3.07
Net Earnings (Losses) Per Share—Diluted	($124.44)	($17.58)	($4.00)	($1.56)	($0.36)	($0.37)	($2.45)	($0.37)	($0.36)	($0.37)	($0.37)	($1.54)	($1.49)	($2.83)	($1.97)	$0.04	$3.07
Shares outstanding—basic	314	318	327	3,738	5,565	5,765	3,849	7,065	7,165	7,465	7,965	7,415	9,415	10,415	11,415	12,415	13,415
Shares outstanding—diluted	314	857	444	3,769	5,565	5,765	3,886	7,065	7,165	7,465	7,965	7,415	9,415	10,415	11,415	12,415	13,415
Margin Analysis (% of Sales/Revenue)																	
Costs of goods													9%	9%	9%	9%	9%
R&D	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	784%	105%	28%	12%
SG&A	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	266%	34%	9%	4%
Operating Income (loss)	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	-1964%	-184%	2%	53%
Net Income	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	-1976%	-185%	1%	33%
Financial Indicator Growth Analysis (YoY%)																	
Total Revenue	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	715%	305%	150%
R&D	NA	229%	196%	104%	80%	232%	136%	300%	195%	78%	67%	123%	28%	13%	9%	9%	8%
SG&A	NA	231%	467%	49%	54%	-6%	56%	-2%	-19%	4%	4%	-4%	9%	14%	5%	5%	5%
Marketing and sales															10%	55%	5%
Operating Income (Losses)	NA	230%	327%	67%	69%	71%	90%	106%	67%	49%	44%	62%	23%	112%	-24%	-104%	6461%
Pretax Income	NA	-157%	-110%	-28%	-122%	-123%	-137%	-749%	130%	38%	38%	134%	22%	111%	-24%	-104%	7766%
Net Income	NA	-103%	-100%	210%	-329%	-136%	-956%	19601%	-51%	38%	38%	21%	22%	111%	-24%	-102%	7766%
EPS	NA	-103%	-100%	-74%	-113%	-102%	-171%	813%	-74%	3%	0%	-37%	-4%	90%	-30%	-102%	7179%

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Source: Bloomberg LP; Company reports; Laidlaw & Company estimates

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Aldeyra Therapeutics – Balance Sheet

($'000)	2012	2013	1Q14	2Q14	3Q14	4Q14E	2014E	1Q15E	2Q15E	3Q15E	4Q15E	2015E	2016E
Assets													
Cash and cash equivalents	1,224	3,262	2,146	11,536	10,142	8,423	8,423	15,592	13,011	11,082	9,753	9,263	26,782
Short term investments	0	0	0	0	0	0	0					0	0
Liqid assets	***1,224***	***3,262***	***2,146***	***11,536***	***10,142***	***8,423***	***8,423***	***15,592***	***13,011***	***11,082***	***9,753***	***9,263***	***26,782***
Preferred stock issuance receivable – related party	750												
Prepaid expenses and other current assets	3	8	10	270	203	250	250	220	255	315	298	298	320
Total Current Assets	1,977	3,271	2,155	11,805	10,345	8,673	8,673	15,812	13,266	11,397	10,051	9,561	27,102
Deferred offering cost	0	472	735	-	-	-	0	-	-	-	-	0	0
Fixed Assets, net	0	0	-	-	6	7	7	7	7	8	8	8	15
Total Assets	**1,977**	**3,743**	**2,891**	**11,805**	**10,351**	**8,680**	**8,680**	**15,819**	**13,273**	**11,405**	**10,059**	**9,569**	**27,117**
Liabilities and Stockholders' Equity													
Accounts payable	73	342	166	556	482	557	557	580	639	653	677	677	741
Convertible notes payable - related parties	0	85	155	-	-	-	0	-	-	-	-	0	0
Accrued interest on convertible notes payable - relate	0	2	5	-	-	-	0	-	-	-	-	0	0
Accrued expenses	124	118	133	211	364	404	404	439	470	477	463	463	502
Current portion of credit facility	167	58	233	407	-	-	0	-	-	-	-	0	0
Total current liabilities	363	605	692	1,175	846	961	961	1,019	1,109	1,129	1,140	1,140	1,243
Credit facility, net of current portion and debt discount	266	1,129	972	816	1,241	1,584	1,584	2,021	2,151	2,290	2,996	2,996	4,642
Accrued deferred offering costs	0	394	604	-	-	-	0	-	-	-	-	0	0
Convertible preferred stock rights and rights option lia	24,234		-	-	-	-	0	-	-	-	-	0	0
Convertible preferred stock warrant liability	88	253	132	-	-	-	0	-	-	-	-	0	0
Convertible preferred stock warrant liabilities - related	2,181	3,266	1,627	-	-	-	0	-	-	-	-	0	0
Total Liabilities	27,132	5,647	4,027	1,990	2,087	2,546	2,546	3,041	3,260	3,420	4,136	4,136	5,886
Series A and B preferred stock	29,230	38,317	38,509	-	-	-	0	-	-	-	-	-	-
Preferred stock	0	0	-	-	-	-	0	-	-	-	-	-	-
Common stock	0	0	0	6	6	6	6	8	7	7	7	7	9
Additional paid-in capital	0	1,103	1,277	51,867	52,325	52,345	52,345	61,595	61,445	62,195	63,095	63,095	92,895
Deficit accumulated during the development stage	(54,385)	(41,324)	(40,923)	(42,058)	(44,067)	(46,216)	(46,216)	(48,825)	(51,439)	(54,217)	(57,179)	(57,669)	(71,673)
Total Stockholders' Equity	(54,384)	(40,221)	(1,137)	9,815	8,264	6,135	6,135	12,778	10,013	7,985	5,923	5,433	21,231
Total Liabilities and Stockholders' Equity	**1,977**	**3,743**	**2,891**	**11,805**	**10,351**	**8,680**	**8,680**	**15,819**	**13,273**	**11,405**	**10,059**	**9,569**	**27,117**

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Source: Bloomberg LP; Company reports; Laidlaw & Company estimates

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Aldeyra Therapeutics – Cash Flow Statement

($'000) Cash Flows From Operating Activities:	2012	2013	3Q14	4Q14E	2014E	1Q15E	2Q15E	3Q15E	4Q15E	2015E	2016E
Net income (loss)	(23,075)	13,060	(2,009)	(2,149)	(4,892)	(2,608)	(2,614)	(2,778)	(2,962)	(11,452)	(14,004)
Adjustments to reconcile net loss to net cash used in operating activities:											
Stock-based compensation	84	1,702	458	300	1,872	410	290	310	790	1,800	2,000
Interest converted to preferred stock	306										
President and CEO contributed services	170	46	0	0	0	0	0	0	0	0	0
Amortization of debt discount – non-cash interest expense	21	121	18	22	161	30	20	30	30	110	146
Change in fair value of warrant liability, purchase rights and warrant purchase rights	135	(16,896)	0	0	(2,328)	0	(180)	(200)	(110)	(490)	(500)
Value provided in excess of issuance price of Series B redeemable convertible preferred	21,485										
Depreciation	4	0	0	25	25	5	5	5	5	20	30
Changes in operating assets and liabilities:											
Prepaid expenses and other current assets	1	(5)	67	(47)	(242)	30	(35)	(60)	17	(48)	(22)
Deferred offering costs	0	0	0								
Accounts payable	47	269	103	75	216	23	59	14	24	120	64
Security deposit		0	0	0	0	0	0	0	0	0	0
Accrued interest on convertible notes related parties		2	0		0	0	0	0	0	0	0
Accrued deferred offering costs		0	0	0	0	0	0	0	0	0	0
Accrued expenses	43	(6)	152	40	285	35	30	7	(14)	59	40
Net Cash from Operating Activities	**(778)**	**(1,707)**	**(1,211)**	**(1,734)**	**(4,903)**	**(2,075)**	**(2,425)**	**(2,672)**	**(2,219)**	**(9,882)**	**(12,246)**
Cash flows from investing activities:											
Acquisitions of property and equipment	0	0	(6)	(5)	(11)	(6)	(6)	(7)	(9)	(28)	(35)
Net Cash from Investing Activities	**0**	**0**	**(6)**	**(5)**	**(11)**	**(6)**	**(6)**	**(7)**	**(9)**	**(28)**	**(35)**
Cash Flows From Financing Activities:											
Principal payments on notes	0	0									
Proceeds from convertible notes payable – related parties	0	170	0	0	0	0	0	0	0	0	0
Issuance of common stock and units, net of issuance costs	0	7	(177)	20	10,075	9,250	0	750	1,000	11,000	30,000
Borrowings under credit facility, net	500	1,000	0	0	0	0	0	0	0	0	0
Cash paid for deferred offering costs	0	(78)	0	0	0	0	0	0	0	0	0
Repayments of credit facility	0	(104)	0	0	0	0	(150)	0	(100)	(250)	(200)
Net proceeds from issuance of Series B redeemable convertible preferred stock	1,251	2,750	0	0	0	0	0	0	0	0	0
Net Cash Provided by Financing Activities	**1,751**	**3,745**	**(177)**	**20**	**10,075**	**9,250**	**(150)**	**750**	**900**	**10,750**	**29,800**
Net increase (decrease) in cash	**973**	**2,039**	**(1,394)**	**(1,719)**	**5,161**	**7,169**	**(2,581)**	**(1,929)**	**(1,328)**	**840**	**17,519**
Cash at beginning of period	251	1,224	11,536	10,143	3,262	8,423	15,592	13,011	11,082	8,423	9,263
Cash at end of period	**1,224**	**3,262**	**10,143**	**8,423**	**8,423**	**15,592**	**13,011**	**11,082**	**9,753**	**9,263**	**26,782**

**Net income (loss) are the same as "Net loss and comprehensive loss" from income statement and as reported at S1/A filing as of 05/01/2014

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Source: Bloomberg LP; Company reports; Laidlaw & Company estimate

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DISCLOSURES:

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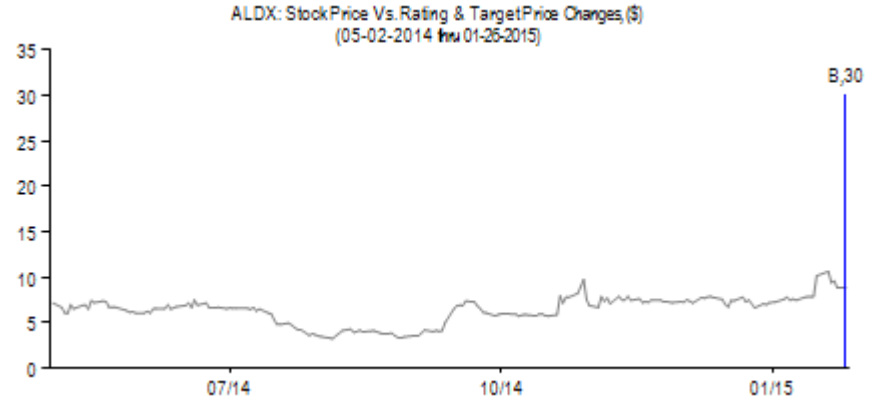
Additional information available upon request.

‡ Laidlaw & Company has received compensation from the subject company for investment banking services in the past 12 months and expects to receive or intends to seek compensation for investment banking services from the company in the next three months.

RATINGS INFORMATION

Rating and Price Target Change History



3 Year Rating Change History

Date	Rating	Closing Price ($)
01/26/2015	Buy (B)	8.86*

3 Year Price Change History

Date	Target Price ($)	Closing Price, ($)
01/26/2015	30.00	8.86*

** Previous Close1/23/2015*

Source: Laidlaw & Company **Created by: Blue-Compass.net**

Laidlaw & Company Rating System*		% of Companies Under Coverage With This Rating	% of Companies for which Laidlaw & Company has performed services for in the last 12 months	
			Investment Banking	Brokerage
Strong Buy (SB)	Expected to significantly outperform the sector over 12 months.	0.00%	0.00%	0.00%
Buy (B)	Expected to outperform the sector average over 12 months.	81.82%	36.36%	9.09%
Hold (H)	Expected returns to be in line with the sector average over 12 months.	4.55%	0.00%	0.00%
Sell (S)	Returns expected to significantly underperform the sector average over 12 months.	0.00%	0.00%	0.00%

ADDITIONAL COMPANIES MENTIONED

Valeant Pharmaceuticals (VRX – Not Rated)

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