UNITED STATES
SECURITIES AND EXCHANGE COMMISSION
Washington, D.C. 20549

FORM 8-K

CURRENT REPORT
Pursuant to Section 13 or 15(d)
of the Securities Exchange Act of 1934

Date of Report (Date of earliest event reported): June 20, 2024

ALDEYRA THERAPEUTICS, INC.
(Exact name of Registrant as specified in its charter)

Delaware	001-36332	20-1968197
(State or other jurisdiction of incorporation)	(Commission File No.)	(IRS Employer Identification No.)

131 Hartwell Avenue, Suite 320
Lexington, MA 02421
(Address of principal executive offices and zip code)

Registrant's telephone number, including area code: (781) 761-4904

Not Applicable
(Former Name or Former Address, if Changed Since Last Report)

Check the appropriate box below if the Form 8-K filing is intended to simultaneously satisfy the filing obligation of the registrant under any of the following provisions:

☐ Written communications pursuant to Rule 425 under the Securities Act (17 CFR 230.425)

☐ Soliciting material pursuant to Rule 14a-12 under the Exchange Act (17 CFR 240.14a-12)

☐ Pre-commencement communications pursuant to Rule 14d-2(b) under the Exchange Act (17 CFR 240.14d-2(b))

☐ Pre-commencement communications pursuant to Rule 13e-4(c) under the Exchange Act (17 CFR 240.13e-4(c))

Securities registered pursuant to Section 12(b) of the Act:

Title of each class	Trading Symbol(s)	Name of each exchange on which registered
Common Stock, $0.001 par value per share	ALDX	The Nasdaq Stock Market LLC

Indicate by check mark whether the registrant is an emerging growth company as defined in Rule 405 of the Securities Act of 1933 (§230.405 of this chapter) or Rule 12b-2 of the Securities Exchange Act of 1934 (§240.12b-2 of this chapter).

Emerging growth company ☐

If an emerging growth company, indicate by check mark if the registrant has elected not to use the extended transition period for complying with any new or revised financial accounting standards provided pursuant to Section 13(a) of the Exchange Act. ☐

Item 8.01. Other Events.

On June 20, 2024, Aldeyra Therapeutics, Inc. (the "Company") issued a press release (the "Press Release") to announce the advancement of new RASP modulators and recent preclinical data in obesity. As part of a virtual Investor Roundtable on June 20, 2024, the Company will discuss the announcement, among other topics. The Press Release is filed herewith as Exhibit 99.1 and is incorporated by reference herein.

Item 9.01. Financial Statements and Exhibits.

(d) Exhibits

Exhibit No.	Description
99.1	Aldeyra Therapeutics, Inc. Press Release dated June 20, 2024
104	Cover Page Interactive Data File (embedded within the Inline XBRL document)

SIGNATURES

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

ALDEYRA THERAPEUTICS, INC.

By: /s/ Todd C. Brady
Name: Todd C. Brady, M.D., Ph.D.
Title: Chief Executive Officer

Dated June 20, 2024

News Release

Aldeyra Therapeutics Announces Advancement of New RASP Modulators and Recent Preclinical Data in Obesity at 2024 Investor Roundtable

Live Webcast Scheduled to Begin at 8:00 a.m. ET Today

Lexington, Mass., June 20, 2024 – Aldeyra Therapeutics, Inc. (Nasdaq: ALDX) (Aldeyra), a biotechnology company devoted to discovering and developing innovative therapies designed to treat immune-mediated and metabolic diseases, today announced advancement of new RASP modulators and recent preclinical data in obesity in conjunction with an Investor Roundtable scheduled to begin at 8:00 a.m. ET today.

Pipeline Updates

- Following positive biomarker results in adults, including near-normalization of lipid profiles, ADX-629, a first-in-class orally administered investigational RASP modulator, advanced to the pediatric cohort of the Phase 2 clinical trial in Sjögren-Larsson Syndrome, a rare inborn error of metabolism caused by mutations in fatty aldehyde dehydrogenase and characterized by cognitive dysfunction and skin disorders. Top-line results from approximately five pediatric patients are expected in 2025.

- Based on new results in a preclinical model of obesity, novel RASP modulator ADX-743 was advanced to Investigational New Drug (IND)-enabling studies. ADX-743 is an analog of ADX-629, which has demonstrated lowering of triglycerides and fatty acids in Phase 1 and Phase 2 clinical trials. Pending additional results, Aldeyra expects to submit an IND application for ADX-743 or an alternative RASP modulator for obesity or hypertriglyceridemia in 2025.

- Based in part on superior preclinical results in atopic dermatitis and unprecedented exposure in preclinical RASP modulator pharmacokinetic models, ADX-248 will be advanved in lieu of ADX-246 to Phase 1/2 clinical testing in atopic dermatitis. Phase 1 clinical testing of ADX-248 is expected to begin in the second half of 2024.

- Novel RASP modulator ADX-631 has initiated preclinical testing in models of retinal disease. ADX-631 is an analog of ADX-103, which previously demonstrated activity in animal models of the dry form of age-related macular degeneration (dry AMD), retinal inflammation, and diabetic macular edema. Aldeyra expects to submit an IND application for ADX-631 or an alternative RASP modulator for the treatment of dry AMD or geographic atrophy in the first half of 2025.

“Consistent with the new data presented at our Research and Development Day in April of this year, we continue to expand our novel RASP modulator pipeline with the discovery and advancement of new RASP modulators for the treatment of inflammatory and metabolic diseases,” stated Todd C. Brady, M.D., Ph.D., President and CEO of Aldeyra. “We look forward to discussing our recent progress on today’s call with investors and are excited to share subsequent updates on our pipeline throughout the remainder of 2024.”

Investor Roundtable Webcast Information

The 2024 Aldeyra Therapeutics Investor Roundtable will take place at 8:00 a.m. ET today, Thursday, June 20, 2024. A live audio webcast will be accessible from the “Investors & Media” section of the Aldeyra website at https://ir.aldeyra.com/. A replay will be available for 90 days following the event.

About Aldeyra

Aldeyra Therapeutics is a biotechnology company devoted to discovering innovative therapies designed to treat immune-mediated and metabolic diseases. Our approach is to develop pharmaceuticals that modulate protein systems, instead of directly inhibiting or activating single protein targets, with the goal of optimizing multiple pathways at once while minimizing toxicity. Our product candidates include RASP (reactive aldehyde species) modulators ADX-629, ADX-248, ADX-743, ADX-631, and chemically related molecules for the potential treatment of systemic and retinal immune-mediated and metabolic diseases. Our late-stage product candidates are reproxalap, a RASP modulator for the potential treatment of dry eye disease and allergic conjunctivitis, and ADX-2191, a novel formulation of intravitreal methotrexate for the potential treatment of retinitis pigmentosa.

Safe Harbor Statement

This release contains forward-looking statements within the meaning of the Private Securities Litigation Reform Act of 1995, including, but not limited to, statements regarding Aldeyra's future expectations, plans, and prospects, including without limitation statements regarding: the goals, opportunity, and potential for Aldeyra's product candidates; the outcome and expected timing and the results of Aldeyra's planned preclinical and clinical trials, including planned and ongoing trials; the outcome and timing of the FDA's review, acceptance and/or approval of IND submissions for Aldeyra's product candidates. Aldeyra intends such forward-looking statements to be covered by the safe harbor provisions for forward-looking statements contained in Section 21E of the Securities Exchange Act of 1934 and the Private Securities Litigation Reform Act of 1995. In some cases, you can identify forward-looking statements by terms such as, but not limited to, "may," "might," "will," "objective," "intend," "should," "could," "can," "would," "expect," "believe," "anticipate," "project," "on track," "scheduled," "target," "design," "estimate," "predict," "contemplates," "likely," "potential," "continue," "ongoing," "aim," "plan," or the negative of these terms, and similar expressions intended to identify forward-looking statements. Such forward-looking statements are based upon current expectations that involve risks, changes in circumstances, assumptions, and uncertainties. Aldeyra is at an early stage of development and may not ever have any products that generate significant revenue. All of Aldeyra's development timelines may be subject to adjustment depending on recruitment rate, regulatory review, preclinical and clinical results, funding, and other factors that could delay the initiation, enrollment, or completion of clinical trials. Important factors that could cause actual results to differ materially from those reflected in Aldeyra's forward-looking statements include, among others, the timing of enrollment, commencement and completion of Aldeyra's clinical trials, the timing and success of preclinical studies and clinical trials conducted by Aldeyra and its development partners; delay in or failure to obtain regulatory approval of Aldeyra's product candidates, including as a result of the FDA not accepting Aldeyra's regulatory filings, issuing a complete response letter, or requiring additional clinical trials or data prior to review or approval of such filings or in connection with resubmissions of such filings; the ability to maintain regulatory approval of Aldeyra's product candidates, and the labeling for any approved products; the risk that prior results, such as signals of safety, activity, or durability of effect, observed from preclinical or clinical trials, will not be replicated or will not continue in ongoing or future studies or clinical trials involving Aldeyra's product candidates in clinical trials focused on the same or different indications; the scope, progress, expansion, and costs of developing and commercializing Aldeyra's product candidates; uncertainty as to Aldeyra's ability to commercialize (alone or with others) and obtain reimbursement for Aldeyra's product candidates following regulatory approval, if any; the size and growth of the potential markets and pricing for Aldeyra's product candidates and the ability to serve those markets; Aldeyra's expectations regarding Aldeyra's expenses and future revenue, the timing of future revenue, the sufficiency or use of Aldeyra's cash resources and needs for additional financing; the rate and degree of market acceptance of any of Aldeyra's product candidates; Aldeyra's expectations regarding competition; Aldeyra's anticipated growth strategies; Aldeyra's ability to attract or retain key personnel; Aldeyra's commercialization, marketing and manufacturing capabilities and strategy; Aldeyra's ability to establish and maintain development partnerships; Aldeyra's ability to successfully integrate acquisitions into its business; Aldeyra's expectations regarding federal, state, and foreign regulatory requirements; political, economic, legal, social, and health risks, public health measures, and war or other military actions, that may affect Aldeyra's business or the global economy; regulatory developments in the United States and foreign countries; Aldeyra's ability to obtain and maintain intellectual property protection for its product candidates; the anticipated trends and challenges in Aldeyra's business and the market in which it operates; and other factors that are described in the "Risk Factors" and "Management's Discussion and Analysis of Financial Condition and Results of Operations" sections of Aldeyra's Annual Report on Form 10-K for the year ended December 31, 2023 and Aldeyra's Quarterly Report on Form 10-Q for the quarter ended March 31, 2024, which are on file with the Securities and Exchange Commission (SEC) and available on the SEC's website at https://www.sec.gov/. Additional factors may be described in those sections of Aldeyra's Quarterly Report on Form 10-Q for the quarter ended June 30, 2024, expected to be filed with the SEC in the third quarter of 2024, and Aldeyra's other filings with the SEC.

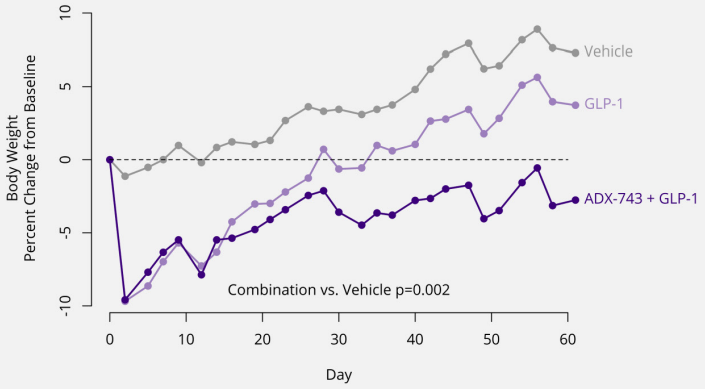
In addition to the risks described above and in Aldeyra's other filings with the SEC, other unknown or unpredictable factors also could affect Aldeyra's results. No forward-looking statements can be guaranteed and actual results may differ materially from such statements. The information in this release is provided only as of the date of this release, and Aldeyra undertakes no obligation to update any forward-looking statements contained in this release on account of new information, future events, or otherwise, except as required by law.

Investor & Media Contact:

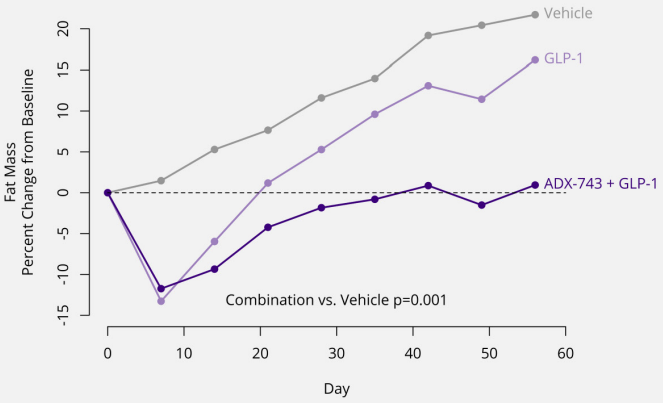
David Burke
Tel: (917) 618-2651
investorrelations@aldeyra.com

ADX-743, an Analog of ADX-629, Demonstrated Preclinical Weight Loss and Reduction of Fat Mass in Combination with GLP-1 Agonist

Body Weight



Fat Mass



ADX-743 is an investigational drug candidate. SEM = standard error of the mean. GLP-1 = glucagon-like peptide-1 receptor agonist. Data are from murine diet-induced obesity model.

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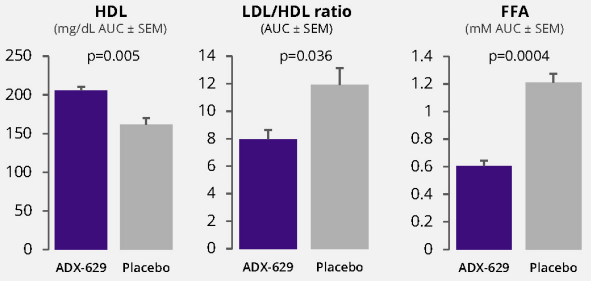
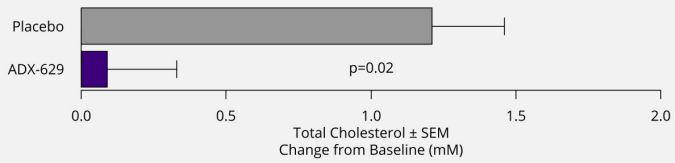
Statistically Significant Changes Observed in Lipid Profiles in Multiple Clinical Trials with RASP Modulator ADX-629

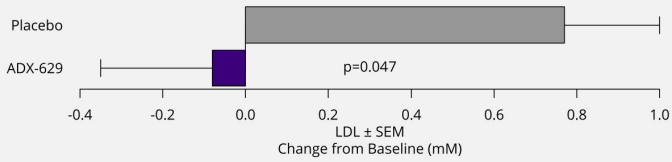
Phase 1 Clinical Trial



Phase 2 Psoriasis Clinical Trial

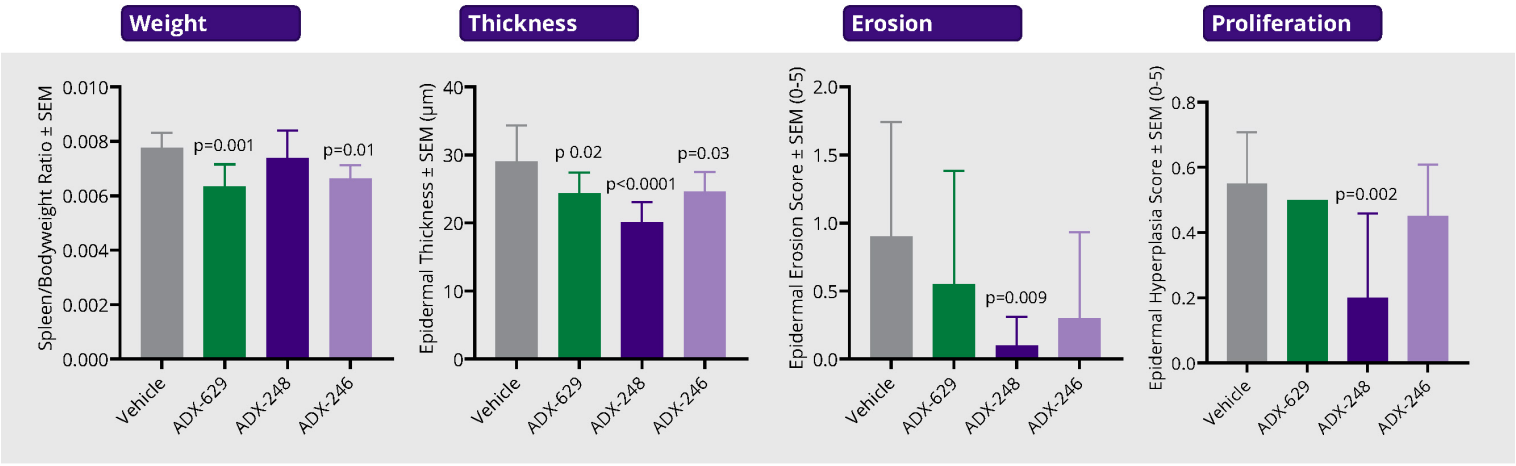
Phase 1/2 Ethanol Toxicity Clinical Trial





ADX-629 is an investigational drug candidate. SEM = standard error of the mean. HDL = high-density lipoprotein. LDL = low-density lipoprotein. FFA = free fatty acids. AUC = area under the curve. mM = millimolar.

RASP Modulators ADX-629, ADX-248, and ADX-246 Reduced Histopathology and Spleen Weight in a Preclinical Model of Atopic Dermatitis



ADX-629, ADX-248, and ADX-246 are investigational drug candidates. SEM = standard error of mean.

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Treatment with ADX-629 Normalized Biomarkers of Representative Adult Sjögren-Larsson Syndrome Patient After 12 Weeks

Metabolite	Baseline Z-score	ADX-629 12 weeks Z-score
1-pentadecanoyl-2-arachidonyl-GPC	3.1	2.6
Arginate	2.8	3.5
Pyruvate	2.7	NS
1-Myristoyl-2-arachidonoyl -GPC	2.5	NS
1 Myristoyl-2-linoleoyl-GPC	2.4	NS
1-Palmitoyl-2-pentadecanoyl-GPC	2.4	NS
1-Pentadecanoyl-2-linoleoyl-GPC	2.4	NS
Imidazole lactate	2.3	2.4
1-Myristoyl-2-palmitoyl-GPC	2.2	NS
Homoarginine	2.1	2.6
Pipecolate	2.1	NS
3-hydroxy-2-ethylpropionate	2.1	NS
1-Steroyl-GPC	-2.0	NS
2-Acetamidobutanoate	-2.1	NS
Linolenoylcarnitine	-2.1	NS
5-(galactosylhydroxy)-lysine	-2.2	NS
N-acetylhistidine	-2.2	NS
Cis-4-decenoylcarnitine	-2.2	NS
Citrulline	-2.2	NS
Linoleoylcarnitine	-2.5	NS
Gamma-CEHC	-2.5	NS
Betaine	-2.9	-2.9
Pro-hydroxy-pro	-2.9	NS
7-alpha-hydroxy-3-oxo-4-cholestenoate (7-Hoca)	-3.6	-3.5
N-acetyltaurine	-3.8	NS
Tauro-cholenate sulfate	-3.9	-3.8
Glycocholenate sulfate	-4.5	-5.8

ADX-629 is an investigational new drug candidate. Baseline Z-score is number of standard deviations above (positive) or below (negative) normal subjects. NS = not significant.

ADX-103, a Novel Aldehyde Trap, Decreases Retinal Edema and Inflammation in a Rat Model of Diabetic Macular Edema

Adna Halilovic Ph.D.; Todd Brady M.D., Ph.D.; Susan Macdonald, Ph.D.
Aldeyra Therapeutics, Inc., Lexington, MA USA

INTRODUCTION

Diabetic Macular Edema (DME) is a common cause of vision loss. Hyperglycemia leads to carbonyl stress in the retina, resulting in accumulation of toxic aldehydes such as glyoxal, methylglyoxal, malondialdehyde, and 4-hydroxy-trans-2-nonenal. Aldehydes bind covalently to amine and thiol residues on proteins, and initiate inflammatory signaling cascades (Figure 1). The ability of the novel aldehyde trap, ADX-103, to prevent ocular inflammation was tested in a rat streptozotocin (STZ)-induced model of DME.

Schematic depicting inflammation pathways initiated by aldehyde mediators.

MATERIALS and METHODS

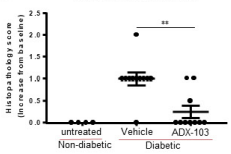
Brown Norway rats were administered STZ for six weeks to induce diabetes. Single doses of 3.5 µL of 0.5% ADX-103 (n=10) or saline vehicle (n=12) were administered by intravitreal (IVT) injection to STZ-treated rats at Weeks 6 and 8. Non-diabetic rats (n=4) did not receive any treatment and served as a negative control.

Retinal thickness, vascularity, and function were monitored weekly by optical coherence tomography (OCT) and fundus angiography (FA), from Weeks 6 through 10; electroretinograms (ERG) were generated on Weeks 6, 8 and 10.

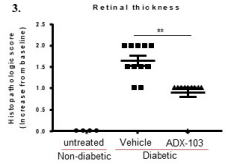
Rats were sacrificed at Week 10, and retinas were processed for histopathology. Six microscopic sections were examined for each eye, and retinas were assessed and scored by a pathologist for changes in retinal thickness, vascularity, and neutrophil infiltration.

RESULTS

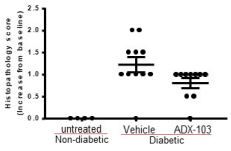
2. Neutrophil infiltration



3. Retinal thickness



4. Retinal vascularity

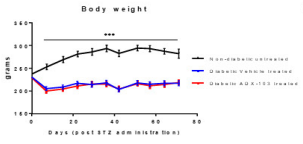


Treatment with ADX-103 significantly inhibited neutrophil infiltration in diabetic retinas. Microscopic sections of the retinas were assessed for changes in neutrophil infiltration. Reduction in neutrophil infiltration was observed in the diabetic rats treated with ADX-103 compared to vehicle-treated rats. Scoring: 0=normal, 1=minimal microscopically visible changes, 2=mild microscopically visible changes, 3=moderate microscopically visible changes. **p<0.01 Statistical analysis was performed by a non-parametric Dunn's multiple comparison followed by the Kruskal-Wallis test.

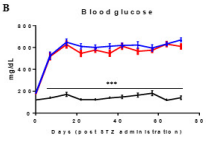
Treatment with ADX-103 resulted in a statically significant decrease in retinal thickness. Microscopic sections of the retinas were assessed for changes retinal thickness. Reduction in retinal thickness was observed in the diabetic rats treated with ADX-103 compared to vehicle treated rats. Scoring: 0=normal, 1=minimal microscopically visible changes; 2=mild microscopically visible changes; 3=moderate microscopically visible changes. **p<0.01 Statistical analysis was performed by a non-parametric Dunn's multiple comparison followed by the Kruskal-Wallis test.

Treatment with ADX-103 inhibited diabetes-induced retinal vascular changes. Microscopic sections of the retinas were assessed for vascular leakage. Compared to vehicle, reduction in retinal vascularity was observed in ADX-103-treated rats but was not statistically significant. Scoring: 0=normal, 1=minimal microscopically visible changes, 2=mild microscopically visible changes, 3=moderate microscopically visible changes.

A Body weight



B Blood glucose



Metabolic parameters. Body weight is significantly decreased and blood glucose is significantly increased in animals administered STZ. Body weight **(A)** and **(B)** non-fasting blood glucose were measured prior to STZ administration and weekly thereafter. ***p<0.001 from non-diabetic group. Statistical analysis was performed using Dunnett's test (compared to diabetic Vehicle treated group) following ANOVA.

SUMMARY

Diabetic retinopathy was successfully induced in STZ-treated rats. The diabetic animals lost approximately 10% of body weight in the first week post STZ administration, and maintained the weight until the end of study. Non-fasting glucose blood levels were significantly elevated in the diabetic rats compared to non-diabetic rats.

Increases in retinal thickness, vascularity, and neutrophil infiltration were observed in the vehicle-treated diabetic rats compared to the non-diabetic rats. ADX-103 treatment decreased retinal inflammation, as measured by statistically significant decreases in retinal thickness, neutrophil infiltration, and retinal vascular changes.

A decrease in vascular leakage was observed between the diabetic ADX-103-treated group and the vehicle-treated group, but the decrease did not reach statistical significance.

Although significant histopathological improvements were observed following treatment with ADX-103, ERG, OCT, or FFA did not show statistically significant effects following treatment with ADX-103.

CONCLUSIONS

Increasing evidence points to inflammation being one of the key contributors to the pathophysiology of diabetic retinopathy.

In the present DME study, histopathologic scoring showed statistically significant reductions in severities of retinopathy lesions, including neutrophil infiltration and retinal thickness, in diabetic rats treated with ADX-103 compared to vehicle.

Accumulation of intraretinal fluid, increase in cytokines, infiltration of leukocytes, neutrophils, and monocytes are early signs of disease progression[1].The data suggest that sequestration of aldehydes represents a novel therapeutic approach for the treatment of the ophthalmic inflammatory sequelae of diabetes.

1.Rübsam, A., Parikh, S., Fort, P.E. Role of Inflammation in Diabetic Retinopathy. *Int. J. Mol. Sci.* **2018**, *19*, 942.

Disclosures: Halilovic (E), Brady (E), Macdonald (E)

Presented at Association for Research in Vision and Ophthalmology 2018 Annual Meeting

Novel Small Molecule Aldehyde Sequestering Agents Demonstrate Broad Therapeutic Potential for Ocular Inflammation

Susan G. Macdonald, Ph.D.; Adna Halilovic, Ph.D.; Todd Brady, M.D.,
Ph.D. Aldeyra Therapeutics, Inc.

PURPOSE

Pro-inflammatory aldehydes, such as malondialdehyde (MDA), 4-hydroxy-nonenal, glyoxal, methylglyoxal, and retinal, have been implicated in the pathogenesis of several ocular inflammatory diseases. Aldehydes initiate activation of pro-inflammatory pathways, including the NFκB pathway. Recently, the aldehyde trap reproxalap (ADX-102) demonstrated statistically and clinically significant activity in Phase 2 clinical trials in allergic conjunctivitis, dry eye disease, and noninfectious anterior uveitis, all of which are ocular inflammatory diseases. To demonstrate the potential of aldehyde sequestration in inflammatory diseases involving the anterior and posterior segments of the eye, two structurally distinct aldehyde traps (reproxalap and ADX-103) were tested in two models of ocular inflammation.

METHODS

A mouse knockout model (abcr$^{-/-}$) of macular degeneration (MD) was used to test the activities of reproxalap and ADX-103. The retinal transport protein, ABCA4, is not expressed in these mice, allowing retinal to escape the light cycle and form a toxic metabolite with phosphatidylethanolamine: *N*-retinylidene-phosphatidylethanol-amine (A2E). Mice were treated intraperitoneally (IP) for 56 days with 10 mg/kg reproxalap (n = 24), ADX-103 (n = 24), or vehicle (n = 22), starting at week 10 to 12 of life. An untreated control group (n = 12) was sacrificed on the first day of dosing. After treatment, A2E concentrations in retinas were measured by HPLC. Statistical significance from vehicle control was determined by t-test.

In a rat model of lipopolysaccharide (LPS)-induced uveitis, 50 µg of reproxalap or ADX-103 was topically administered (TO) to the eye at hours 1, 3, 7, 10, and 17, after LPS induction, or by a single intravitreal (IVT) injection (25 µg) 1 hour after LPS induction (n = 10 per group). Ocular exams were performed 6 and 24 hours after LPS injection, after which retina-choroid specimens were processed for MDA adduct ELISA. Anterior segments were scored using a combined Draize and McDonald-Shattuck scoring system, and posterior segments were scored using 0 to 1 (vitreous, optic disc, retinal vasculature) and 0 to 4 (retinal and choroidal hemorrhage, exudation, and detachment) scales. Statistical significance from vehicle control was determined by ANOVA, followed by Tukey's *post hoc* test.

Data represent mean ± SEM. * $p \leq 0.05$ compared to vehicle; ** $p \leq 0.01$ compared to vehicle; *** $p \leq 0.001$ compared to vehicle; **** $p \leq 0.0001$ compared to vehicle

RESULTS

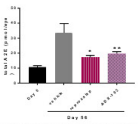


Figure 1: Both reproxalap and ADX-103 significantly decrease the formation of A2E in the retinas of abcr$^{-/-}$ mice. Mice were treated daily, IP, for 56 days with 10 mg/kg vehicle, reproxalap, or ADX-103

RESULTS (continued)

In the MD model, daily reproxalap or ADX-103 treatment decreased formation of A2E by 71% or 69%, respectively, compared with vehicle controls (Figure 1). In a separate study, systemic (IP) doses of reproxalap, 5-fold greater than the effective dose in the A2E study, administered for 56 days, had no effect on dark adaptation (data not shown). Daily treatment with reproxalap or ADX-103 for 56 days had no effect on body weight, nor were any drug-related adverse effects observed.

In the LPS-induced uveitis model, ocular exam scores were significantly improved, compared to vehicle, at 6 hours and 24 hours after TO administration of reproxalap or ADX-103. After IVT administration of reproxalap or ADX-103, ocular exam scores were also significantly improved vs. vehicle. Small, although not significant, reductions in MDA adducts were observed in the reproxalap and ADX-103 groups after TO and IVT administration (data not shown). To control for variations between individual animals, a within-subject statistical analysis, examining the co-variates of treatment and time, was conducted on the retina-choroid scores from the IVT groups. Retina-choroid scores in rats were significantly improved following IVT treatment with either reproxalap or ADX-103, compared to vehicle control (Figure 7).

Ocular Exam Scores Following Topical Ocular Dosing

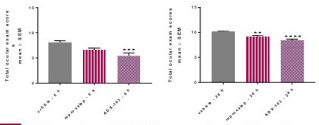


Figure 2: Both reproxalap and ADX-103 decrease total ocular inflammation.

Ocular Exam Scores Following Intravitreal Dosing

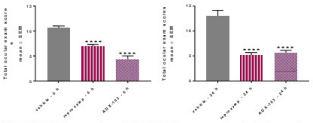


Figure 5: Both reproxalap and ADX-103 decrease total ocular inflammation.

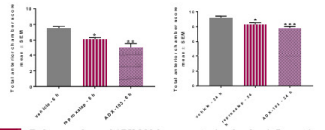


Figure 3: Both reproxalap and ADX-103 decrease anterior chamber inflammation.

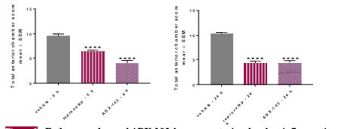


Figure 6: Both reproxalap and ADX-103 decrease anterior chamber inflammation.

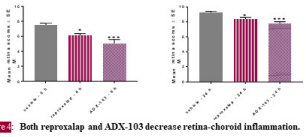


Figure 4: Both reproxalap and ADX-103 decrease retina-choroid inflammation.

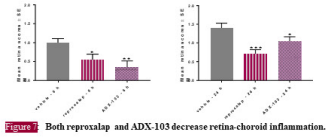


Figure 7: Both reproxalap and ADX-103 decrease retina-choroid inflammation.

CONCLUSIONS

Two structurally distinct aldehyde traps have shown activity in two models of ocular inflammation following ocular (TO, IVT) and systemic (IP) administration. No evidence of ocular or systemic toxicity was noted with any route of administration. Overall, the data suggest that aldehyde sequestration has broad potential for the treatment of a range of ocular diseases in which inflammation plays a role, including diseases involving the posterior pole and the anterior chamber.

Disclosures: Macdonald (E), Halilovic (E), Brady (E)

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