Morgan Stanley

February 29, 2016

Editas Medicine

Starting A Gene Editing Revolution

Industry View	Stock Rating	Price Target
In-Line	**Equal-weight**	**$28.00**

We initiate at EW with a $28 PT; Despite our LT optimism for the promise of gene editing, two near-term headwinds are likely to keep EDIT range bound – IP uncertainty and a lack of clinical catalysts until 2017. Over the long term, however, we see Editas as the premier gene editing company.

We initiate at EW with a $28 PT: Editas is using a new technology called CRISPR/Cas9 to edit (replace/remove) bad genes with good genes and achieve a therapeutic benefit in patients with genetic diseases. This approach differs from the more commonly known gene therapy in that it can accurately remove and replace genes instead of inserting a new gene sequence without specificity as happens in gene therapy. Thus, the potential of CRISPR is broad with ~6,000 genetic diseases of which less than 5% have treatments. At odds with our positive long-term view of Editas and CRISPR technology are two headwinds which we see as keeping EDIT in check: (1) Over the course of the next 1-2 years Editas and its academic parters are going to engage in a substantive IP battle over CRISPR technology versus other companies, namely, Intellia and Crispr Therapeutics, which is likely to limit stock appreciation; and (2) initial clinical data is unlikely to be available until late 2017 at the earliest, limiting any significant derisking events for the platform.

CRISPR is a compelling technology and we see Editas as best positioned to realize its potential: We have high hopes that the potential for CRISPR can be translated into clinical benefit across a wide variety of diseases. Key to our optimism is that CRISPR has been used in many labs across many academic institutions with strickingly similar outcomes, suggesting that many of the key technical hurdles in small systems are understood. We believe Editas has a strong analytic process dedicated to addressing each of the main components of development. Thus, we ultimately see Editas as being successful in delivering therapeutic candidates. Importantly, for the lead clinical program in Leber's disease, we see it as the right first target given that it is relatively easier to deliver therapy to the photoreceptors, and the closed system of the eye limits potential safety risks.

IP a key area of debate over the next 1-2 years: Editas, along with its academic partners from whom it licensed its foundational IP, is currently engaged in a interference proceeding to determine which claims, if any, from its IP can stand versus the competing party (University of California and its licensees). This proceeding is likely to play out over the course of the next 1-2 years. While we expect the outcome is likely to be one which requires both sides to share IP, there are tail scenarios which could lead Editas to have no freedom to operate.

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Editas Medicine (EDIT.O, EDIT US)

Biotechnology / United States of America

Stock Rating	**Equal-weight**
Industry View	**In-Line**
Price target	**$28.00**
Shr price, close (Feb 26, 2016)	$27.49
Mkt cap, curr (mm)	$133
52-Week Range	$29.40-12.57

Fiscal Year Ending	12/14	12/15e	12/16e	12/17e
ModelWare EPS ($)	**(4.79)**	**(2.33)**	**(1.16)**	**(1.81)**
Prior ModelWare EPS ($)	**-**	**-**	**-**	**-**
P/E	**NM**	**NM**	**NM**	**NM**
Consensus EPS ($)§	**-**	**-**	**-**	**-**
Div yld (%)	-	-	-	-

Unless otherwise noted, all metrics are based on Morgan Stanley ModelWare framework
§ = Consensus data is provided by Thomson Reuters Estimates
e = Morgan Stanley Research estimates

QUARTERLY MODELWARE EPS ($)

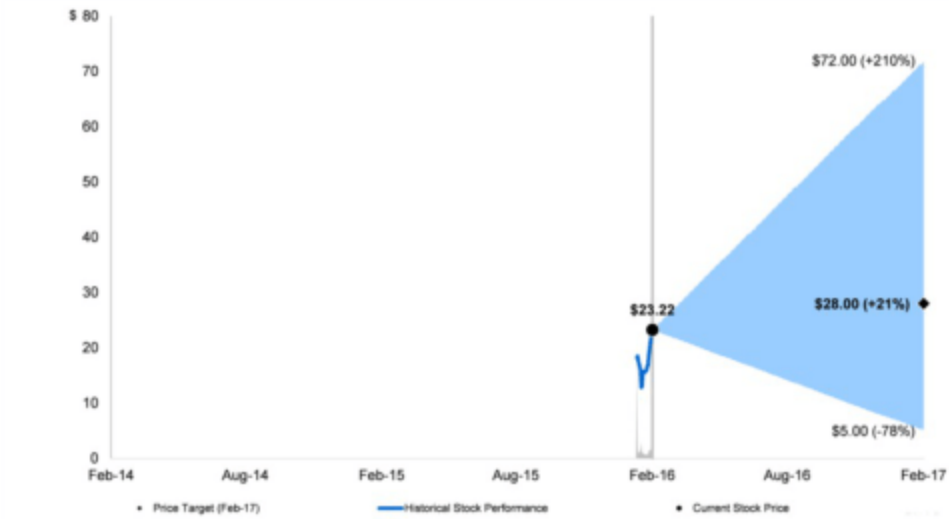
Quarter	2014	2015e Prior	2015e Current	2016e Prior	2016e Current
Q1	(1.00)	-	(0.70)a	-	(0.21)
Q2	(1.00)	-	(0.68)a	-	(0.24)
Q3	(1.36)	-	(0.69)a	-	(0.29)
Q4	(1.89)	-	(0.26)	-	(0.41)

e = Morgan Stanley Research estimates, a = Actual Company reported data

Lack of near-term clinical data and IP dispute likely to keep EDIT range bound: Despite our positive view of CRISPR, we see the lack of near-term derisking clinical data as limiting potential upside to EDIT in the near-term. Further, while we expect the outcome of the IP dispute to be some sort of cross licensing agreement similar to what we have seen with antibodies, the tail scenarios present sizable risk and thus are likely to also limit upside.

Risk Reward

Demonstration of safe administration in humans, IP resolution, and LCA10 success drive risk/reward



Source: Thomson Reuters, Morgan Stanley Research

Price Target	**$28**	Our PT is derived from a DCF that uses a 15% discount rate and a 0% terminal growth rate beyond 2032E.
Bull DCF	**$72**	**Editas is able to develop and commercialize therapies that receive widespread uptake.** The LCA10 therapy launches in the US and EU in 2024E, treats and cures ~80-90% of the addressable LCA10 population, commands premium pricing, and generates peak sales of ~$130M in both the US (2026E) and EU (2027E). Editas is also able to realize ~$6B in sales by 2032E from additional therapies in CAR-T applications, non-malignant hematology, DMD, and CF.
Base DCF	**$28**	**The LCA10 therapy proves successful, but additional therapies obtain modest success.** The LCA10 therapy launches in 2024E in the US and EU, and treats and cures ~70-80% of the addressable LCA10 population to generate ~$85M in peak sales in both the US and EU (2028E). While Editas is able to develop successful therapies for CAR-T, hematology, DMD, and CF, it achieves lower market share for total additional annual revenues of ~$3B by 2032E.
Bear DCF (Cash/share)	**$5**	**Pipeline programs fail.** Editas is not able to commercialize any therapies, and the resulting valuation is cash / share.

Investment Thesis

- We are Equal-weight Editas Medicine. The CRISPR gene editing platform has been derisked from an operational standpoint, and can be geared towards numerous disease targets. However, the delivery and long-term safety of administering clinically relevant doses in humans needs to be proven.
- Editas has a systemic, modular approach that may allow for differentiation over competitors
- The first disease being targeted (LCA10, an inherited retinal dystrophy) lowers the initial risk of proving that gene editing can work in humans, as the eye is immune privileged and provides a small, contained area in which sufficient quantities of vector can be delivered safely
- Overall, we see significant long-term potential for Editas, but remain equal-weight while initial therapies are derisked in the clinic and the IP battle plays out over the next 1-2 years.

Key Value Drivers

- Resolution to IP interference proceedings
- Progressing the LCA10 program into the clinic
- Advancing current discovery stage programs into the clinic, such as for hematologic diseases and genetic diseases of the lung & liver

Potential Catalysts

- Initiation of PhI LCA10 trial in 2017
- Interference proceedings in 2016/2017
- Entering additional therapies into the clinic in 2017/2018

Risks to Achieving Price Target

- IP outcome that limits Editas' freedom to operate
- Development risk associated with early nature of pipeline, and the timeline is long to initial data (first data in humans is expected from the LCA10 PhI study in 2017)
- Competitors that could influence investor perception of the stock

Investment Case

Summary & Conclusions

We are initiating coverage of Editas with a $28 PT and an Equal-weight rating. Our rating is based on two near-term factors at odds with our longer term view of CRISPR as a platform: (1) Over the course of the next 1-2 years Editas and its academic parters are going to engage in a substantive IP battle over CRISPR technology versus other companies, namely, Intellia and Crispr Therapeutics which is likely to limit stock appreciation; and (2) initial clinical data is unlikely to be available until late 2017 at the earliest, limiting any significant derisking events for the platform. Thus, while we continue to view CRISPR as one of the more compelling next-generation technologies to address a wide variety of disease targets, we think the stock is likely to remain range bound ahead of clarity on both IP and clinical activity.

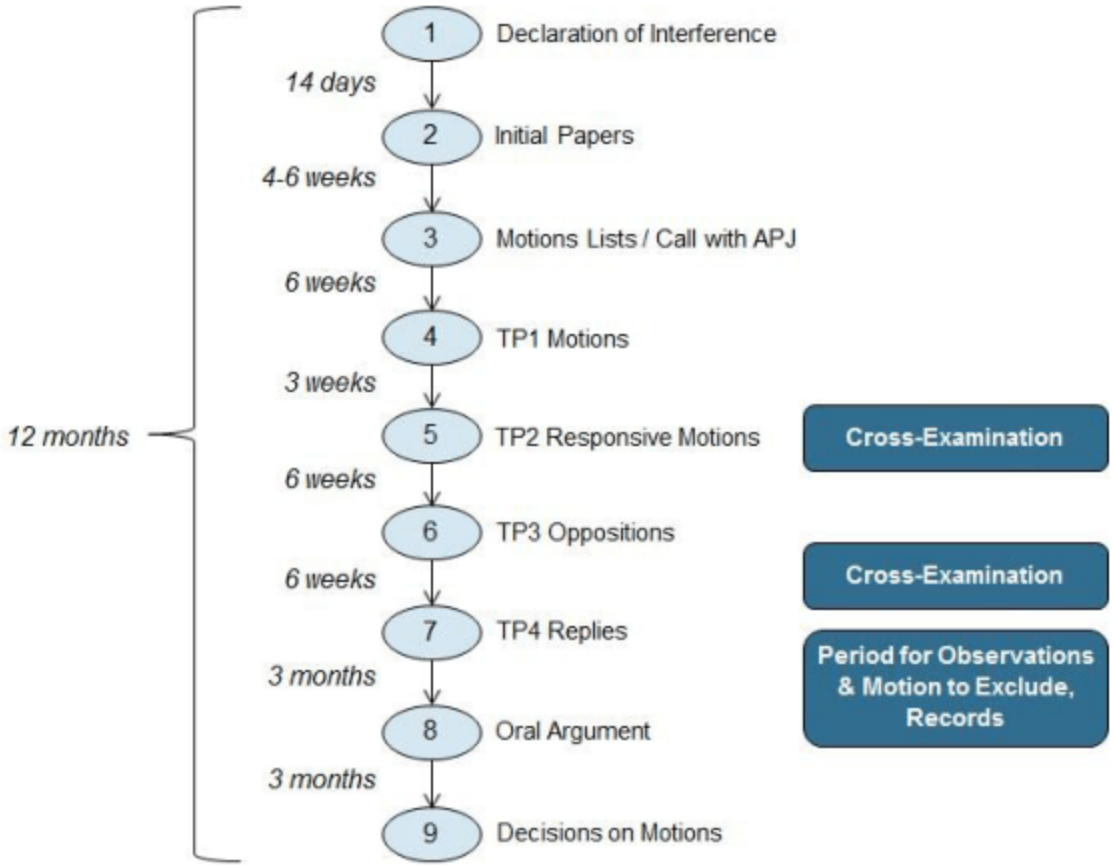
Note: The authors of this material are not acting in the capacity of attorneys, nor do they hold themselves out as such. This material is not intended as either a legal opinion or legal advice. The information provided herein does not provide all possible outcomes or the probabilities of any outcomes. The result of any legal dispute or controversy is dependent on a variety of factors, including but not limited to, the parties' historical relationship, laws pertaining to the case, relative litigation talent, trial location, jury composition, and judge composition. Investors should contact their legal advisor about any issue of law relating to the subject matter of this material.

Key Investment Points

1. CRISPR is a compelling technology and we believe Editas is best suited to translate the technology into therapeutic benefit - We are positive about the potential for CRISPR to be translated into clinical benefit across a wide variety of diseases. Key to our optimism is that CRISPR has been used in many labs across many academic institutions with similar outcomes, suggesting that many of the key technical hurdles in small systems are understood. That said, there are still many key risks and challenges that need to be overcome including identifying efficient edits for each target, appropriate delivery to the tissue, expanding the platform's applicability across a range of different types and kinds of gene edits and various other engineering challenges. However, we believe Editas has a strong analytic process dedicated to addressing each of the main components of development. Thus, we ultimately see Editas as being successful in delivering therapeutic candidates.

2. IP battle will remain an overhang, but we continue to see cross licensing as the most probable outcome - The IP in the CRISPR space is complicated, varied and nuanced. Thus, it is not surprising that various academic institutions believe they each have foundational IP. Early in January 2016, foundational patents which Editas has licensed from Harvard, the Broad Institute and MIT were named in a patent interference proceeding with the University of California, University of Vienna, and Emanuel Charpentier. These patents are held by Caribou Biosciences and licensed to Intellia. Crispr Therapeutics also has rights to the same IP as Intellia though the second scientific founder, Dr. Doudna. The debate between the parties, which we discuss further in this report, is whether the initial discovery of CRISPR in prokaryotic cells is easily translated into mammalian cells (Broad was first to discover in the latter category while UoC in the former). Given the complexity of the IP – 11 patents are named in the interference – we believe a plausible outcome is one where certain claims in Editas IP are narrowed (right now Editas is the only company with granted IP) and certain claims in the Caribou IP are granted, but overall both parties would need rights to the other party, similar to the situation which developed from the foundational antibody IP. We assume modest royalties which generally cancel each other out. Nonetheless, the uncertainty created by this situation – including the fact that one outcome could be that Editas would have no freedom to operate if all its IP was overturned – is likely to keep EDIT range bound as the interference proceeds.

Exhibit 1: Indicative Timeline for IP Interference Proceedings



Source: Company Data, Morgan Stanley Research

3. Clinical data is the key upside driver, but is not due until late 2017 at the earliest- Editas and its CRISPR platform has the ability to target a wide variety of diseases with over 6,000 diseases caused by a genetic mutation and 95% having no approved therapy. Key targets include muscular dystrophy, cystic fibrosis, various malignant and non-malignant hematologic diseases and other liver direct targets. Thus, the universe of available diseases is large; however, all of these programs are currently in preclinical development and unlikely to move into the clinic in the near-term. The most advanced program is for a eye disease called Leber congenital amaurosis (LCA), which impacts a patient's retina and leads to blindness. This program is likely to enter clinical testing in 2017. Importantly, we view this as an appropriate target for an initial clinical program. Based on the literature ~10-20% of the photoreceptors in the eye need to be edited to produce a therapeutic response (i.e., maintenance of some vision) and since the eye is a closed body, both delivery is likely achievable and off target toxicity is likely to be limited. Thus, we see the overall risk to be more modest with the LCA10 program. Despite our positive view of this program, the timeline is likely to limit stock appreciation given clinical data is unlikely until late 2017.

4.Business development has the potential to be a near-term upside driver - Editas has already completed one licensing deal with Juno in the CAR-T space. While we think that partnership can drive value for both companies, the timelines are not near-term. Further, we have seen other companies like Crispr Therapeutics strike partnership deals with Vertex and Bayer. Thus, there is clearly interest in CRISPR therapies by larger companies. Unlike Crispr Therapeutics, we believe Editas' management strategy is better suited to preserving shareholder value and would expect management to target deals where the assets are well defined (i.e., no open-ended target deals) and either the time to market or the basic knowledge in the therapeutic category (i.e., neurology) could be accelerated by the larger party. Thus, we do expect business development and expect it to be positive for EDIT, but the timing is hard to predict.

Exhibit 2: Correction of mRNA Expression in Cells from LCA10 Patients

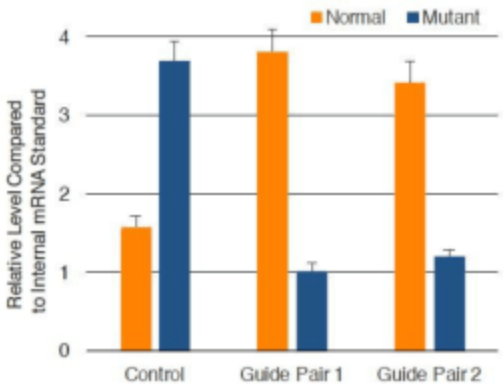


Exhibit 3: Editas Pipeline

Program	Target Gene	Stage
Leber Congenital Amaurosis 10	CEP290	Discovery, IND enabling studies in 2016, Ph1 start in 2017
Genetic and Infectious Diseases of the Eye	Multiple	Discovery
Gene Editing in T-Cells to Treat Cancer	Multiple	Discovery
Non-Malignant Hematologic Diseases	Multiple	Discovery
Genetic Diseases of Muscle	Multiple	Discovery
Genetic Diseases of the Lung	Multiple	Discovery
Genetic and Infectious Diseases of the Liver	Multiple	Discovery

Source: Company Data, Morgan Stanley Research

Source: Company Data

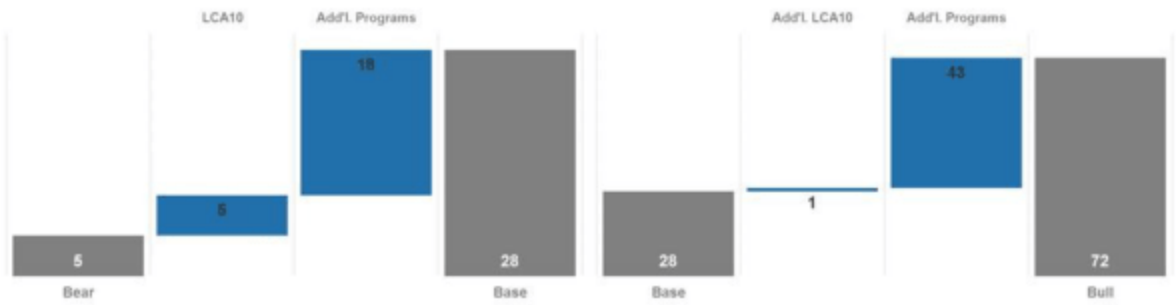
Exhibit 4: Recent Gene Editing Deals

Deal					*Payments to Gene Editing Partner*			
Biopharma Partner	Gene Editing Partner	Collaboration Focus	Announcement Date	Duration of Collaboration	Upfront	Potential Milestones	Royalty Eligibility?	Other / Comments
Bayer	CRISPR Therapeutics	JV to address blood disorders, blindness, and congential heart disease	12/21/2015	Bayer invests $300M+ to JV over five years	NA	NA	NA	(1) Bayer invests $300M+ in newly created JV owned 50/50 by Bayer and CRISPR Therapeutics (2) Bayer acquires $35M equity in CRISPR Therapeutics
Vertex	CRISPR Therapeutics	Initial focus on cystic fibrosis and sickle cell disease, along with additional diseases	10/26/2015	Four years	$105M	$420M	Yes	(1) $105M upfront consists of $75M cash and $30M equity (2) Vertex will fund 100% of development costs for treatments in-licensed by Vertex
Juno	Editas	CAR-T and TCR cancer therapies	5/27/2015	Five years	$25M	$700M	Yes, tiered royalties on product sales	$22M in research support over five year collaboration
Novartis	Intellia	Engineering CAR-Ts and hematopoietic stem cells	1/7/2015	Five years	Not disclosed	Not disclosed	Yes	(1) R&D funding (2) Novartis increases original equity stake in Intellia

Source: Company Data, Morgan Stanley Research

5.Given the early stage of development Editas is tough to value, but we think a unique approach is warranted - We value Editas using a two pronged approach. As is typical, we value the defined clinical programs - in this case LCA10 - and assign a value. However, separately, we also try to value the potential upside associated with the platform. Here our approach is unique as we take our revenue models for DMD, Cystic Fibrosis, CAR-T and non-malignant hematology and assume Editas could penetrate that market starting in 2025 and reach peak share of 30% of that market by 2030. We than assume a 10% probability of success as a way to gauge the potential of the platform across a wide variety of targets and diseases.

Exhibit 5: Bear to Bull Case Bridge for Editas Valuation



Source: Morgan Stanley Research

Key Upcoming Catalysts

The key upcoming catalysts for Editas include updates to the interference proceeding, updated preclinical data at the ASGCT conference in May and an IND filing on its first clinical candidate for LCA10. Obviously, general developments in the CRISPR space are likely to occur as well and could have an impact on Editas.

Exhibit 6: Editas Catalyst Calendar

Milestones	Timing
Updated preclinical data at ASGCT 2016	May 4-7, 2016
Potential new business development	2016
Potential interference proceedings and updates	2016/2017
Initiation of Phase I LCA10 study	2017
Additional therapies enter the clinic	2017/2018
End of Juno collaboration	2020

Source: Company Data, Morgan Stanley Research

Valuation

Exhibit 7: DCF drives valuation

(USD in millions)	2015E	2016E	2017E	2018E	2019E	2020E	2021E	2022E	2023E	2024E	2025E	2026E	2027E	2028E	2029E	2030E	2031E	2032E	Terminal 2033E
Free cash flow (FCF)	($43)	($45)	($69)	($91)	($110)	($122)	($134)	($151)	($173)	($158)	($27)	$102	$266	$484	$667	$873	$1,117	$1,404	$9,158
Present value of FCF	($40)	($41)	($60)	($69)	($72)	($70)	($67)	($65)	($65)	($52)	($8)	$45	$57	$91	$108	$123	$137	$150	$870

Source: Company Data, Morgan Stanley Research

Sum of Discounted CF ($M)	$1,009
Net Cash	$203
Equity Value	$1,213
Equity Value Per Share	$28
Discount Rate	15%
Terminal Growth Rate	0%
Time of Valuation	2016
Shares Outstanding (millions)	43

Our $28 price target includes ~$3.3B in peak (2032E) global revenue. We derive our price target from a discounted cash flows (DCF) analysis that uses a WACC of 15.0% and a terminal growth rate of 0%.

Valuation Methodology: We prefer the use of a DCF analysis to value biotechnology companies. Given the defined patent life for each product, we believe a DCF fully captures both the upfront investment period as well as the long-term earnings power. While investors do look at biotechnology on a multiples basis (P/E), we prefer a DCF as it is more rigorous and requires more explicit assumptions about the long-term prospects of a company.

Discount Rate: We use a 10% discount rate for all commercial companies, a 12.5% rate for companies with randomized PhII data and a 15% rate for all development stage companies. Given the stage of development we use a 15.0% discount rate for EDIT.

Terminal Growth Rate: We model explicit revenues through 2032E with a 0% terminal growth rate.

Revenue: We model 5-25% penetration of the LCA10 market in the US and 3-17% in the EU starting in 2024. We assume $1M pricing in the US, increasing 1.5% annually. We assume a $900k price in the EU, increasing 1% annually. For other products we assume a blend of revenues from CAR-T, non-malignant hematology, DMD, and CF with the company capturing a small fraction of sales and having a 10% probability of success. Total estimated peak sales (2032) are ~$3.3B.

Economics: Editas maintains worldwide commercial rights to many of its product candidates, except for their CAR-T program. We expect the company to retain global rights and launch their products themselves.

COGS: We model COGS as a continuous 17%.

Operating Expenses: We assume R&D of $25M in 2016E growing to ~$80M in 2021E and ~$250M in 2032E. R&D growth is expected to be aggressive in the next few years to account for the simultaneous development of several products. We assume SG&A of ~$25M in 2016E, growing to ~$56M in 2021E and ~$210M by 2032E, assuming product launches starting in 2024E.

Key Risks To Our Price Target Include: (1) Lack of freedom to operate driven by losses int he interference proceeding around CRISPR IP; (2) Inability to deliver CRSIPR candidates to the correct tissue or inability to achieve high editing efficiency; (3) Lack of efficacy with initial clinical data or unknown safety.

Debate 1 - What is CRISPR/Cas9?

Overview: CRISPR is a new technology that may not be familiar to many investors. While the new wave of genetherapy companies - where a gene sequence is inserted into a viral carrier (typically lenti or AAV) and then incorporated into the body's genes - has educated investors on that technique, many have heard about CRISPR, but do not know all of the details. CRISPR is a protein-RNA complex where guide RNAs locate a specific gene sequence and then allow cas9 nucleases to "edit" that sequence. It is a precise way to edit a specific gene sequence, potentially curing the patient of the disease caused by the genetic defect. Significant academic success has been made with the CRISPR/Cas9 system, though all the work has been completed in animal models. The first in-human testing is to be completed by Editas and its competing commercial entities.

Street's take: Overall, we believe consensus recognizes that CRISPR is a significant new technology that could have a large impact on the treatment of genetic diseases. We believe most investors understand the significant potential of CRISPR and the debate centers around how effectively it can be implemented, if the technology is ready to be used in humans and if it is derisked enough yet for an equity investment. As is to be expected, some investors see near-term potential while others would prefer to wait for greater clinical experience.

Our take: We believe CRISPR is likely to play a major role in gene repair and modification long-term. While we are not entirely sure how long and what the path to clear therapeutic effect will involve, we do believe CRISPR as a technology is proven from a technical perspective and now needs to be translated into humans. Thus, we see great promise in the platform and believe CRISPR has the best chance to create durable, functional cures of many genetic diseases.

Gene therapy vs Gene editing?

Gene therapy differs from gene editing in both the approach and the possible results seen. Gene therapy generally involves the addition of a new gene to a genome. These new genes can be to replace a defective gene or to add a new gene. The original genes remain intact. Gene editing involves changing the genome, which can involve the addition, deletion, or substitution of components of the genome. CRISPR/Cas9 is not a genetherapy and is a gene editing platform.

What is CRISPR/Cas9?

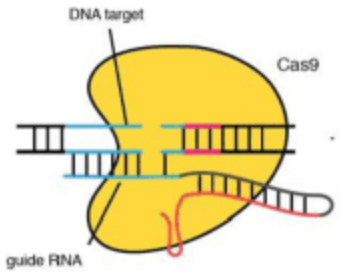
CRISPR/Cas9 is a novel method of genetic engineering that uses guide RNA to edit DNA by allowing the enzyme Cas9 to cut and insert the desired genome sequences.

The CRISPR/Cas system is formed from CRISPR arrays and CAS genes. CRISPR arrays are clustered, regularly interspaced short palindromic repeats found in certain prokaryotic genomes. CRISPR arrays involve sections of repeated base pair sequence separated by non-repeating spacer sequences. Both the repeated sequences and the spaces come in a large number of variations. Many CRISPR-genes also contain domains associated with DNA manipulation and many of the spacer sequences contained plasmid or phage-derived DNA.

CAS, or CRISPR-associated genes, are almost always found adjacent to the repeat arrays. CAS genes come in a variety of subtypes. The CAS9 subtype gene encodes the RNA-guided endonuclease Cas9 which can cleave double stranded DNA. CRISPR RNA (crRNAs) guides with Cas9 proteins and can lead to cleavage of specific sites on DNA and the introduction of templates for gene insertion, as demonstrated in the illustration below.

niques, CRISPR/Cas9 has the advantage of being cheaper, faster, and easier to use than other techniques. The system is easy to configure between species and allows for an increased precision of insertion. Overall the CRISPR/Cas9 system allows for greater control than usual gene augmentation from gene therapy.

Exhibit 8: Illustration of Gene Editing Using CRISPR/Cas9



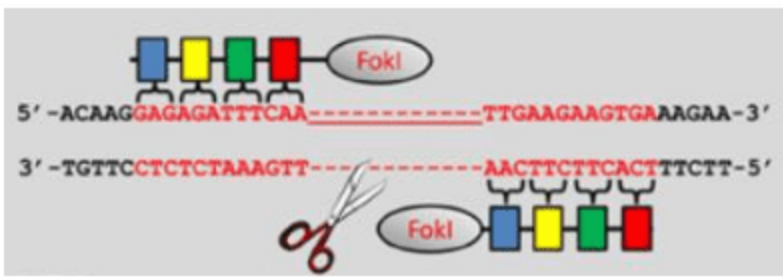
Source: Company Data

The CRISPR/Cas9 technology has seen rapid uptake in a number of academic laboratory groups due to the relative low cost and ease of use. The technology was initially designed for prokaryotic cells but was later adapted to eukaryotic cells, and has since been demonstrated to work in Mammalian cells. A useful review of the history of CRISPR by Lander can be found in *Cell 164*, January 14, 2016.

What other approaches are available for gene editing?

Zinc finger nucleases – These are restriction enzymes designed to cleave specific target sequences on DNA. They involve a zinc finger DNA binding domain with site specificity and a DNA-cleavage domain for cutting the phosphodiester bone between nucleic acids in DNA strands. By taking advantage of a cell's intrinsic DNA repair mechanisms, zinc finger nucleases can be used for gene editing. Zinc finger nucleases are being commercialized by Sangamo Biosciences (not covered).

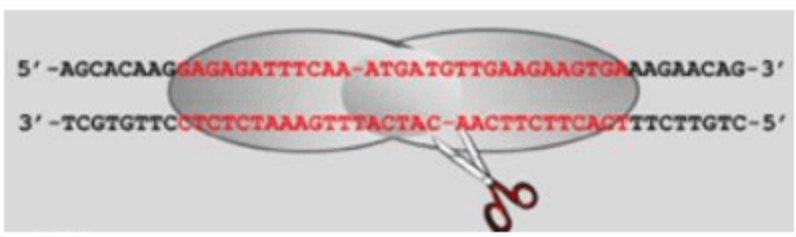
Exhibit 9: Illustration of Gene Editing Using Zinc Finger Nucleases (Codon Identification)



Source: Front. Physiol., 21 April 2014; "Emerging gene editing strategies for Duchenne muscular dystrophy targeting stem cells," Carmen Bertoni; Department of Neurology, David Geffen School of Medicine, University of California Los Angeles

Engineered meganucleases – Meganucleases are naturally occurring proteins that can recognize and cleave specific DNA sequences. They have a DNA recognition sequence that can be modified, thus allowing for engineered meganucleases that can target specific DNA sequences of genetic disorders.

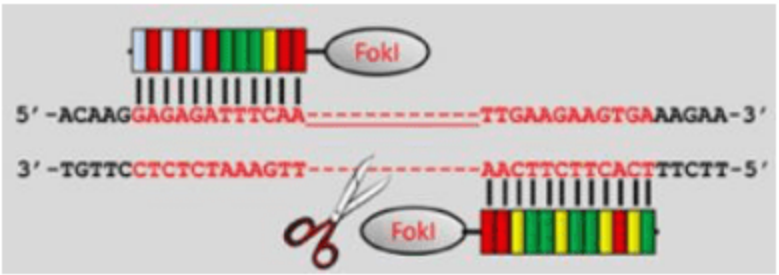
Exhibit 10: Illustration of Gene Editing Using Meganucleases



Source: Front. Physiol., 21 April 2014; "Emerging gene editing strategies for Duchenne muscular dystrophy targeting stem cells," Carmen Bertoni; Department of Neurology, David Geffen School of Medicine, University of California Los Angeles

Transcription-activator like effector nucleases (TALENs) – Transcription activator-like effectors (TALEs) are proteins that bind promoter genes and can enhance gene expression. TALEs have a central repeat domain that confers the ability of TALEs to recognize specific DNA sequences along genomes. TALENs are engineered proteins that involve fusing a TALE and a DNA cleavage domain.

Exhibit 11: Illustration of Gene Editing Using TALENs (Nucleotide Identification)



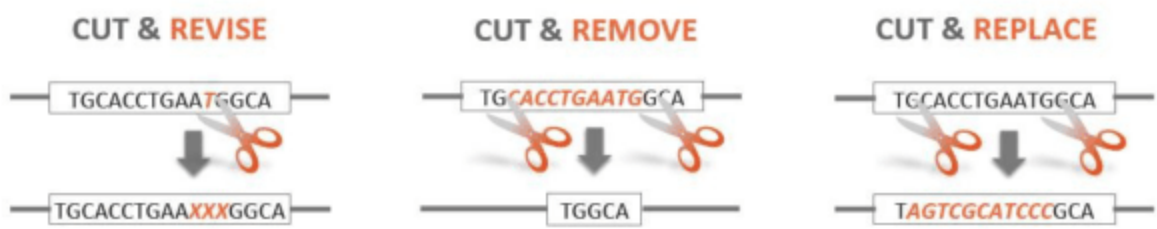
Source: Front. Physiol., 21 April 2014; "Emerging gene editing strategies for Duchenne muscular dystrophy targeting stem cells," Carmen Bertoni; Department of Neurology, David Geffen School of Medicine, University of California Los Angeles

What is Editas's approach to gene editing?

Editas's approach to gene editing has four components: 1) nuclease engineering; 2) delivery; 3) control and specificity; and 4) directed editing. Each of these components can be independently optimized in order to develop the best therapeutic candidate. We see Editas as using a comprehensive analytical approach to systematically build a library of CRISPR/Cas9 for a variety of editing approaches. We outline the key features of Editas' approach below.

1. Nuclease Engineering - The CRISPR/Cas9 system involves both a Cas9 protein and an RNA guide molecule. Both of these components can be tailored to allow Editas' gene editing platform to comprehensively target a wide variety of diseases. Editas is developing Cas9 variants tailored to specific genetic defects, and is also making targeted gRNA chemical and structural modifications to build a library of guide RNAs that will allow for enhanced targeting. Additionally, Editas is also using iterative *in silico* design and high-throughput screening to identify optimal Cas9 / guide RNA combinations. This approach could allow Editas to perform gene editing through various methodologies to address most disease-causing mutations.

Exhibit 12: Approaches Available to Gene Editing for Editas



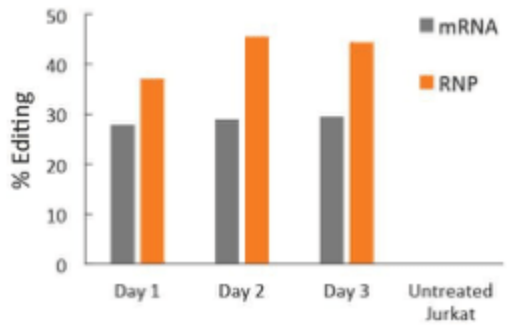
Source: Company Data

2. Delivery – Different disease indications will involve different cell types and tissue structures and thus delivery mechanisms will need to be specifically designed for each disease type. Editas's CRISRP/Cas9 system is adaptable to different delivery modalities, and the company intends to use existing delivery technologies for *in vivo* and *ex vivo* delivery through modalities including viral vectors, nanoparticles, and electroporation. Preclinical data for both *ex vivo* and *in vivo* genome editing has provided encouraging early results, as demonstrated below. Next-generation delivery methods will be studied based on product needs going forward.

3. Control & Specificity – One of the largest advantages of gene editing techniques over conventional gene therapy techniques is the high level of specificity available when altering, deleting, or inserting new genetic material. The specific DNA cut sites must be optimized in order to deliver optimal therapy. Cellular exposure to the Cas9-guide/RNA complex can also affect the outcome and can be optimized to provide maximal benefit.

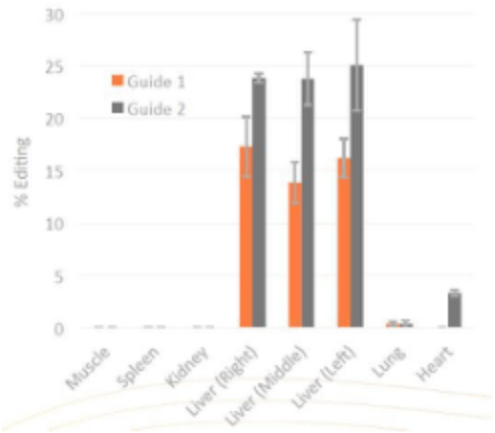
Editas is exploring codon optimization for Cas9, to be able to identify the ideal codon set for each tissue. The company is also working to identify tissue-specific promoters for both guide RNAs and Cas9 proteins. Specific methodologies the company is exploring to control the editing process include self-targeting gRNAs to turn expression off, developing small molecule modulators of Cas9, and incorporating functional motifs and

Exhibit 14: Ex vivo: mRNA & RNP Delivery of PD-1 Targeted Cas9 in T-Cells



Source: Company Data

Exhibit 13: In vivo: Factor VII Gene Knockdown in the Liver with AAV



Source: Company Data

developing variants to control cellular Cas9 degradation.

4. Directed editing – There are several mechanisms by which the CRISPR/Cas9 system and the cell's intrinsic repair systems can work together to cut and repair the target cell's DNA. Non-homologous end joining (NHEJ) is a method of DNA repair that occurs in the absence of a DNA template for each cell to copy that leads to small insertions and deletions. This is the best option when treatment requires the deletion of gene segments. The difficulty of using this mechanism increases as the length of DNA requiring deletion increases. Homologous directed repair (HDR) is a method of DNA repair that occurs in the presence of a DNA template. This method leads to the replacement of defective sequences with functional ones. This technique is the best option for inserting new genes. Editas is studying the impact that Cas9 variants and different cutting approaches have on the NHEJ and HDR repair pathways, and is working to enhance the efficiency of HDR by using donor DNA modifications.

Important Papers in CRISPR/Cas9's development

The development of CRISPR/Cas9 for Human Cells

"Multiplex Genome Engineering Using CRISPR/Cas Systems" - L Cong, et al. In Science 2013

This paper demonstrates that RNA-guided Cas9 nucleases can be engineered that precisely target genomic loci in mammalian cells and cause double stranded breaks in mammalian chromosomes.

"RNA-Guided Human Genome Engineering via Cas9" - P Mali, et al. In Science 2013

This paper focuses on the development and testing of engineered CRISPR/Cas9 with a custom guide RNA in human cells. This paper established that this technology was capable of human genome engineering. These tests were done in vitro.

Improving Delivery

"Cationic lipid-mediated delivery of proteins enables efficient protein-based genome editing in vitro and in vivo" - J Zuris, et al. In Nature Biotechnology 2014

This paper discusses a delivery system that can be applied to the CRISPR/Cas9 system proteins. Experiments were performed both in vitro and in vivo that demonstrated that the Cas9 system could be delivered and would

modify the target genome.

"In vivo genome editing using Staphylococcus aureus Cas9" - F Ran et al. In Nature 2015

Earlier studies of Cas9 mainly relied on Streptococcus pyogenes derived Cas9s (SpCas9). These are larger proteins and limit the useful applications. This paper demonstrated that the significantly smaller Cas9 from Staphylococcus aureus can achieve similar efficacies to SpCas9, while maintaining specificity and efficacy.

Improving Characterization

"GUIDE-seq enables genome-wide profiling of off-target cleavage by CRISPR-Cas nucleases" - S Tsai et al. In Nature Biotechnology 2014

This paper highlights a detection system for identifying off-target DNA double strand breaks caused by CRISPR/Cas9 nucleases. Experiments with 13 CRISPR systems in 2 human cell lines demonstrated a high level of variability in off-target activities that were not detected by other computational and experimental detection methods. It was also shown that truncated RNA guides led to a reduction in off target double strand breaks.

"In vivo interrogation of gene function in the mammalian brain using CRISPR-Cas9" - L Swiech et al. In Nature Biotechnology 2014

In this study an AAV vector was used to deliver CRISPR/Cas9 derived from Streptococcus pyogenes to adult mouse brain cells targeting specific genes. The genome editing resulted in biochemical, genetic, electrophysical, and behavioral changes.

Improving Specificity

"Double Nicking by RNA-Guided CRISPR Cas9 for Enhanced Genome Editing Specificity" - F Ran et al. In Cell 2013

This paper discusses the use of paired RNA guides to introduce double strand breaks into target DNA. By using two guides with known spacing the specificity of the DNA cuts drastically increases. These experiments were performed in mouse zygotes.

"Improving CRISPR-Cas nuclease specificity using truncated guide RNASs" - Y Fu et al. In Nature Biotechnology 2014

In this paper, researchers compare guide RNAs with truncated guide RNAs in terms of off-target effects. They demonstrate that the truncated guide RNAs have as much as a 5000-fold decrease in undesired mutagenesis without affecting on-target editing.

"Fusion of catalytically inactive Cas9 to FokI nuclease improves the specificity of genome modification" - J Guilinger et al. In Nature Biotechnology 2014

The fusion of inactive Cas9 and FokI nuclease improves DNA cleavage specificity. These fused Cas9 complexes had a significantly higher specificity than WT Cas9 proteins.

"Dimeric CRISPR RNA-guided FokI nucleases for highly specific genome editing" - S Tsai et al. In Nature Biotechnology 2014

This paper describes dimeric RNA-guided FokI nucleases that have high efficacy and specificity in their DNA targets by recognizing extended genetic sequences. This system uses two guide RNAs with fixed and known spacing to ensure specificity.

"Engineered CRISPR-Cas9 nucleases with altered PAM specificities" - B Kleinstiver et al. In Nature 2015

The range of sequences that can be identified with a Cas9 protein is determined by the specific protospacer adjacent motifs (PAM) on the targeted DNA. This paper demonstrates that the specificity of the CRISPR/Cas9 system for PAMs can be altered so that a wider range of targets is available to therapy.

"High Fidelity CRISPR-Cas9 nucleases with no detectable genome-wide off target effects" - B Kleinstiver et al. In Nature 2015

This paper describes a variant of SpCas9 that minimized non-specific DNA contacts that results in the retention of on-target activity while rendering nearly all off target effects undetectable.

"Rationally engineered Cas9 nucleases with improved specificity" - I Slaymaker et al in Science 2015

In this paper, structure-guided protein engineering is used to alter and improve the specificity of SpCas9. These altered Cas9 proteins reduced off-target effects while still maintaining on-target editing.

Key Academic Institutions with CRISPR/Cas9 Progams

The development of CRISPR/Cas9 technology provides a model for what can be done through incremental progress in science. In the late 1980s/early 1990s, Dr. Francisco Mojica, a post doctoral student at the University of Alicante in Spain, noticed a unique/repeating genetic sequence in Haloferax mediterranei. It altered between a repeat sequence of 30 base pairs followed by ~36 spacer base nucleotides. Dr. Mojica initially called the finding short regularly spaced repeats (SRSRs), eventually renaming it to clustered regularly interspaced palindromic repeats or CRISPR. This finding from a microbe usually found in salty environments, and noted for its ability to tolerate excessively high salinity, marks the beginning of an incremental set of advances that eventually led to the gene editing technologies being pursued by Editas and its competitors.

Post the early discovery in Spain, there was a great deal of work that needed to be done to determine the purpose (if any) of these repeat sequences. After years of work, Dr. Mojica noted that these spacers were noted in E. coli strains with bacterial resistance leading to the hypothesis that CRISPR's purpose was part of the adaptive immune system offering a way for cells to adapt to their environment. While Dr. Mojica made some early findings, many others contributed new discoveries at a rapid pace. From Horvavth, Barrangou and Moineau who discovered that Cas nucleases contributed to this adaptive response to Marrafani and Sontheimer, who discovered that CRISPR specifically targeted DNA. As more incremental advances occurred it became increasingly apparent that not only is CRISPR, and in particular the Cas9 nucleotide, an important component of the adaptive immune system but also that it could be utilized as a gene editing tool. Such a tool would have immense potential for treating a massive array of diseases and disorders. Below we outline the key academic institutions who claim to have IP for CRISPR/Cas9 and their corporate affiliation.

Broad Institute, Massachusetts Institute of Technology, Harvard University

- **Contribution:** Dr. Feng Zhang, one of the founders of Editas and a principal scientist in the intellectual property dispute, conducted his research at the Broad Institute/MIT. Zhang's team examined CRISPR-Cas9 and in partnership with Dr. George Church from Harvard University learned that the nuclease Cas9 could be used to cut DNA in a highly specific location. This work was published in January 2013 in the journal Science. Dr. Zhang's team demonstrated that the cut DNA was replaceable with another piece of DNA, changing the overall sequence of the gene. Zhang and team also discovered Cpf1, which could also have similar application with the advantage of being a smaller protein than Cas9. Dr. George Church of Harvard University, and also a Broad Institute researcher, conducted work concurrently with Dr. Zhang and reported similar findings. Dr. Church's work was also published in the January 2013 issue of Science. It is important to note that both Church and Zhang also were able to translate the use of CRISPR/Cas9 into mammalian cells.
- **Intellectual Property Status:** The Broad Institute filed for patents through the Prioritized Patent Examination Program, a fast-track review program. In December 2015, only six months after application, the US Patent and Trademark Office (USPTO) issued 23 patents

with respect to CRISPR/Cas9, 13 of which were issued to the Broad Institute, MIT and Dr. Feng Zhang. Harvard University was issued 4 patents relating to CRISPR.

- **Affiliation:** Editas Therapeutics

University of California, Berkeley, University of Vienna

- **Contribution:** Dr. Jennifer Doudna of the University of California at Berkeley partnered with Dr. Emmanuelle Charpentier from the University of Vienna. Much of their work together was focused on Cas9. They made similar discoveries as Dr. Zhang and Dr. Church's team, demonstrating that the enzyme could be used to cut DNA. Dr. Doudna was one of the original founders of Editas; although, she left the company to start Caribou Biosciences, a company that eventually cofounded Intellia Therapeutics.
- **Intellectual Property Status:** Doudna and Charpentier submitted a patent application for the UC Berkeley and the University of Vienna to the USPTO seven months ahead of Zhang. However, the application was submitted via the normal track as opposed to the fast tracked Zhang application. As such, the Broad Institute teams were granted broad Cas9 patents. UC Berkeley & University of Vienna initiated multiple Suggestions of Interference proceedings to challenge the Broad Institute patents.
- **Affiliation:** Intellia Therapeutics and CRISPR Therapeutics are affiliated with Dr. Doudna and Dr. Charpentier, respectively.

University of Vilnius

- **Contribution:** Dr. Virginijus Siksnys of the University of Vilnius also concurrently conducted and published work evaluating Cas9 activity. In this work, it was demonstrated that Cas9 could be targeted to cut double stranded DNA exactly three nucleotides from the protospacer adjacent motif (PAM) sequence. Dr. Siksnys submitted his work for publication, a month before the Doudna/Charpentier paper had been published (after it was fast-tracked through *Science's* review process)
- **Intellectual Property Status:** Dr. Siksnys filed patent application in March of 2012 based on this work.
- **Affiliation:** Dupont for agricultural purposes.

The Rockefeller University

- **Contribution:** Dr. Luciano Marraffini is affiliated with the Rockefeller University. In his work he determined that CRISPR specifically targets DNA. In his work, he concluded that CRISPR was a programmable restriction enzyme that could potentially be employed as a gene editing technology.
- **Intellectual Propery Status:** The Rockefeller University is a joint patent applicant on certain patent applications along with the Broad Institute.
- **Affiliation:** Intellia Therapeutics

ToolGen, Inc.

- **Contribution:** Affiliated with Seoul National University in South Korea, ToolGen has developed genetic tools based on zinc finger engineering technology, with technology ultimately evolving toward the use of Cas9 nucleases. The company has also refined a process called double-nicking approach with zinc finger nucleases to clip the DNA. The belief

is that this will reduce off-target modification.

- **Intellectual Propery Status:** ToolGen has filed Suggestions of Interference claims that two of their patents interfere with five Broad Institute patents. UC Berkeley has filed a similar claim on these five Broad Institute patents.
- **Affiliation:** Thermo Fisher Scientific

Exhibit 15: Various Academic Institutions and Players With CRISPR/Cas9 Programs

Academic institution	Contribution	IP Status	Affiliation
Massachussetts Institute of Technology (MIT) - Broad Institute	Dr. Zhang led one effort to develop Cas9 for the purpose of gene editing. Granted initial patents in large part due to decision to apply through USPTO Prioritized Patent Examination Program, leading to 6-month review process.	First to be granted patents for CRISPR Cas9 gene editing technology in December 2015.	Editas
Harvard University - Broad Institute	Dr. Church of Harvard and Broad Institute conducted work on Cas9 concurrently with Dr. Zhang.	Granted multiple initial patents in tandem with MIT - Broad Institute.	Editas
UC Berkeley	Dr. Doudna conducted similar work on Cas9 for gene editing in unrelated effort. An original founder of Editas, left to form Caribou Biosciences which co-founded Intellia.	Applied for Cas9 patents 7 months ahead of Zhang's team. However, fell behind because Zhang's fast-tracked process. Filed multiple Suggestions of Interference claims.	Intellia
University of Vienna	Dr. Charpentier worked in partnership with Dr. Doudna in Cas9 research. She is a founder of CRISPR Therapeutics.	Univeristy of Vienna was named as part of UC Berkely's patent application and is included in Suggestion of Interference claims.	Intellia
University of Vilnius	Dr. Siksnys of the University of Vilnius showed that Cas9 could be targeted to cut double stranded DNA exactly three nucleotides from the protospacer adjacent motif (PAM) sequence. His work was submitted for publication 1-month before Doudna/Charpentier's efforts were published.	Filed for patents in 2012.	Dupont
The Rockefeller University	Dr. Marrafini conducted much work on CRISPR, being one of the first to determine it actually targeted DNA and suggesting it could be a gene editing technology.	Rockefeller is named on five Broad Institute patents. Though they received no rights as part of Broad's license agreement with Editas.	Intellia
ToolGen, Inc.	A Korean company affiliated with Seoul National University that used zinc finger technology in CRISPR research, moving eventually toward Cas9. Employs double-nicking to potentially reduce off target effects.	Initiated Suggestion of Interference proceeding regarding five Broad Institute patents.	Thermo Fisher Scientific

Source: Cell 164, January 14, 2016 and Morgan Stanley

Debate 2 - What Is Going On With IP And What Potentially Can be a Plausible Outcome?

Overview: In early January, an interference proceeding was declared between the Broad Institute/Harvard/MIT (from whom Editas has licensed its IP) and University of California, University of Vienna and Emmanuelle Charpentier (from whom Intellia and Crispr Therapeutics have licensed IP). At the core of the debate is who was first to invent the use of CRISPR/Cas9 in eukaryotic cells (particularly mammalian cells). The Broad has 12 issued patents named in the interference proceeding versus a yet to be issued University of California patent. Because California claims to have invented first it has been named the senior party while the Broad the junior party, though those designations could change. The total interference proceeding is expected to play out over the next two years. Potential outcomes could be that the Patent Trial and Appeal Board (PTAB) invalidates the Broad's IP, leaving Editas without freedom to operate, that the PTAB does not award a patent to the University of California, meaning Editas has the only granted IP for CRISPR/Cas9, or a limiting of claims for both parties with issued IP for both sides. An appeal is also possible after the initial ruling.

Street's take: Investors have seen a few interference proceedings before, namely, Gilead versus Idenix for sofosbuvir and Biogen versus Forward Pharma related to Tecfidera IP. However, neither case has been potentially so central to the investment debate on the stock. Thus, for many investors this is an area they find hard to completely derisk. Despite that, we believe the prevailing opinion is either to invest broadly across the CRISPR/Cas9 space, thereby having investments in both parties or assuming, as do we, that given the breadth of the Editas IP, while some IP may be narrowed, the likelihood that all IP will fall is low.

Our take: We believe a plausible outcome of the interference could be that both parties would end up with issued IP for CRISPR/Cas9, requiring both parties to engage in cross licensing deals. Our base case assumption is that these deals would end up being single digit royalties (one side may be slightly higher than the other), such that the net impact to the "losing" party would be a low single digit royalty to the other side. Thus, our long-term view is that IP is unlikely to have a major impact on Editas. However, in the near-term, the IP case is likely to create volatility and a certain amount of uncertainty that we believe is likely to keep the stock in check.

Overview of Editas' IP Portfolio

Editas' IP portfolio consists of wholly owned IP as well as licensed patents and patent applications. Within the wholly owned portion of its IP portfolio, Editas has two pending US non-provisional patent applications and 14 pending US provisional patent applications. The company has 13 pending PCT patent applications, including claims for the direct editing component of the genome editing platform, composition of matter, and method of use claims for the therapeutic programs. These patents all expire between 2034 and 2036, excluding any term adjustments and extensions. For LCA10, Editas' first indication, there is one pending US patent and one pending PCT patent application. These are composition of matter and methods of use patents that are expected to expire in 2035.

In addition to wholly owned IP, Editas has a number of in-licensed patents related to foundational CRISPR/Cas9 IP. Editas has in-licensed 16 US patents, 54 pending US patent applications, four EU patents and related validations, 19 pending EU applications, and 21 pending PCT patent applications. These patents come from a variety of sources, including the Broad Institute, Harvard, MIT, Massachusetts General Hospital, and Duke University. A summary of Editas' licensing deals is provided in **Exhibit 16**.

The ongoing debate around gene editing IP that may affect Editas relates to Broad Institute patents licensed by Editas. We summarize and provide our view on this issue in our next section.

Exhibit 16: IP Licensing Deals

Institute	Broad Institute	General Hospital Corp./MGH	Duke University
Number of Patents	-20 US patents -55 pending US patents -4 EU patents and related validations -19 pending EU patent applications -19 pending PCT applications -Other related patent applications outside the US/EU	-10 pending US patent applications -8 pending EU patent applications -2 pending PCT applications -Other related patent applications outside the US/EU	-2 pending US patent applications -1 pending EU patent application -1 pending PCT application
Patent Content	Relates to certain CRISPR/Cas9 and TALE-related composition of matter, use for genome editing, and to certain CRISPR/Cas9 and TALE-related delivery technologies	Relate to CRISPR/Cas9 and TALE-related composition of matter patent and their use in genome editing	For genome editing approaches, including CRISPR/Cas9 and TALEN approaches for treatment DMD.
Allowed Uses	Prevention and treatment of human disease, excluding certain fields, including modification of animals or animal cells for the creation and sale of organs suitable for xenotransplantation into humans and the development and commercialization of products/services with livestock applications.	Prevention or treatment of human or animal disease and agriculture, including for human consumption. Does not include products or processes used for clinical diagnostic assays.	Exclusive rights for use in prevention and treatment of human disease, but excludes research reagents. Non-exclusive rights to all patents, including research reagents, for internal use.
Fees	**Upfront license fee:** Low 6 figures and a single digit percentage of Editas stock **Annual License Maintenance fee:** Low to mid five figures to low six figures begining in 2016, creditable against royalties	**Upfront license fee**: Low 6 figures and less than 1% of common stock. **Annual License Maintenance fee:** Low to mid five digit dollar amounts, beginning in 2017.	**Upfront license fee:** High five digits. **Annual License Maintenance fee:** Ranges from mid-four digit to low five digit dollar amounts beginning in 2015.
Milestones/Royalties	**Milestones:** Up to $54M/indication for larger indications and $4.1M/indication for smaller indications **Royalties:** Mid single-digits percentages for preventative therapies and a range from log single-digit to high single-digit for other products and services	**Milestones:** Up to $1.4M in aggregate for first licensed product/process. Milestones up to $125k in aggregate for 2nd-4th indication to enter the clinic and $625k for 2nd-4th products that make it to market. There are $1.8M in sales milestones. **Royalties:** Low single digit percentage of net sales for the prevention and treatment of human disease. Low single-digit to low double digit on net sales of other products/services.	**Milestones:** Clinical, regulatory, and commercial milestones up to $625k in aggregate per licensed product. **Royalties:** Low single digit percentage based on annual net sales.

Source: Company Data

Interference

What is the issue?

The IP surrounding CRISPR/Cas9 has recently become a topic of substantial discussion. The interference centers around the question of who has broad rights to use CRISPR/Cas9 technology in eukaryotic cells, and the legal case is between the Broad Institute (where a large portion of Editas' IP is licensed from) and the University of California Berkeley in conjunction with the University of Vienna and microbiologist Emmanuelle Charpentier.

The first CRISPR/Cas9 related patent application was filed in March 2013 and named Jennifer A. Doudna and Martin Jinek (University of California Berkeley), Emmanuelle Charpentier, and Krzysztof Chylinski (University of Vienna) as inventors. The Broad Institute's first patent, credited to Feng Zhang, was filed in October 2013. Eleven additional CRISPR/Cas9 patents related to the work of Dr. Zhang were awarded by the USPTO to the Broad Institute in 2013-2014. The University of Berkeley, Dr. Charpentier, and the University of Vienna believe their patent filed March 2013 invalidates the subsequent patents issued to the Broad Institute.

The underpinning of the ongoing legal case between the two parties is the framework under which patent validity is being evaluated. In March 2013 the US adopted a system in which patent disputes were resolved based on a "first-to-file" system. Under this system, this case would be straightforward and Berkeley would likely win patent rights given its first CRISPR/Cas9 patent was filed roughly seven months prior to the Broad Institute's first CRISPR/Cas9 patent. However, as discovery work related to both patents was conducted before March 2013, the case is being evaluated under the "first-to-invent" system implemented prior to March 2013. Under the "first-to-invent" system, when competing patents claim overlapping inventions, patent rights are awarded to the party who can demonstrate that its patent was the first to reduce a concept to a practice.

Berkeley, Dr. Charpentier, and the University of Vienna contend that their patent filed March 2013 covers the use of CRISPR/Cas9 in eurkaryotic cells. As a result, they believe the twelve CRISPR/Cas9 patents awarded to the Broad Institute by the USPTO are invalid and have asked the USPTO to reconsider Broad's patents through a patent interference proceeding. Following Berkeley's request, the USPTO has declared an interference.

The Broad Institute concurs that its patents were not the first CRISPR related patent applications, but contends that Broad was first in describing mammalian genome editing using experimental data. Broad believes the work underlying the Berkeley patent describes the ability of purified Cas9 protein and purified RNA to cleave DNA, but does not demonstrate mammalian genome editing and therefore does not qualify for priority given it does not prove that Berkeley was "first-to-invent."

Eukaryotic Cells Key To Interference Dispute

The so-called reduction of practice issued related to the CRISPR/Cas9 patent interference debate relates mainly to the Broad's contention that it is not obvious to translate non-mammalian uses of the CRISPR system to mammalian systems (of which its IP describes) versus California's contention that a person skilled in the art would understand how to translate the use across eukaryotic cells. We found this quote from Jennifer Doudna in a publication which says, "it was not known whether such a bacterial system would function in eukaryotic cells" as particularly instructive in potentially supporting the Broad's claims. (see Jinek et al. eLife 2013;2:e00471).

The patents under debate are summarized in the exhibit below. Each patent from each party maps to multiple claims that each party believes speaks to the count that is the centerpiece of this interference. This count is the use of a Cas9 protein and a DNA-targeting RNA comprising a targeter-RNA or guide sequence and an activator-RNA or tracer sequence wherein the Cas9/DNA-targeting RNA complex is used to cleave or edit target DNA or modulate the transcription of at least one gene encoded by the target DNA in ***eukaryotic cells*** (emphasis added).

Exhibit 17: Summary of Berkeley and Broad Institute Patents

Party	Patent	Filing Date	Named Inventors	Title
University of California University of Vienna	Application 13/842,859	3/15/2013	Jennifer A. Doudna (Berkeley) Martin Jinet (Berkeley) Krzysztof Chylinski (Vienna) Emmanuelle Charpentier	Methods and Compositions for RNA-Directed Target DNA Modification and for RNA-Directed Modulation of Transcription
The Broad Institute Harvard College* MIT	8,697,359	10/15/2013	Feng Zhang	CRISPR-Cas Systems and Methods for Altering Expression of Gene Products
	8,771,945	2/18/2014	Feng Zhang	
	8,945,839	4/18/2014	Feng Zhang	
	8,795,965	2/18/2014	Feng Zhang	CRISPR-Cas Component Systems, Methods, and Compositions for Sequence Manipulation
	8,871,445	4/23/2014	Le Cong, Feng Zhang	
	8,865,406	3/24/2014	Feng Zhang, Fei Ran	Engineering and Optimization of Improved Systems, Methods and Enzyme Compositions for Sequence Manipulation
	8,895,308	6/2/2014	Feng Zhang, Fei Ran	
	8,889,356	2/18/2014	Feng Zhang	CRISPR-Cas Nickase Systems, Methods, and Compositions for Sequence Manipulation in Eukaryotes
	8,932,814	4/22/2014	Le Cong, Feng Zhang	
	8,906,616	5/29/2014	Feng Zhang, Le Cong Patrick Hsu, Fei Ran	Engineering of Systems, Methods and Optimized Guide Compositions for Sequence Manipulation
	8,993,233	12/13/2013	Feng Zhang, Le Cong, Randall Jeffrey Platt Neville Espi Sanjana, Fei Ran	Engineering and Optimization of Systems, Methods and Compositions for Sequence Manipulation with Functional Domains
	8,999,641	3/26/2014	Feng Zhang, Le Cong Randall Jeffrey Platt, Neville Espi Sanjana	

**Harvard College is not an assignee for the following patents: 8,697,359; 8,771,945; 8,795,965; 8,889,356; 8,945,839*

Source: USPTO.gov, Morgan Stanley Research

What is an interference?

Interference proceedings occur when two groups file potentially conflicting patents. These proceedings are declared by the USPTO after reviewing a party's suggestion of interference, and are contested in the USPTO between two applications or patents to determine which party has priority with their patent. In this case, the University of California, the University of Vienna, and Emmanuelle Charpentier filed suggestions of interference throughout 2015, and the USPTO declared the interference proceeding in January 2016.

In an interference proceeding, the "senior party" initially refers to the group with the earlier patent filing date while the "junior party" is the one with the later filing date. The burden of proof in an interference case falls on the junior party. The Broad Institute is the junior party in this case. However, it is worth noting that "senior" and "junior" designations in this case may change based on a judge's review of which party had the earliest patent filing to demonstrate use of CRISPR for genome editing in a eukaryotic cell.

A timeline for the proceeding will be established in early March 2016. Based on our diligence, we have learned that interference proceedings typically take ~1.5 years to resolve. A timeline of key recent and upcoming events for the proceeding are provided below. All of the filings for this interference are available online at **https://acts.uspto.gov/ifiling/PublicView.jsp**. The interference is 106,048.

Interference timeline

April 13, 2015 - University of California on behalf of itself and the University of Vienna and Emmanuelle Charpentier filed a suggestion of interference in the USPTO against the 10 U.S. patents in-licensed from Broad

April 13, 2015 – ToolGen filed a suggestion of Interference in the USPTO against 5 U.S. patents licensed from Broad

November 5, 2015 – University of California and Emmanuelle Charpentier filed a supplemental suggestion of interference against 2 U.S. patents and 5 pending U.S. patents applications from Broad

January 11, 2016 – The USPTO declared an interference proceeding for claims related to CRISPR

March 3, 2016 – Each party will file and serve a list of motions

March 10, 2016 – Appeal board will hold a conference call to discuss the interference, and a time line will be set on this call

Late 2017 – Interference proceedings take ~1.5 years on average. Expected resolution in late 2017

Overview of European Patents and Current Opposition Proceedings

Patents at issue and nature of the opposition

There are two key patents at issue in Europe currently. Both have been in-licensed from Harvard/MIT/Broad and describe various CRISPR applications, as summarized below.

Exhibit 18: Summary of Broad/Harvard/MIT Patents at Dispute in the EU

Party	Patents	Filing Date	Named Inventors	Title	Key Claims
The Broad Institute Harvard College MIT	2,771,468 2,784,162	12/12/2013	Feng Zhang, Le Cong Patrick Hsu, Fei Ran	Engineering of Systems, Methods and Optimized Guide Compositions for Sequence Manipulation	**(1)** Methods for design and use of vectors that encode components of a CRISPR complex **(2)** Methods of directing CRISPR complex formation in eukaryotic cells **(3)** Methods for selecting specific cells by introducing precise mutations utilizing the CRISPR-Cas system

Source: EPO.org, Morgan Stanley Research

Nine oppositions have been filed against Patent 2,771,468. Parties opposing the patent include CRISPR Therapeutics, Novozymes, and several IP attorneys and legal firms (likely representing industry). The notices of opposition against this patent were filed in late 2015, and Broad/Harvard/MIT were notified in late December 2015 that they have four months from 12/22/15 to file their observations in response to all nine oppositions. All oppositions request the complete revocation of Broad's patent, and oral proceedings in case the European Patent Office does not decide to revoke the patent based on the oppositions and supporting documents filed.

Eight oppositions have been filed against Patent 2,784,162. The group opposing this patent comprises the same parties that filed oppositions to Patent 2,771,468, barring industrial biotechnology firm Novozymes. These oppositions were filed in early January 2016, and Broad/Harvard/MIT were notified in mid-February 2016 that they must file their observations in response to all oppositions within four months from 2/18/16.

What is an opposition proceeding?

An opposition proceeding is the EU equivalent to an interference. The opposition process is initiated through the formal filing of a notice of opposition by a party (or parties) that believe a certain patent is invalid on the grounds that (1) the subject matter of the patent is not patentable, (2) the patent does not describe the discovery clearly and completely enough for a skilled person to execute it, and/or (3) the patent's subject matter reaches beyond the content of the patent filing. Oppositions must be filed within nine months after the publication of a mention of the patent in question being granted. Oppositions filed to both Broad/Harvard/MIT patents cut across all three criteria as grounds for revocation.

Following the filing of an opposition the defending party is allowed a set amount of time (usually four to six months) to file their observations in response to the opposition(s). Following a review of documents filed by both sets of parties, the EPO Opposition Division may seek additional information and may also provide a preliminary opinion. If the division is not able to reach a final verdict based on documentation filed, or if either party requests an oral proceeding, an oral hearing is held to determine whether the patent in question will be (1) in effect as originally granted, (2) maintained but with amendments, or (3) revoked. Requests for an appeal must be filed within two months of the notification of the final decision. While variability exists on how long specific cases take to resolve, our diligence suggests that proceedings typically take ~1.5-3 years to resolve (not

including an appeal process).

Opposition timeline

November 11, 2015 – Nine oppositions were filed in the EU against an EU patent in-licensed from Broad (EP 2,771,468 B1)

January 8, 2016 - Eight oppositions were filed in the EU against a different EU patent in-licensed from Broad (EP 2,784,162 B1)

April 2016 - Deadline for Broad/Harvard/MIT to submit their observations in response to oppositions filed against Patent 2,771,468

June 2016 - Deadline for Broad/Harvard/MIT to submit their observations in response to oppositions filed against Patent 2,784,162

Potential Outcomes to the IP case?

Overall, we see three potential outcomes to the overall IP case that seem plausible. A total win or loss for Editas or a third scenario in which each party has some of their claims narrowed so as not to interfere with either parties IP, but where both parties still require each others' IP in order to have freedom to operate.

Potential Outcome 1: A complete loss for Editas

In this scenario, the reduction of practise argument would be won by California and all of the Broad's patents would be invalidated. While this would not be the end of Editas' commercial potential, it would severely limit their freedom to operate and likely mean they would have to acquire a license (which we assume would be at great cost) from California. We would expect the stock to trade near our bear case of cash only value in this scenario.

Potential Outcome 2: A complete win for Editas

In this scenario, the reduction of practise argument would be won by Editas and the University of California patents would be invalidated. This would clearly lift an overhang and likely lead to significant appreciation in EDIT. That said, given that our base case already assumes freedom to operate, it would not significantly impact our fundamental valuation which relies on CRISPR products demonstrating a therapeutic benefit.

Potential Outcome 3: A draw where both parties must cross license.

In this scenario, neither party has a clear win. Both parties would have their patents modified and the claims in each patent and the statements attached to those claims could all be modified. We see this as making the most sense for the interference proceeding. While one party would likely have somewhat of an upper hand in the cross licensing negotiations, we think this scenario would at most lead to a net single digit royalty to the party with the upper hand. Thus, given the size of the potential market for CRISPR therapy, this would likely have a very modest impact on our valuation given we already assume some modest royalty in our COGS.

Note that we are not acting in the capacity of attorneys, nor do we hold ourselves out as such. This material is not intended as either a legal opinion or legal advice. The potential outcomes discussed above are hypothetical and for illustrative purposes only.

Debate 3 - How do you value the opportunity?

Overview: Valuing early stage technology always presents a challenge. There is little, if any, clinical data on which to base one's valuation, yet the promise of the technology platform is huge. Thus, accounting for the value of "what if" is offset by the reality of the significant risks ahead to realize that "what if" leads to a wide variety of interpretations about intrinsic value.

Street's take: Consensus has always been of two minds with early stage technology. Those with short term investing horizons typically only value what is nearest to market while those with longer-term time horizons value the "what if" much more heavily. In this case, given that we believe many investors see the strategic potential and significant market opportunity, an ability to value more than just the initial program in LCA10 is warranted. That said, given the current market, investors also understand they must be measured in their approach.

Our take: We value the LCA10 program in the eye directly given our view that this program presents the highest likelihood of an initial proof of concept for CRISPR/Cas9. There are a few items, particularly that the program only targets the eye which is a closed system and limits off-target concerns, that only 10-20% of the photoreceptors need to be edited (and one allele at that) to maintain vision and that since the eye is small and physicians have direct access, delivery should not impede a clinical benefit. That said, LCA10 is a small market. Thus, we also believe that some value needs to be assigned broadly to CRISPR/Cas9. In this case we have chosen to use our annualized market sizes for cystic fibrosis, malignant and non-malignant hematology (leukemia, lymphoma, sickle cell disease and beta-thalassemia) and duchenne muscular dystrophy as a proxy for the market potential of CRISPR. We have then assumed Editas could achieve 30% market share into that market at peak (2032E) and launch an Editas product in 2025E. We ramp that revenue from 2025E-2032E and then assign a 10% probability of success. The latter calculation represents the majority of our non-cash base valuation.

Overview

Editas's approach is based on the idea that DNA mutations are the root cause of a substantial number of diseases. There are approximately 10,000 genetic diseases and approximately 7,000 rare diseases. Of these rare diseases, approx. 80% (5,600) have a genetic basis and only ~400 have approved therapies. There remains a substantial unmet medical need in many genetic diseases. Recent advances in the technology surrounding gene editing are beginning to allow direct editing of genetic material that may allow these previously unaddressable diseases to be addressed.

Editas's CRISPR/Cas9 approach is highlighted in **Debate 1 - What is CRISPR/Cas9?** This is a versatile technology that can be applied over a wide range of genetic disorders. Editas is developing this technology for a number of indications. The Editas pipeline includes Leber Congenital Amaurosis 10 (LCA10), genetic and infectious diseases of the eye, gene editing in T-cells to treat cancer, non-malignant hematological disorders, genetic diseases of muscle, genetic diseases of the lung, and genetic and infectious diseases of the liver.

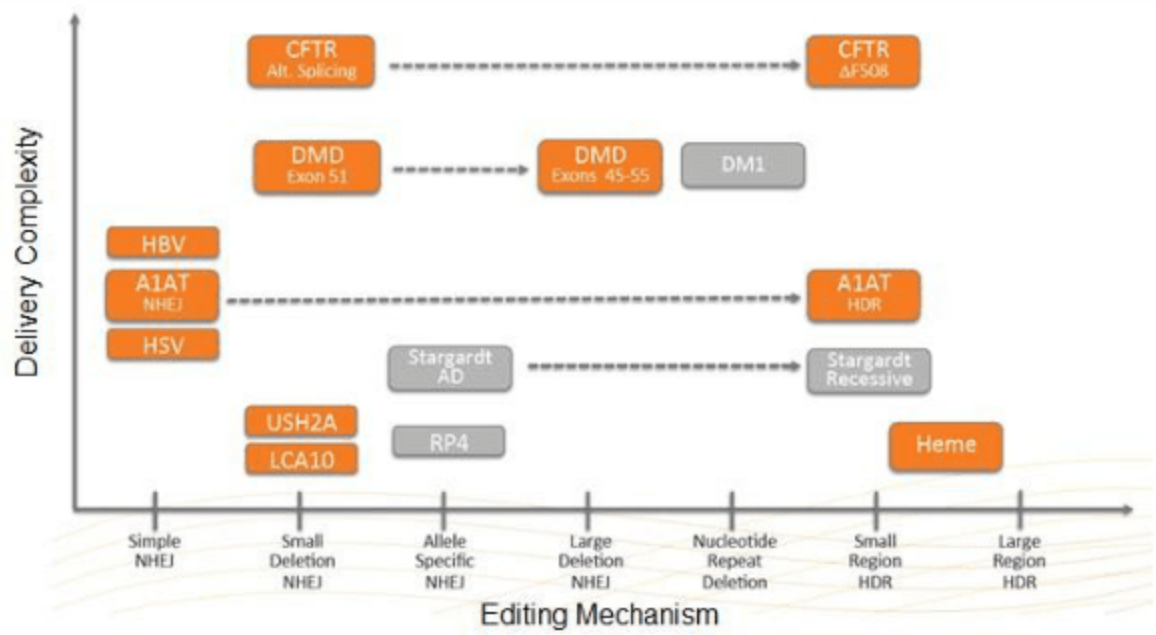
Exhibit 19: Editas Pipeline

Program	Target Gene	Stage	Delivery	Editing Mechanism	IP
Leber Congenital Amaurosis 10	CEP290	Discovery, IND enabling studies in 2016, Phl start in 2017	AAV in vivo	NHEJ – Small Deletion	Patents expire 2033-2036
Genetic and Infectious Diseases of the Eye	Multiple	Discovery	AAV in vivo	NHEJ	Patents expire 2033-2036
Gene Editing in T Cells to Treat Cancer	Multiple	Discovery	Ex vivo	NHEJ	Patents expire 2033-2036
Non-Malignant Hematologic Diseases	Multiple	Discovery	Ex vivo	NHEJ & HDR	Patents expire 2033-2036
Genetic Diseases of Muscle	Multiple	Discovery	Multiple	NHEJ – Small & Large Deletion	Patents expire 2033-2036
Genetic Diseases of Lung	Multiple	Discovery	Multiple	NHEJ & HDR	Patents expire 2033-2036
Genetic and Infectious Diseases of Liver	Multiple	Discovery	Multiple	NHEJ & HDR	Patents expire 2033-2036

Source: Company Data

The first program in development is for LCA10. This particular indication was selected because it requires both a simpler editing mechanism and because there is an established method of delivery. The size and complexity of the mutation being addressed will inform the editing mechanism necessary. Together with the complexity of delivery, these will determine the difficulty in developing therapies.

Exhibit 20: Mutation Complexity vs. Delivery



Source: Company Data

The company has a number of key upcoming catalysts. In 2016 management is focused on moving programs through preclinical testing as well as new business development and the on-going interference proceedings discussed in **Debate 2 - What Is Going On With IP And What Is The Most Likely Outcome?** Management plans to enter the clinic in 2017 and treat the first patient with LCA10 using their CRISPR/Cas9 technology, with additional medicines to enter the clinic in 2017/2018. Editas currently has a collaboration with Juno for the development of CAR-T cells. If this collaboration is not renewed it will expire in 2020.

Leber Congenital Amaurosis 10

Leber Congenital Amaurosis (LCA) consists of a heterogenous group of inherited retinal dystrophies. This family of diseases can be caused from at least 18 different mutations. The overall incidence of LCA is ~2-3/100,000.

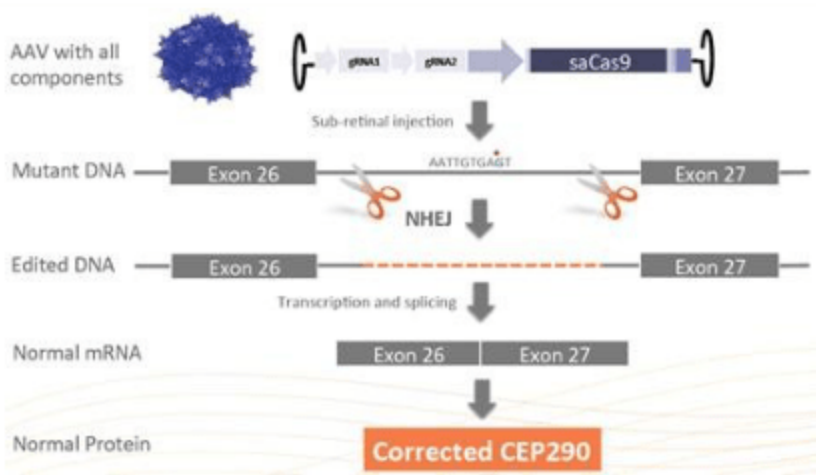
The disease usually manifests with significant vision loss, rapid involuntary eye movements, and there is an absence of measurable electroretinogram recordings. The most common form of LCA is LCA10, which makes up ~20-30% of all LCA cases. LCA10 is caused by an autosomal recessive mutation int the CEP290 gene. There is no approved treatment and no potential therapies for LCA10 other than the one Editas has in development.

Editas is currently applying their CRISPR/Cas9 system to LCA10 in preclinical testing. In preclinical models the Cas9 and guide RNA pairs can edit the mutation and lead to correctly spliced DNA. They are currently characterizing the frequency of these modifications seen with treatment of patient cells which will allow them to select the proper combinations of RNA and Cas9 proteins. These preclinical trials are being done to assess the efficacy of CEP290 editing in human photoreceptors.

Gene therapy is a viable treatment for certain forms of LCA. LCA2 is caused by mutations in the RPE65 gene. This is a 65 kDa gene. Gene therapy has been shown to be a viable treatment for LCA2. However, CEP290 is significantly larger at 290 kDA and is too large for an AAV vector. Thus conventional gene therapy cannot be applied to treatment of LCA10, but gene editing therapies are still an option. Editas is also able to deliver the CRISPR/Cas9-based treatments through subretinal AAV injections, an established delivery method for other ophthalmic diseases.

LCA10 provides a near-ideal indication for Editas to demonstrate proof-of-concept for their CRISPR/Cas9 system. The most common nucleotide mutation on the CEP290 gene is an A to G change that occurs on an intron, a non-coding portion of DNA that is removed during RNA splicing. No new genetic elements need to be added, thus NHEJ genome editing can be used with low risk of altering the protein coding structure. The NHEJ system is less complex than the HDR gene editing mechanism. A common concern between gene therapy and gene editing is the delivery of sufficient quantities of vector . The eye is of limited size and is an enclosed compartment, thus sufficient quantities of vector can be delivered safely. Furthermore, the eye is an immune-privileged region and thus there is less risk for a systemic toxic response. The subretinal injection will allow treatment to be delivered directly into the eye, minimizing overall systemic exposure. Further, because the second eye serves as an internal control, the trial itself can be completed more easily.

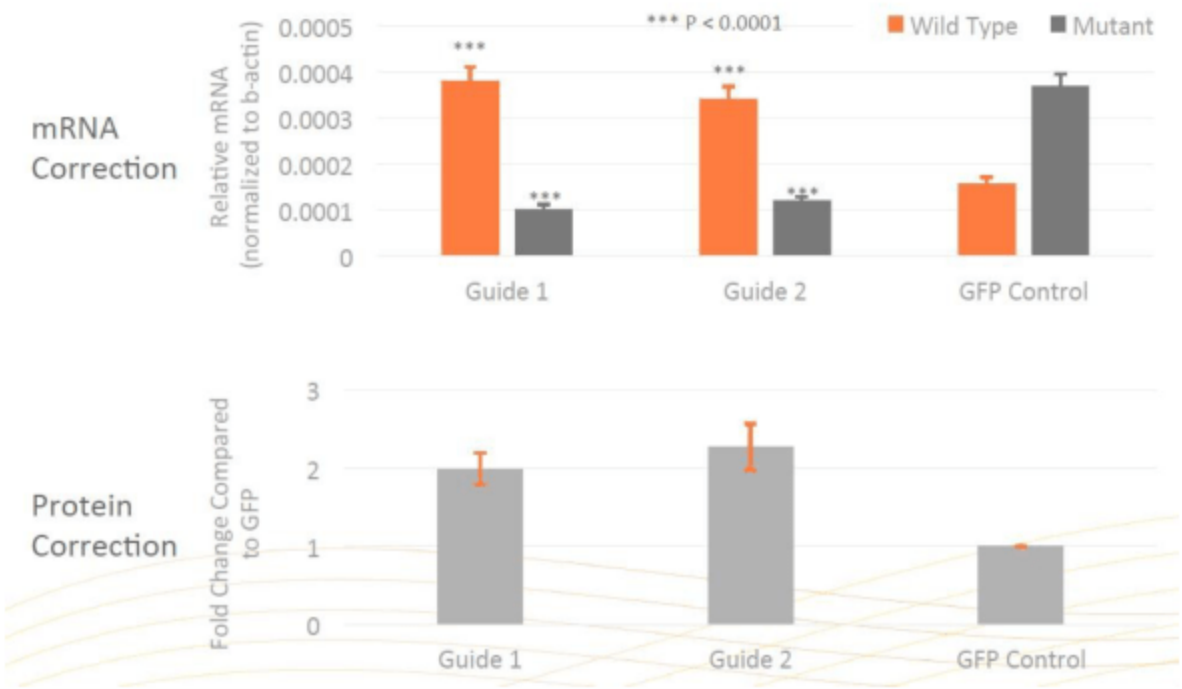
Exhibit 21: CRISPR/Cas9 Treatment for LCA10



Source: Company Data

Over 140 different guides were tested in order to optimize the guide/RNA components. The optimal guides lead to significant increases in amount of correct protein over controls.

Exhibit 22: mRNA Guides Compared with Controls



Source: Company Data

Editas management plans to enter the clinic and treat their first patient with LCA10 using the CRISPR/Cas9 system in 2017. We model LCA10 as beginning to generate US revenues in 2024 with LCA10 prevalence of ~700 patients. We model Editas initially achieving 5% market share in 2024, growing to 25% by 2029 and an initial price of $1M, growing 1.5% yearly. We model the LCA10 treatment entering the EU in 2024 with LCA10 prevalence of ~1,050 patients. We model an initial 3% market share increasing to 17% by 2029 and an initial price of $900,000 decreasing 1.5% yearly. We assume Editas' LCA10 treatment is curative, and as a result model the addressable LCA10 market size decreasing year-on-year in both the US and EU.

Other Programs

Editas has several other programs in preclinical development and a partnership with Juno for the development of CAR-T based therapies. Several other solely owned preclinical programs are also in development.

Juno Partnership

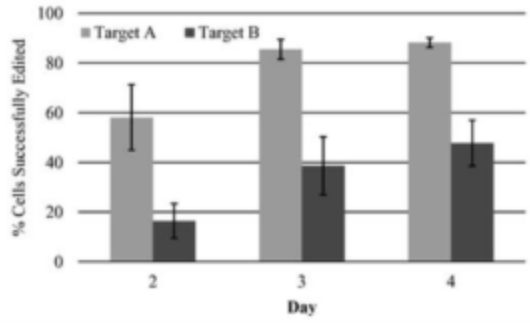
Editas has entered into a partnership with Juno for the development of engineered CAR-T cells and engineering T-cell receptors. Editas is responsible for the generation of genome editing reagents that modify targets selected by Juno. Juno will evaluate these agents and will be responsible for global development, manufacturing, and commercialization of the CAR and TCR engineered products, excluding those for medullary cystic kidney disease. The targets will use both NHEJ and HDR approaches and will be both *in vivo* and *ex vivo*.

Editas received a $25M upfront payment from Juno and will receive up to $22M more in research support over the 5 year collaboration (until May 26, 2020). Editas can also receive up to ~$700M in aggregate from potential research, regulatory, and commercial milestones for each of the three research programs with Juno. Editas will also receive a net sales royalty in the low double-digit percentages. The Juno collaboration ends in 2020, but Juno retains the option to extend the collaboration through May 26, 2022 upon paying extension fees in the mid-single digit millions range per year. Early testing of the use of genome editing in T-cells has proven encouraging, as demonstrated below.

Non-Malignant Hematological Diseases Mutations in the gene for human beta globin can result in diseases such as sickle cell disease and beta-thalassemia. Editas is studying how to best apply their CRISPR/Cas9 system

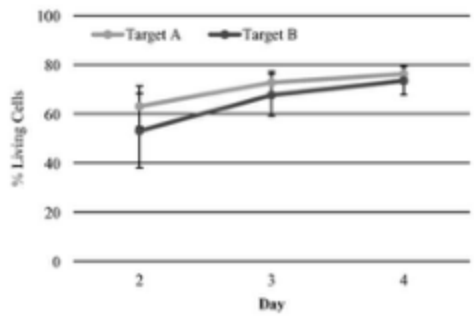
to address these mutations. The editing for these diseases could be done *ex vivo* on harvested hematopoietic stem cells. Once the cells are edited and it is verified that they produce the proper proteins, then the edited cells can be reintroduced into the body and used to repopulate the bone marrow. Through an *ex vivo* study, Editas

Exhibit 23: Editing of T-Cell Target Genes in Human T-Cells



Source: Company Data

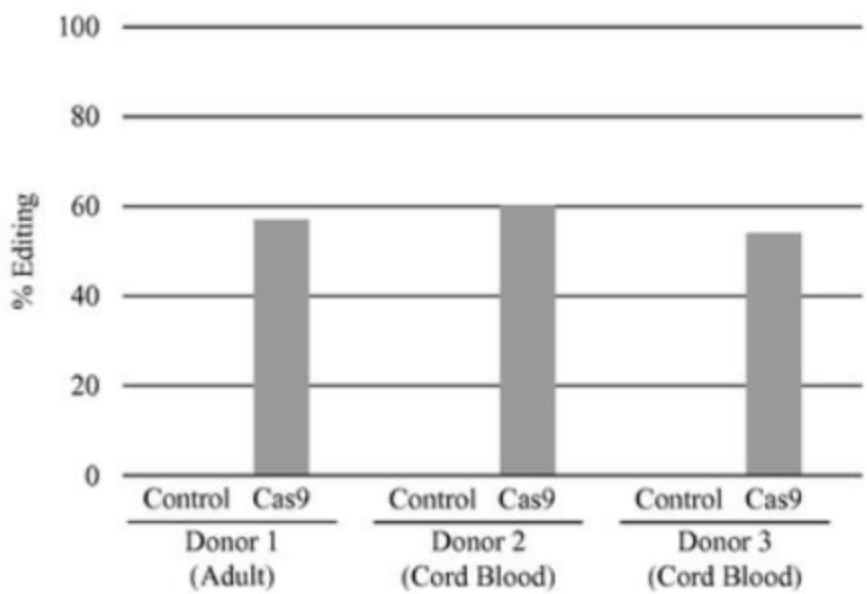
Exhibit 24: Cell Viability of Human T-Cells Following Delivery of Cas9-Guide RNA Complexes



Source: Company Data

observed ~60% editing activity for the human beta globin gene in cells treated with Cas9-guide RNA complexes over control cells, as demonstrated in **Exhibit 22**. Treatment of the most common form of sickle cell disease would involve fixing the mutation of a single base pair, but treatment of beta-thalassemia would require the insertion of a new gene, thus likely requiring use of HDR editing.

Exhibit 25: Ex-vivo Editing of Hemoglobin Beta Gene in Human Stem Cells



Source: Company Data

Duchenne Muscular Dystrophy

Duchenne Muscular Dystrophy (DMD) can be caused by a variety of mutations of the gene that encodes the protein Dystrophin. The Dystrophin protein helps provide structural stability to a complex found on the cell surface, particulary in muscle tissue cells. Mutations of the gene encoding this protein leads to progressive muscle weakness and atrophy, eventually leading to the failure of respiratory muscles. The dystrophin gene is one of the largest found in the human genome and mutations throughout that gene can occur which result in DMD. Some of these mutations lead to deformed proteins that still retain some functionality. Gene editing in DMD could serve to make a smaller, but functional protein. NHEJ editing of a small deletion on exon 51 would address ~13% of DMD patients, while a NHEJ-mediated large deletion therapy applied to exons 45-55 would

address ~60% of DMD patients. Delivery is complicated by the need to deliver the CRISPR/Cas9 therapy to striated muscle distributed throughout the body. Research at Duke is moving ahead on DMD and is a potential area of focus for Editas.

Cystic Fibrosis

Cystic fibrosis is a result of mutations in the cystic fibrosis conductance regulatory (CFTR) protein. The CFTR maintains water balance in the lung. Mutations of the gene encoding this protein lead to an imbalance of ions and water, resulting in a mucus excess. Similar to other genetic disorders, there are a variety of mutations that can lead to cystic fibrosis. An initial NHEJ based approach could be used to delete rare mutations, which would help address the disease in a smaller segment of the CF population. An HDR approach could be used to correct the Del F508 mutation, which accounts for the mutation in ~70% of CF patients. Delivery in CF is complicated and will require efficient editing of the epithelial cells of the airways along the lungs. Novel delivery methods are in development such as liposomal encapsulation.

Usher Syndrome 2A (USH2A)

Usher Syndrome is a group of disorders that can be caused by a variety of mutations and leads to hearing loss and/or visual impairment. Usher Syndrome 2A occurs in patients that have mutations in the gene encoding the Usherin protein, which is found in the inner ear and in the retina. Over 200 mutations have been associated with the USH2A gene and loss of the Usherin protein leads to retinal degeneration and progressive vision loss. The most common location of the mutations of the USH2A gene are on exon 13. NHEJ editing could be used to delete a small section of the gene.

Herpes Simplex Virus 1 (HSV-1)

A Herpes Simplex Virus infection is a lifelong infection and activation of HSV-1 usually leads to oral and ocular disease. The virus will remain latent in neuron ganglia and will retain the ability to reactivate. However, in this latent phase the virus does not integrate into the host's genome. CRISPR/Cas9 could be used to cleave and inactivate the HSV-1 DNA, thus preventing reactivation. Inactivating DNA would likely require simple NHEJ editing.

Alpha-1 Antitrypsin (A1AT) Deficiency

Alpha-1 antitrypsin is a protease inhibitor. In alpha-1 antitrypsin deficiency, the protein is defective so that it accumulates in the liver and there is decreased activity in the blood and lungs. Due to this decreased activity, a number of proteases are under inhibited, including neutrophil elastase. This leads to elastin destruction in the lungs, ultimately causing emphysema. A1AT accumulation in the liver can lead to jaundice. The A1AT gene could be deleted using NHEJ, or HDR could be used to add the correct gene to the cells in the liver.

Valuing these other programs

We model other program revenues as coming from the CAR-T program, non-malignant hematology, DMD, and CF. These programs are still in preclinical development, and we therefore assign them a low overall probability of success. We define the revenue opportunity to be 30% market share of the four target markets mentioned above, and our base case assigns Editas a 10% probability of success in achieving this share. We further discount by assuming Editas is able to realize a certain portion (which increases every year) of the revenues implied by a 10% probability of success (POS) of obtaining 30% market share. We assume these blended revenues begin in 2025 at $240M and increase to $3.2B by 2032.

Our Revenue Models

Leber Congenital Amaurosis 10 (LCA10)

Our key base case assumptions for our LCA10 revenue model are outlined below.

- US and EU market launch in 2024E

- Addressable US and EU LCA10 populations in 2024E of ~700 and ~1,050, respectively, based on LCA prevalence of ~1/100,000 and ~20% of LCA cases caused by CEP290 mutations
- Curative potential assumed for Editas' LCA10 therapy in both the US and EU
- US market share grows from 5% of the addressable market to 25% by 2029E, with 25% share maintained through the end of the projection period in 2032E. Give our assumption that Editas' therapy is curative, our base case assumes ~80% of the addressable LCA10 population in 2024E is treated by 2032E.
- EU market share grows from 3% of the addressable population to 17% by 2029E, with 17% of the remaining addressable population treated each year through the end of the projection period. Our base case assumes ~70% of the addressable population as of 2024E is treated by end of 2032E.
- US gross pricing of $1M/patient at launch, growing at 1.5%/year with a 15% gross-to-net discount
- EU gross pricing of $900K/patient at launch, decreasing 1.5%/year with a 20% gross-to-net discount

Exhibit 26: US LCA10 Revenue Model

	2015E	2024E	2025E	2026E	2027E	2028E	2029E	2030E	2031E	2032E
U.S. Population	321,000,000	341,798,590	344,191,181	346,600,519	349,026,722	351,469,910	353,930,199	356,407,710	358,902,564	361,414,882
% growth	NA	0.7%	0.7%	0.7%	0.7%	0.7%	0.7%	0.7%	0.7%	0.7%
LCA prevalence	3,210	3,418	3,442	3,466	3,490	3,515	3,539	3,564	3,589	3,614
% growth	NA	0.7%	0.7%	0.7%	0.7%	0.7%	0.7%	0.7%	0.7%	0.7%
LCA10 prevalence	642	684	688	693	698	703	708	713	718	723
% growth	NA	0.7%	0.7%	0.7%	0.7%	0.7%	0.7%	0.7%	0.7%	0.7%
Addressable LCA10 population	642	684	654	594	509	419	329	253	195	152
% growth	NA	0.7%	-4.3%	-9.3%	-14.2%	-17.8%	-21.3%	-23.2%	-22.8%	-22.2%
Market share scenarios										
Bull	*0%*	*8%*	*15%*	*23%*	*28%*	*34%*	*37%*	*37%*	*37%*	*37%*
Base	*0%*	*5%*	*10%*	*15%*	*19%*	*23%*	*25%*	*25%*	*25%*	*25%*
Bear	*0%*	*0%*	*0%*	*0%*	*0%*	*0%*	*0%*	*0%*	*0%*	*0%*
Editas LCA10 share	0%	5%	10%	15%	19%	23%	25%	25%	25%	25%
LCA10 patients treated	0	34	65	89	96	94	82	63	48	38
% growth	NA	NA	91.4%	36.1%	7.3%	-1.3%	-13.5%	-23.2%	-22.8%	-22.2%
Pricing scenarios										
Bull	*$0*	*$1,200,000*	*$1,218,000*	*$1,236,270*	*$1,254,814*	*$1,273,636*	*$1,292,741*	*$1,312,132*	*$1,331,814*	*$1,351,791*
Base	*$0*	*$1,000,000*	*$1,015,000*	*$1,030,225*	*$1,045,678*	*$1,061,364*	*$1,077,284*	*$1,093,443*	*$1,109,845*	*$1,126,493*
Bear	*$0*	*$0*	*$0*	*$0*	*$0*	*$0*	*$0*	*$0*	*$0*	*$0*
Price per patient, gross	$0	$1,000,000	$1,015,000	$1,030,225	$1,045,678	$1,061,364	$1,077,284	$1,093,443	$1,109,845	$1,126,493
Price per patient, net	$0	$850,000	$862,750	$875,691	$888,827	$902,159	$915,691	$929,427	$943,368	$957,519
Revenues	$0	$29,052,880	$56,441,321	$77,971,671	$84,896,252	$85,007,318	$74,670,938	$58,172,548	$45,596,480	$36,016,789

Source: Company Data, Morgan Stanley Research

Exhibit 27: EU LCA10 Revenue Model

	2015E	2024E	2025E	2026E	2027E	2028E	2029E	2030E	2031E	2032E
EU Population	509,000,000	$22,909,076	$24,477,803	$26,051,236	$27,629,390	$29,212,278	$30,799,915	$32,392,315	$33,989,492	$35,591,460
% growth	NA	0.3%	0.3%	0.3%	0.3%	0.3%	0.3%	0.3%	0.3%	0.3%
LCA prevalence	5,090	5,229	5,245	5,261	5,276	5,292	5,308	5,324	5,340	5,356
% growth	NA	0.3%	0.3%	0.3%	0.3%	0.3%	0.3%	0.3%	0.3%	0.3%
LCA10 prevalence	1,018	1,046	1,049	1,052	1,055	1,058	1,062	1,065	1,068	1,071
% growth	NA	0.3%	0.3%	0.3%	0.3%	0.3%	0.3%	0.3%	0.3%	0.3%
Addressable LCA10 population	1,018	1,046	1,023	975	880	774	661	555	467	393
% growth	NA	0.3%	-2.2%	-4.7%	-9.7%	-12.1%	-14.6%	-16.0%	-15.9%	-15.8%
Market share scenarios										
Bull	0%	4%	8%	15%	19%	23%	25%	25%	25%	25%
Base	0%	3%	5%	10%	13%	15%	17%	17%	17%	17%
Bear	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%
Editas LCA10 share	0%	3%	5%	10%	13%	15%	17%	17%	17%	17%
LCA10 patients treated	0	26	51	97	110	116	109	92	77	65
% growth	NA	NA	95.6%	90.6%	12.9%	5.4%	-6.0%	-16.0%	-15.9%	-15.8%
Pricing scenarios										
Bull	$0	$1,080,000	$1,063,800	$1,047,843	$1,032,125	$1,016,643	$1,001,394	$986,373	$971,577	$957,004
Base	$0	$900,000	$886,500	$873,203	$860,104	$847,203	$834,495	$821,977	$809,648	$797,503
Bear	$0	$0	$0	$0	$0	$0	$0	$0	$0	$0
Price per patient, gross	$0	$900,000	$886,500	$873,203	$860,104	$847,203	$834,495	$821,977	$809,648	$797,503
Price per patient, net	$0	$720,000	$709,200	$698,562	$688,084	$677,762	$667,596	$657,582	$647,718	$638,002
Revenues	$0	$18,824,727	$36,268,848	$68,096,977	$75,731,438	$78,647,087	$72,781,767	$60,206,738	$49,859,929	$41,345,825

Source: Company Data, Morgan Stanley Research

Additional Programs

We value Editas' platform by valuing revenue streams from the application of the platform in CAR-T, non-malignant hematology, DMD, and cystic fibrosis. Given these are very early-stage programs, we heavily discount their expected revenue potential to Editas. For each program, our base case first assigns Editas a 10% probability of success in achieving 30% share of the market. We then further discount to account for blended product launches so that Editas earns an increasing portion of the revenue opportunity year-on-year. For 2025E, Editas earns 10% of the ~$2.4B total revenue opportunity implied by a 10% POS of achieving ~$24B, which represents 30% share of the four target markets combined. The amount of the revenue opportunity earned increases 10% every year, such that Editas' additional programs generate ~$3.2B in revenue by 2032E (80% of the ~$4B opportunity implied by a 10% POS of achieving 30% market share of the four target markets combined).

Exhibit 28: Additional Revenue Opportunities for Editas

	2016E	2017E	2018E	2019E	2020E	2021E	2022E	2023E	2024E	2025E	2026E	2027E	2028E	2029E	2030E	2031E	2032E
CAR-T	$3,500	$3,920	$4,390	$4,917	$5,507	$6,168	$6,908	$7,737	$8,666	$9,706	$10,870	$12,175	$13,636	$15,272	$17,105	$19,157	$21,456
Non-malignant Hematology	$4,545	$4,567	$4,590	$4,613	$4,636	$4,659	$4,683	$4,706	$4,730	$4,754	$4,778	$4,803	$4,827	$4,852	$4,876	$4,974	$5,073
DMD	$4,071	$4,304	$4,551	$4,812	$5,088	$5,380	$5,688	$6,014	$6,359	$6,724	$7,110	$7,536	$7,988	$8,468	$8,976	$9,514	$10,085
CF	$2,423	$2,457	$2,490	$2,522	$2,554	$2,586	$2,618	$2,690	$2,744	$2,799	$2,855	$2,912	$2,970	$3,030	$3,090	$3,152	$3,215
Total Potential Revenues (30% market share)	$14,539	$15,248	$16,021	$16,864	$17,785	$18,793	$19,917	$21,148	$22,499	$23,983	$25,613	$27,426	$29,421	$31,621	$34,047	$36,798	$39,830
Bull	20%	20%	20%	20%	20%	20%	20%	20%	20%	20%	20%	20%	20%	20%	20%	20%	20%
Base	10%	10%	10%	10%	10%	10%	10%	10%	10%	10%	10%	10%	10%	10%	10%	10%	10%
Bear	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%
Probability of success	*10.0%*	*10.0%*	*10.0%*	*10.0%*	*10.0%*	*10.0%*	*10.0%*	*10.0%*	*10.0%*	*10.0%*	*10.0%*	*10.0%*	*10.0%*	*10.0%*	*10.0%*	*10.0%*	*10.0%*
	$1,454	$1,525	$1,602	$1,686	$1,779	$1,879	$1,992	$2,115	$2,250	$2,398	$2,561	$2,743	$2,942	$3,162	$3,405	$3,680	$3,983
Assume blended 2025-2028 launches										$240	$512	$823	$1,177	$1,581	$2,043	$2,576	$3,186

Source: Company Data, Morgan Stanley Research

Income Statement

Exhibit 29: Editas Medicine Income Statement

Editas Medicine Income Statement ($M)	2013	2014	2015E	2016E	2017E	2018E	2019E	2020E	2021E	2022E	2023E	2024E	2025E
Total Revenue	-	-	**1.3**	**9.0**	**9.0**	**9.0**	**9.0**	**7.5**	**9.4**	**11.8**	**14.7**	**66.3**	**355.5**
Collaboration Revenue	-	-	1.3	9.0	9.0	9.0	9.0	7.5	9.4	11.8	14.7	18.4	23.0
LCA10 Product Revenue (WW)	-	-	-	-	-	-	-	-	-	-	-	47.9	92.7
Additional Product Revenue	-	-	-	-	-	-	-	-	-	-	-	-	239.8
Consensus Revenue													
Costs and expenses													
Cost of sales	-	-	-	-	-	-	-	-	-	-	-	8.1	56.5
Research and development	0.5	5.0	13.8	25.0	45.0	60.8	72.9	80.2	88.2	97.0	106.7	117.4	129.1
General and administrative	1.2	7.7	14.5	25.0	35.0	38.5	42.4	46.6	55.9	67.1	80.5	96.6	106.3
Operating Income (Loss)	**(1.72)**	**(12.7)**	**(27.1)**	**(41.0)**	**(71.0)**	**(90.2)**	**(106.2)**	**(119.2)**	**(134.7)**	**(152.3)**	**(172.5)**	**(155.9)**	**63.6**
Other expense / Income	(0.0)	(0.9)	(38.3)	(1.0)	(1.1)	(1.1)	(1.2)	(1.2)	(1.3)	(1.3)	(1.4)	(1.5)	(1.6)
Interest income	-	-	0.1	1.5	2.0	1.3	0.4	1.6	0.4	1.6	0.1	1.8	0.3
Interest expense	-	(0.0)	(0.1)	(0.1)	(0.1)	(0.1)	(0.1)	(0.1)	(0.1)	(0.1)	(0.1)	(0.1)	(0.1)
Pretax Income (Loss)	**(1.7)**	**(13.6)**	**(65.4)**	**(40.6)**	**(70.1)**	**(90.1)**	**(107.1)**	**(118.9)**	**(135.7)**	**(152.2)**	**(173.9)**	**(155.7)**	**62.2**
Income Taxes (expense) benefit	-	-	-	-	-	-	-	-	-	-	-	-	(21.8)
Tax Rate	*0.0%*	*0.0%*	*0.0%*	*0.0%*	*0.0%*	*0.0%*	*0.0%*	*0.0%*	*0.0%*	*0.0%*	*0.0%*	*0.0%*	*35.0%*
Net Income (Loss) before preferred stock ac	**(1.7)**	**(13.6)**	**(65.4)**	**(40.6)**	**(70.1)**	**(90.1)**	**(107.1)**	**(118.9)**	**(135.7)**	**(152.2)**	**(173.9)**	**(155.7)**	**40.4**
Convertible preferred stock preferences and cc	(0.0)	(0.3)	(0.3)	-	-	-	-	-	-	-	-	-	-
Net Income (Loss), non-GAAP	**(1.8)**	**(13.9)**	**(65.7)**	**(40.6)**	**(70.1)**	**(90.1)**	**(107.1)**	**(118.9)**	**(135.7)**	**(152.2)**	**(173.9)**	**(155.7)**	**40.4**
Non-GAAP EPS (excluding options)	**($2.26)**	**($4.77)**	**($5.19)**	**($1.10)**	**($1.89)**	**($2.41)**	**($2.50)**	**($2.47)**	**($2.54)**	**($2.60)**	**($2.69)**	**($2.20)**	**$0.47**
Consensus EPS													
Options Expense	0.0	0.1	2.6	9.2	10.1	11.1	12.2	13.4	14.8	16.2	17.9	19.6	21.6
% of operating expense	1.2%	0.4%	9.2%	18.3%	12.6%	11.2%	10.6%	10.6%	10.2%	9.9%	9.5%	8.8%	7.4%
Tax impact	-	-	-	-	-	-	-	-	-	-	-	-	-
Net Income (non-GAAP, incl option exp)	(1.8)	(14.0)	(68.3)	(49.8)	(80.2)	(101.2)	(119.3)	(132.4)	(150.4)	(168.4)	(191.8)	(175.3)	18.8
EPS, diluted (non-GAAP, incl option exp)	($2.28)	($4.79)	($5.40)	($1.35)	($2.17)	($2.70)	($2.79)	($2.75)	($2.82)	($2.88)	($2.96)	($2.47)	$0.22

Source: Company Data, Morgan Stanley Research

Balance Sheet

Exhibit 30: Editas Medicine Balance Sheet

Editas Medicine Balance Sheet ($M)	2013	2014	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024	2025
Cash and ST Investments:	**2.0**	**10.6**	**150.2**	**203.8**	**134.5**	**43.2**	**164.8**	**43.0**	**164.9**	**14.8**	**180.3**	**30.6**	**11.1**
Cash and cash equivalents	2.0	10.6	150.2	203.8	134.5	43.2	164.8	43.0	164.9	14.8	180.3	30.6	11.1
Accounts receivables	-	-	-	-	-	-	-	-	-	-	-	-	-
Prepaid expenses	0.0	0.1	0.6	3.6	2.7	1.8	1.8	1.5	1.9	2.4	2.9	6.6	35.6
Preferred stock tranche asset	0.1	-	-	-	-	-	-	-	-	-	-	-	-
Total current assets	2.1	10.7	150.8	207.4	137.2	45.0	166.7	44.5	166.8	17.2	183.2	37.2	46.7
Property and equipment, net	0.1	1.1	2.2	5.6	8.8	11.8	14.7	16.8	19.5	22.9	27.2	32.0	64.6
Other non-current assets	0.3	0.4	0.4	0.4	0.4	0.4	0.4	0.4	0.4	0.4	0.4	0.4	0.4
Restricted cash	-	-	-	-	-	-	-	-	-	-	-	-	-
Total Assets	**2.5**	**12.2**	**153.3**	**213.3**	**146.4**	**57.2**	**181.7**	**61.6**	**186.6**	**40.4**	**210.8**	**69.5**	**111.6**
Current liabilities:													
Accounts payable	0.4	2.6	2.1	3.8	6.0	7.4	8.6	9.5	10.8	12.3	14.0	16.1	11.8
Accrued expenses	0.7	1.6	3.5	6.3	10.0	12.4	14.4	15.8	18.0	20.5	23.4	26.8	23.5
Deferred rent, current portion	-	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1
Anti-dilution protection liability	-	0.3	0.3	0.3	0.3	0.3	0.3	0.3	0.3	0.3	0.3	0.3	0.3
Preferred stock tranche liability	1.0	1.5	-	-	-	-	-	-	-	-	-	-	-
Equipment loan, current portion, net of discount	-	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1
Total current liabilities	2.1	6.2	6.2	10.5	16.5	20.3	23.5	25.8	29.3	33.3	37.9	43.3	35.8
Deferred rent, net of current portion	-	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1
Equipment loan, net of current portion and discount	-	0.3	0.3	0.3	0.3	0.3	0.3	0.3	0.3	0.3	0.3	0.3	0.3
Deferred revenue	-	-	25.2	20.1	15.1	10.1	5.0	-	-	-	-	-	-
Warrant liability	-	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Other long-term liabilities	0.0	0.1	0.2	1.4	1.4	1.4	1.4	1.1	1.4	1.8	2.2	9.9	17.8
Total liabilities	**2.1**	**6.7**	**32.0**	**32.5**	**33.4**	**32.2**	**30.4**	**27.5**	**31.2**	**35.6**	**40.6**	**53.7**	**54.1**
Stockholders' equity:													
Redeemable convertible preferred stock	2.1	20.8	199.8	-	-	-	-	-	-	-	-	-	-
Share capital	-	-	-	-	-	-	-	-	-	-	-	-	-
Additional paid-in capital	-	0.2	5.3	314.4	326.7	339.8	585.5	600.7	872.4	890.2	1,247.3	1,268.3	1,291.2
Accumulated deficit	(1.8)	(15.4)	(83.8)	(133.5)	(213.7)	(314.9)	(434.2)	(566.5)	(717.0)	(885.4)	(1,077.1)	(1,252.4)	(1,233.6)
Total stockholders' equity	**0.3**	**5.5**	**121.3**	**180.9**	**113.0**	**24.9**	**151.3**	**34.2**	**155.4**	**4.9**	**170.1**	**15.8**	**57.6**
Total Liabilities and Stockholders' Equity	**2.5**	**12.2**	**153.3**	**213.3**	**146.4**	**57.2**	**181.7**	**61.6**	**186.6**	**40.4**	**210.8**	**69.5**	**111.6**

Source: Company Data, Morgan Stanley Research

Cash Flow Statement

Exhibit 31: Editas Medicine Cash Flow Statement

Editas Medicine Cash Flow Statement ($M)	2013	2014	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024	2025
Cash Flows From Operating Activities													
Net profit (loss)	(1.8)	(13.7)	(68.3)	(49.8)	(80.2)	(101.2)	(119.3)	(132.4)	(150.4)	(168.4)	(191.8)	(175.3)	18.8
Adjustments to reconcile net loss to net cash used in operating activities:													
Stock-based compensation	0.0	0.1	2.6	9.2	10.1	11.1	12.2	13.4	14.8	16.2	17.9	19.6	21.6
Depreciation	0.0	0.2	0.1	0.2	0.4	0.6	0.7	0.9	1.1	1.3	1.6	1.9	2.9
Non-cash research and development expenses	-	0.7	-	-	-	-	-	-	-	-	-	-	-
Non-cash interest expense	-	0.0	-	-	-	-	-	-	-	-	-	-	-
Changes in fair value of warrant liability	-	(0.0)	-	-	-	-	-	-	-	-	-	-	-
Change in fair value of preferred stock tranche asset or lia	0.0	0.9	-	-	-	-	-	-	-	-	-	-	-
Changes in fair value of anti-dilutive protection liability	-	0.0	-	-	-	-	-	-	-	-	-	-	-
Changes in deferred rent	-	0.2	-	-	-	-	-	-	-	-	-	-	-
Changes in operating assets and liabilities:													
Accounts receivable	-	-	-	-	-	-	-	-	-	-	-	-	-
Prepaid expenses	(0.0)	(0.1)	(0.5)	(3.0)	0.9	0.9	-	0.3	(0.4)	(0.5)	(0.6)	(3.7)	(28.9)
Other non-current assets	(0.3)	(0.0)	-	-	-	-	-	-	-	-	-	-	-
Accounts payable	0.4	2.2	(0.5)	1.6	2.3	1.4	1.2	0.9	1.3	1.5	1.7	2.0	(4.3)
Accrued expenses	0.7	0.9	1.9	2.7	3.8	2.4	2.0	1.4	2.2	2.5	2.9	3.3	(3.2)
Other liabilities	-	-	(1.5)	-	-	-	-	-	-	-	-	-	-
Upfront payments from collaborations	-	-	25.2	-	-	-	-	-	-	-	-	-	-
Deferred revenue offset	-	-	-	(5.0)	(5.0)	(5.0)	(5.0)	(5.0)	-	-	-	-	-
Net cash used in operating activities	(0.9)	(8.7)	(41.0)	(44.1)	(67.8)	(89.8)	(108.1)	(120.4)	(131.5)	(147.3)	(168.3)	(152.1)	7.0
Cash Flows From Investing Activities													
Purchase of property, plant, and equipment	(0.1)	(1.2)	(1.1)	(3.6)	(3.6)	(3.6)	(3.6)	(3.0)	(3.8)	(4.7)	(5.9)	(6.6)	(35.6)
Increase in restricted cash	-	-	-	-	-	-	-	-	-	-	-	-	-
Net cash used in investing activities	(0.1)	(1.2)	(1.1)	(3.6)	(3.6)	(3.6)	(3.6)	(3.0)	(3.8)	(4.7)	(5.9)	(6.6)	(35.6)
Cash Flows From Financing Activities													
Proceeds from equipment loan, net of issuance costs	-	0.5	-	-	-	-	-	-	-	-	-	-	-
Proceeds from the issuance of redeemable convertible pre	3.0	18.0	179.0	-	-	-	-	-	-	-	-	-	-
Payments of equipment loan principal	-	-	-	-	-	-	-	-	-	-	-	-	-
Proceeds from the issuance of common stock and restricte	0.0	0.0	2.5	100.1	2.2	2.1	233.5	1.8	256.9	1.6	339.2	1.4	1.3
Other liabilities	-	-	0.1	1.2	-	-	-	(0.2)	0.3	0.4	0.4	7.7	7.8
Net cash provided by financing activities	3.0	18.5	181.7	101.3	2.2	2.1	233.5	1.6	257.2	1.9	339.6	9.1	9.1
Effect of exchange rate changes on cash and cash equiva	-	-	-	-	-	-	-	-	-	-	-	-	-
Net (decrease) increase in cash and cash equivalents	2.0	8.6	139.6	53.6	(69.3)	(91.4)	121.7	(121.9)	122.0	(150.1)	165.4	(149.6)	(19.5)
Cash and cash equivalents at beginning of period	-	2.0	10.6	150.2	203.8	134.5	43.2	164.8	43.0	164.9	14.8	180.3	30.6
Cash and cash equivalents at end of period	2.0	10.6	150.2	203.8	134.5	43.2	164.8	43.0	164.9	14.8	180.3	30.6	11.1

Source: Company Data, Morgan Stanley Research

Analysis

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	COVERAGE UNIVERSE		INVESTMENT BANKING CLIENTS (IBC)		
STOCK RATING CATEGORY	COUNT	% OF TOTAL	COUNT	% OF TOTAL IBC	% OF RATING CATEGORY
Overweight/Buy	**1206**	**36%**	**323**	**43%**	**27%**
Equal-weight/Hold	**1432**	**42%**	**331**	**44%**	**23%**
Not-Rated/Hold	**79**	**2%**	**9**	**1%**	**11%**
Underweight/Sell	**658**	**19%**	**86**	**11%**	**13%**
TOTAL	**3,375**		**749**		

Data include common stock and ADRs currently assigned ratings. Investment Banking Clients are companies from whom Morgan Stanley received investment banking compensation in the last 12 months.

Analyst Stock Ratings

Overweight (O). The stock's total return is expected to exceed the average total return of the analyst's industry (or industry team's) coverage universe, on a risk-adjusted basis, over the next 12-18 months.
Equal-weight (E). The stock's total return is expected to be in line with the average total return of the analyst's industry (or industry team's) coverage universe, on a risk-adjusted basis, over the next 12-18 months.
Not-Rated (NR). Currently the analyst does not have adequate conviction about the stock's total return relative to the average total return of the analyst's industry (or industry team's) coverage universe, on a risk-adjusted basis, over the next 12-18 months.
Underweight (U). The stock's total return is expected to be below the average total return of the analyst's industry (or industry team's) coverage universe, on a risk-adjusted basis, over the next 12-18 months.
Unless otherwise specified, the time frame for price targets included in Morgan Stanley Research is 12 to 18 months.

Analyst Industry Views

Attractive (A): The analyst expects the performance of his or her industry coverage universe over the next 12-18 months to be attractive vs. the relevant broad market benchmark, as indicated below.
In-Line (I): The analyst expects the performance of his or her industry coverage universe over the next 12-18 months to be in line with the relevant broad market benchmark, as indicated below.
Cautious (C): The analyst views the performance of his or her industry coverage universe over the next 12-18 months with caution vs. the relevant broad market benchmark, as indicated below.
Benchmarks for each region are as follows: North America - S&P 500; Latin America - relevant MSCI country index or MSCI Latin America Index; Europe - MSCI Europe; Japan - TOPIX; Asia - relevant MSCI country index or MSCI sub-regional index or MSCI AC Asia Pacific ex Japan Index.

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INDUSTRY COVERAGE: **Biotechnology**

COMPANY (TICKER)	RATING (AS OF)	PRICE* (02/26/2016)
Andrew S Berens		
Acceleron Pharma Inc (XLRN.O)	O (08/13/2015)	$25.32
Akebia Therapeutics Inc (AKBA.O)	O (09/09/2015)	$7.47
Cempra Inc (CEMP.O)	O (11/16/2015)	$17.66
GW Pharmaceuticals PLC (GWPH.O)	O (08/13/2015)	$41.60
Intercept Pharmaceuticals Inc (ICPT.O)	E (01/29/2016)	$113.90
Keryx Biopharmaceuticals Inc (KERX.O)	E (10/05/2015)	$3.64
Ocular Therapeutix Inc (OCUL.O)	O (02/17/2016)	$8.07
Relypsa, Inc. (RLYP.O)	U (08/13/2015)	$14.60
Rockwell Medical Inc (RMTI.O)	U (08/13/2015)	$9.98
Versartis, Inc. (VSAR.O)	E (08/13/2015)	$6.64
Matthew Harrison		
Alexion Pharmaceuticals (ALXN.O)	O (10/01/2015)	$140.16
Amgen Inc. (AMGN.O)	O (12/14/2015)	$147.60
Biogen Inc (BIIB.O)	O (03/26/2014)	$264.49
Bluebird Bio Inc (BLUE.O)	E (12/07/2015)	$49.73
Celgene Corp (CELG.O)	E (03/26/2014)	$103.37
Chimerix Inc (CMRX.O)	U (02/22/2016)	$4.72
DBV Technologies SA (DBVT.O)	O (09/15/2015)	$25.70
Editas Medicine (EDIT.O)	E (02/29/2016)	$27.49
Galapagos NV (GLPG.O)	O (06/08/2015)	$42.18
Gilead Sciences Inc. (GILD.O)	E (10/01/2015)	$88.10
Global Blood Therapeutics Inc (GBT.O)	O (09/08/2015)	$14.87
ImmunoGen Inc. (IMGN.O)	U (09/21/2015)	$7.21
Infinity Pharmaceuticals Inc (INFI.O)	O (09/21/2015)	$6.21
Innoviva Inc (INVA.O)	U (08/14/2014)	$11.62
Ironwood Pharmaceuticals, Inc. (IRWD.O)	E (08/14/2014)	$9.58
Juno Therapeutics Inc (JUNO.O)	E (01/13/2015)	$36.67
MacroGenics Inc (MGNX.O)	E (02/25/2016)	$15.82
Ophthotech Corp (OPHT.O)	O (08/14/2014)	$46.71
Portola Pharmaceuticals Inc (PTLA.O)	O (08/14/2014)	$27.79
Regeneron Pharmaceuticals Inc. (REGN.O)	E (10/01/2015)	$394.23
Regenxbio Inc (RGNX.O)	O (10/12/2015)	$13.08
Ultragenyx Pharmaceutical Inc (RARE.O)	E (07/27/2015)	$62.30
Vertex Pharmaceuticals (VRTX.O)	O (10/01/2015)	$87.00

Stock Ratings are subject to change. Please see latest research for each company.
* Historical prices are not split adjusted.