Fate Therapeutics

(FATE-NASDAQ)

Stock Rating: **Outperform**
Industry Rating: **Outperform**

October 28, 2013

Jim Birchenough, M.D. **415-591-2129**
BMO Capital Markets Corp
jim.birchenough@bmo.com

Chuck Whitesell / Nick Abbott, PhD
BMO Capital Markets Corp
212-885-4120 / 206-542-2492
charles.whitesell@bmo.com /
nicholas.abbott@bmo.com

Initiating With Outperform Rating

Investment Thesis

We are initiating coverage of Fate Therapeutics with an Outperform rating and an $11 price target. Our positive rating is supported by Fate's proprietary expertise in ex-vivo and in-vivo stem cell modulation and by a stem cell platform that is leveragable across multiple orphan disease indications. While primary focus has been on phase 2 development of ex-vivo modulated stem cell product ProHema for stem cell transplant (SCT) in hematologic malignancy, we believe that greater value may exist for earlier stage programs for ProHema in Hurler's Syndrome as well as Wnt7a protein analogs in Muscular Dystrophy. Indeed, we believe that review of pre-clinical data for Fate's Wnt7a protein analog in models of muscular dystrophy demonstrate unique benefits in increasing muscle mass, increasing force of muscle contraction independent of muscle mass and changing the muscle biology to a more disease resistant phenotype. Similarly, published data for umbilical cord blood stem cells in lysosomal storage diseases like Hurler's syndrome may provide validation for pre-clinical stage efforts for ProHema in this area. Thus, while near-term attention may be on phase 2 development of ProHema in hematologic malignancy, we view this program as pure option value and would look to phase 1 initiation for the Wnt7a protein analog in Muscular Dystrophy and ProHema in Hurler's syndrome as greater value drivers.

Forecast & Valuation

We forecast losses per share of $1.60 in 2013 and $1.43 in 2014. We expect decreasing losses annually until 2020, when we estimate initial profitability with EPS of $1.68. We arrive at our $11 price target by applying a 25x multiple to 2020 EPS estimate of $1.68 and discounting at 30%.

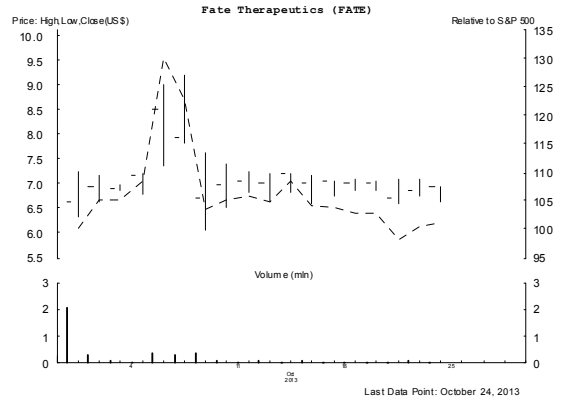
Recommendation

We rate FATE shares at Outperform.

Securities Info

Price (25-Oct)	**$6.87**	**Target Price**	**$11.00**
52-Wk High/Low	$9/$6	Dividend	--
Mkt Cap (mm)	$143	Yield	--
Shs O/S (mm, BASIC)	20.8	Float O/S (mm)	7.6
Options O/S (mm)	na	ADVol (30-day, 000s)	245

Price Performance



Valuation/Financial Data

(FY-Dec.)	2011A	2012A	2013E	2014E
EPS Pro Forma	-$2.49	-$0.30	-$1.60	-$1.43
P/E			nm	nm
First Call Cons.				
EPS GAAP	-$2.49	-$0.30	-$1.60	-$1.43
FCF	na	na	na	na
P/FCF			na	na
EBITDA ($mm)	-$13	-$14	-$17	-$30
EV/EBITDA			nm	nm
Rev. ($mm)	$1	$3	$1	$0
EV/Rev			102.4x	#DIV/0!

Quarterly EPS	**1Q**	**2Q**	**3Q**	**4Q**
2012A	na	na	na	na
2013E	-$0.57A	-$0.57A	-$0.23	-$0.23

Balance Sheet Data (30-Jun)			
Net Debt ($mm)	-$61	Total Debt/EBITDA	nm
Total Debt ($mm)	$1	EBITDA/IntExp	na
Net Debt/Cap.	nm	Price/Book	-0.2x

Notes: All values in US$.

Source: BMO Capital Markets estimates, Bloomberg, Thomson Reuters, and IHS Global Insight.

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Investment Thesis

Fate Therapeutics (FATE) expertise in both in-vivo and ex-vivo stem cell modulation should be leveragable across multiple orphan indications, including stem cell transplant in cancer, lysosomal storage diseases, and muscular dystrophies. We believe that upside potential from current valuation levels exists from progress into clinical development with its Wnt7a protein analog in muscular dystrophy alone, and that strong pre-clinical data should bode well for clinical proof-of-concept. While further ahead in development, we believe that ex-vivo modulated stem cell product ProHema in stem cell transplant (SCT) for hematologic malignancy is a more binary opportunity, which should be considered as option value beyond Wnt7a protein analogs.

Key Investment Highlights

- Fate Therapeutics (FATE) is an early-stage biotechnology company focused on stem cell therapy for cancer and orphan diseases.
- Platform focused on modulation of existing stem cells to optimize therapeutic effects for stem cell transplant in cancer and actual treatment of orphan disease.
- Potential for clinical proof-of-concept in 3 separate indications within 18-24 months (ProHema Cancer, ProHema Hurler's, Wnt7a muscular dystrophy).
- Lead product ProHema is designed to treat cord blood stem cells before use in stem cell transplant to improve success rates.
- ProHema cord blood stem cells, modulated chemically with Fate's FT-1050, demonstrate 2x better homing to bone marrow and 4x better engraftment pre-clinically.
- Interim phase 2 data for ProHema in stem cell transplant for patients with blood cancers expected in 2014.
- ProHema phase 1b data demonstrated 4.5 day improvement in bone marrow recovery (17.5 days vs. 22 days), with time to bone marrow recovery as phase 2 and approval endpoint.
- ProHema also being evaluated for rare genetic diseases like Hurler's syndrome with phase 1 clinical validation data possible in 2015.
- ProHema potential in rare genetic diseases has been validated with published data on success with cord blood stem cells in Hurler's disease.
- Earlier pre-clinical program focused on Wnt7a analog to stimulate muscle regeneration in muscular dystrophy.
- Wnt7a analog has demonstrated in mice a four-fold increase in muscle stem cells, 15%-20% increase in muscle mass and 18% increase in muscle force.

Risks

- Risks to investing in early-stage biotechnology stocks include, but are not limited to risk of clinical trial failure, risk of unforeseen safety issues, risk to manufacturing, regulatory risk and commercialization risks. Several specific risks to FATE are included below.
- Phase 1b data for ProHema is limited by small patient numbers and has wide range of outcomes in terms of time to bone marrow recovery beyond apparent median benefit.
- Change in ProHema culture media in phase 2 introduces a new variable from phase 1b experience.
- Difficult to generalize pre-clinical data with Wnt7a modulator in muscular dystrophy to clinical benefit.
- Early-stage nature of both programs limits safety experience.

Review of Financials

We estimate initial launch of ProHema in the US in 2018 with $12M in estimated sales, increasing to $278M in 2025. We estimate and ex-US ProHema launch in 2019 with estimated sales of $5M, increasing to $91M in 2025. We anticipate a US launch of Wnt7A in 2020 with sales of roughly $65M, increasing to roughly $600M by 2025. For the ex-US, we expect a Wnt7A launch in 2021 with sales of roughly $55M, increasing to $455M by 2025. We forecast initial profitability for FATE in 2020 with EPS of $1.68.

Valuation

We base our valuation of FATE on a relative value P/E multiple on future earnings. We arrive at our $11 price target by applying a 25x multiple to 2020 EPS estimate of $1.68 and discounting at 30% per year. Our $11 price target can also be supported by the probability adjusted NPV of the Wnt7a protein analog program in muscular dystrophy alone, assuming peak sales of ~$1B and 12.5% probability of success.

Exhibit 1: Fate Therapeutics Comps

EARLY STAGE ONCOLOGY/ORPHAN DISEASE COMPANIES

Company	Ticker	Market Cap (M)	Cash (M)	EV (M)	Stage of Development	Therapeutic Focus
Ambit Biosciences	AMBI	$292.3	$85.3	$208.8	Phase 2	Oncology
Array BioPharma	ARRY	$636.7	$108.2	$627.5	Phase 2	Oncology
Curis	CRIS	$353.7	$57.1	$327.1	Phase 1	Oncology
Epizyme	EPZM	$1,103.2	$148.7	$954.5	Phase 1	Oncology
KaloBios	KBIO	$135.3	$63.7	$81.6	Phase 1	Oncology
OncoMed Pharmaceuticals	OMED	$429.1	$60.2	$368.9	Phase 1	Oncology
Stemline Therapeutics	STML	$385.6	$92.7	$292.9	Phase 2	Oncology
Verastem	VSTM	$271.6	$57.5	$214.1	Phase 2	Oncology
bluebird bio	BLUE	$543.2	$131.8	$411.4	Phase 2	Cerebral ALD
PTC Therapeutics	PTCT	$510.8	$50.2	$464.4	Phase 3	Muscular Dystrophy
Regulus Therapeutics	RGLS	$305.4	$82.7	$237.3	Preclinical	Alport Syndrome
Sarepta Therapeutics	SRPT	$1,447.4	$163.4	$1,285.7	Phase 2	Muscular Dystrophy
Mean		$534.5		$456.2		
Median		407.4		348.0		
Fate Therapeutics	FATE	$139.4	$61.7	$78.4	Preclinical	Transplant/Musc Dys

FATE THERAPEUTICS SUM-OF-THE-PARTS

Target	Indication	Launch Year	Peak Sales ($M)	Probability	NPV ($M)
Wnt7A - U.S.	Muscular Dystrophy	2020	598.8	12.5%	127.5
Wnt7A - Ex-U.S.	Muscular Dystrophy	2021	455.0	12.5%	85.0
Total					$212.5

Source: Company reports, Thomson Reuters, and BMO Capital Markets

Company Overview

Fate Therapeutics is a developmental-stage biopharmaceutical company specialized in the discovery and development of pharmacologic modulators of adult stem cells for the treatment of various orphan diseases, including certain hematologic malignancies, lysosomal storage disorders, and muscular dystrophies.

Fate's core drug-development strategy involves leveraging established pharmacologic modalities, including both small molecule and protein therapeutics that target well-characterized biological pathways to optimize the therapeutic potential of adult stem cells.

Fate has built two stem-cell modulation platforms, the hematopoietic stem cell (HSC) modulation platform and the satellite stem cell (SSC) modulation platform. Product candidates generated from these platforms are under development for various orphan indications, with a potential to yield four clinical data readouts by the end of 2015.

The HSC modulation platform focuses on improving patient outcomes in hematopoietic stem cell transplant (HSCT) through *ex vivo* (outside the body) pharmacological modulation of HSCs. HSCs are adult stem cells that can regenerate all types of blood cells, and HSCT is a potentially curative procedure for patients with hematologic malignancies. Fate's novel approach of pharmacological optimization of HSCs can potentially enhance hematopoietic reconstitution through accelerated and durable engraftment, thereby permitting greater donor matching flexibility, reducing the risk of major side effects and enabling the use of less toxic conditioning regimens.

The lead product candidate from the HSC modulation platform is ProHema, a pharmacologically modulated HSC therapeutic derived from umbilical cord blood. In preclinical studies, treatment with FT1050, a long-lasting form of prostaglandin E2, resulted in a 2x increase in homing of HSCs to bone marrow and a 4x increase in long-term HSC engraftment. FT1050 treatment also increased the survival outcomes in a mouse model of HSCT.

In a phase 1b trial, ProHema demonstrated a median time to neutrophil recovery of 17.5 days (versus 20.5 days historical) in double-umbilical cord blood transplants in patients with hematologic malignancies following reduced-intensity conditioning therapy. ProHema also demonstrated preferential reconstitution compared with unmanipulated cord blood. ProHema was also described as well tolerated in the phase 1b experience.

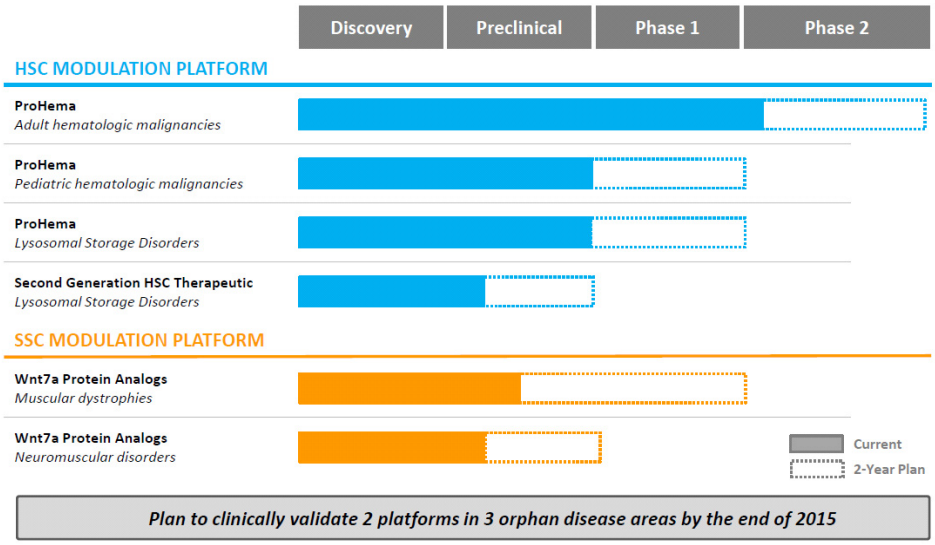
Fate initiated a phase 2 trial of ProHema in the setting of umbilical cord blood transplant in December 2012, but recently paused the enrollment of the trial to enable the manufacture of ProHema using an improved formulation (NRM formulation). Eleven patients had been treated in the phase 2 trial at 8 major allogeneic HSCT centers prior to the pause of enrollment. Seven of 8 patients in the ProHema arm engrafted, with a median time to engraftment of 28 days, as compared with 3 of 3 patients who engrafted in the control arm, with a median time of 31 days. Fate plans to resume enrollment of the phase 2 trial in 1H14, subject to the consent of FDA. Once resumed, the phase 2 trial will enroll and randomize 60 patients with hematologic

malignancies to ProHema (manufactured using the NRM formulation) plus an unmanipulated cord blood unit or two unmanipulated cord blood units, with the goal of generating full data for this trial in mid-2015.

Fate intends to explore ProHema in pediatric patients in the setting a single UCBT. As a first step, a phase 1 trial has been initiated in the setting of single UCBT in adult patients with hematologic malignancies. Of the six adult subjects that are evaluable to date, four subjects engrafted at days 17, 19, 22, and 37, and two subjects had primary graft failure; the 100-day survival rate was 100%. Fate plans to initiate a phase 1b trial in pediatric patients with hematologic malignancies in the single UCBT setting in 2014, subject to FDA consent.

Fate also plans to pursue the development of pharmacologically optimized HSC therapeutics for the treatment of certain lysosomal storage disorders (LSDs), where HSC engraftment in the central nervous system (CNS) represents a unique therapeutic advantage compared with standard enzyme replacement therapies, as most recombinant enzymes cannot cross the blood-brain barrier. In a preclinical study, Fate demonstrated that *ex vivo* modulated cord blood increased the number of donor cells that home and migrate across the blood-brain barrier into the CNS. Fate plans to initiate the first clinical trial of ProHema in pediatric patients with demyelinating LSDs in 2014 after filing an amendment to the existing IND (subject to FDA consent), with the goal of generating data from this trial in 2015. Fate also plans to develop second generation therapeutics specifically designed to enhance the homing of HSCs to the CNS to improve delivery of essential enzymes in patients with LSDs.

Exhibit 2: Fate Therapeutics Pipeline



Source: Fate Therapeutics

Fate's SSC modulation platform focuses on the *in vivo* (within the body) pharmacological activation of satellite stem cells (SSCs) for the treatment of muscle degeneration conditions, such as muscular dystrophies. Fate has identified Wnt7a as a driver of muscle regeneration and

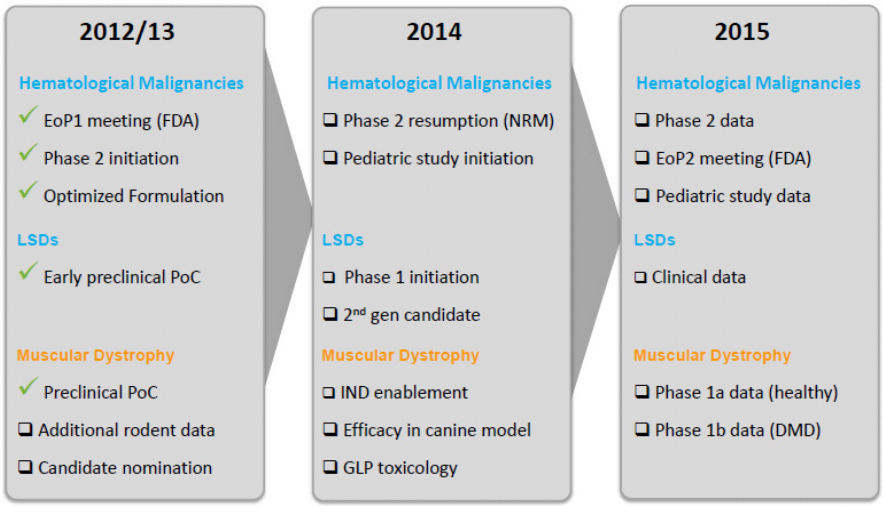
has developed injectable analogs of Wnt7a as recombinant human protein therapeutics with muscle regenerative activity.

In preclinical studies, injection of a Wnt7a analog led to a three-fold expansion of SSC population, a 20% increase in muscle hypertrophy, and an 18% increase in muscle strength in both wild-type mice and a rodent model of muscular dystrophy. A significant reduction in disease-related muscle damage was also observed. Wnt7a analogs demonstrated similar potency as naturally-occurring Wnt7a. In experiments involving *in vitro* cultures of myotubes derived from healthy volunteers and patients with Duchenne, Becker and facioscapulohumeral dystrophies, Wnt7a analogs induced muscle cell hypertrophy (increase in muscle cell size).

Fate's initial clinical focus for the Wnt7a analog program is to assess safety and demonstrate human proof-of-concept in X chromosome-linked dystrophy patients. Fate is currently selecting proprietary Wnt7a analogs with preferred efficacy, manufacturing and formulation characteristics, and will conduct an IND-enabling study in a canine model of muscular dystrophy in 2014. Fate plans to initiate a phase 1 clinical trial in healthy volunteers, with the goal of data readout in 2015. Fate plans to assess biomarkers and measures of muscle strength by electromyography in the phase 1 trial. Based on the results of the phase 1 trial, Fate plans to initiate a dose-escalation study in an X chromosome-linked muscular dystrophy patient population, such as DMD.

Notably, the validation of both of Fate's stem-cell modulation platforms involves orphan disease indications, including hematologic malignancies, LSDs and muscular dystrophies. These target indications may allow a fast-to-market strategy through small clinical trials and the use of relatively short-term efficacy endpoints. Fate also plans to pursue, where possible, Fast Track or Breakthrough Therapy designations. The highly specialized nature of these target markets also means that Fate could commercialize any products it successfully develops in a cost-effective manner.

Exhibit 3: Fate Therapeutics Milestones Through 2015



Source: Fate Therapeutics

Pipeline Focus on ProHema and Pharmacological Modulation of HSCs

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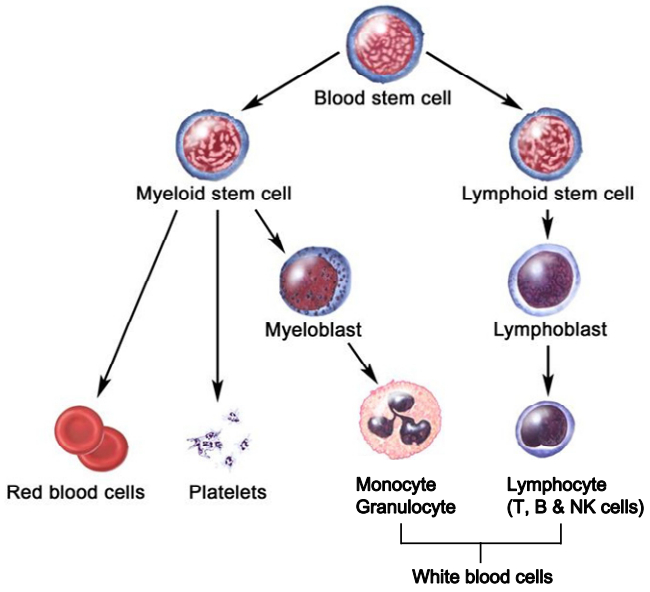
Overview of HSCT

Hematopoietic stem cell transplantation (HSCT) is a potentially curative procedure for diseases such as hematologic malignancies and rare congenital metabolic defects. First pioneered over 40 years ago, HSCT has undergone many improvements. However, presently allogenic HSCT is still associated with significant risks of morbidity and mortality, and further technological improvement is needed to address this significant unmet need.

Adult stem cells play a key role in the growth, maintenance and repair of tissues and organs in the body. Hematopoietic stem cells (HSCs) are adult stem cells in the bone marrow that give rise to all blood cell types, including red blood cells, platelets, and white blood cells. The white blood cells, including lymphocytes, monocytes, and granulocytes, are cells of the immune system that survey the body for signs of foreign pathogens and diseased cells and mount immune responses when necessary. Thus the blood system and the immune system are inextricably linked.

Exhibit 4: HSCs Give Rise to Hematopoietic Lineages



Source: National Cancer Institute

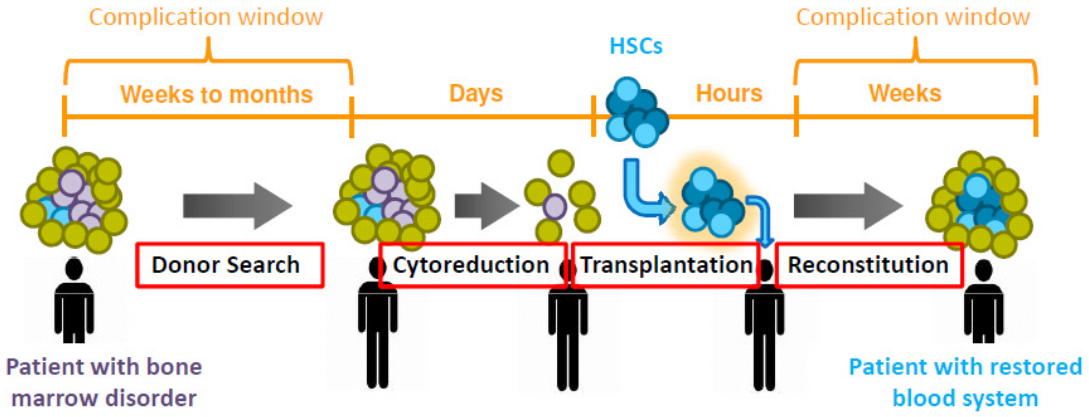
Hematopoietic stem cell transplant (HSCT) involves the intravenous infusion of HSCs of autologous (self) or allogenic (matched donor) origin to reestablish hematopoietic function in patients with damaged or defective bone marrow or immune system. The most common indications for HSCT are hematologic malignancies: over half of the autologous transplantations are performed for multiple myeloma and non-hodgkin lymphoma, and a vast majority of allogenic transplants are performed for hematologic and lymphoid cancers. Other

indications for HSCT include immune-deficiency disorders, as well as congenital metabolic defects whereby HSCT is performed to replace bone marrow cells for the purpose of making corrective enzymes.

In autologous HSCT, a subject's own HSCs are recovered from bone marrow or peripheral blood for transplant. In the allogeneic setting, matched HSCs are obtained from bone marrow or peripheral blood of a donor or from blood taken from the umbilical cord of newborns. According to the Center for International Blood & Marrow Transplant Research (CIBMTR), approximately 55% of HSCT activities in the US in 2011 were autologous HSCT, and the rest were allogenic HSCT with sibling donors (~20%) or unrelated donors (~25%).

Typical allogenic HSCT procedures consist of the following stages: donor search, patient conditioning (also called cytoreduction), transplantation, and reconstitution. Treatment-related morbidity and mortality can be significantly influenced by important factors presented at each stage.

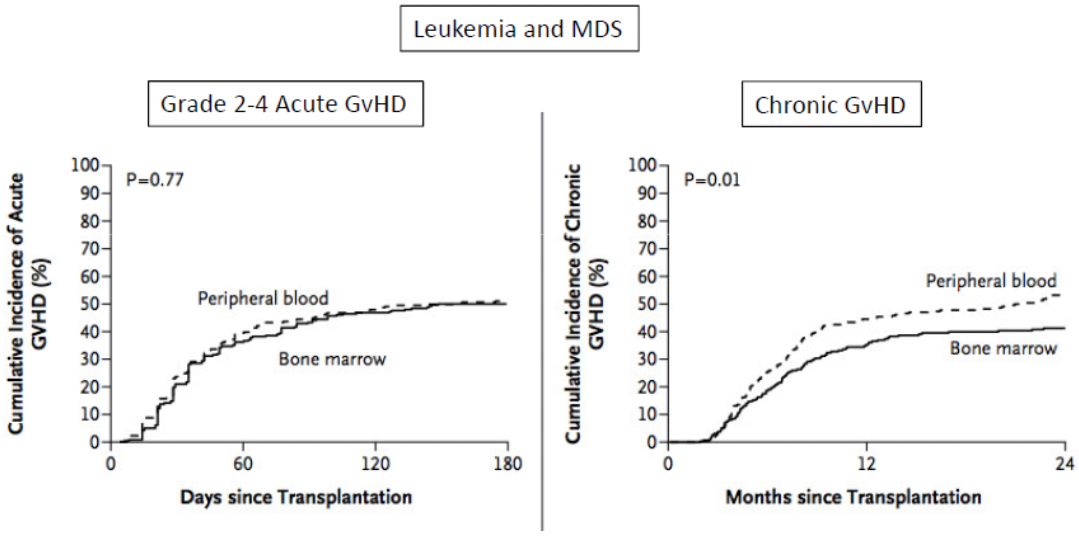
Exhibit 5: Schematic Depiction of Stages of HSCT



Source: Fate Therapeutics

The degree to which a donor's and recipient's tissues are immunologically compatible, termed human leukocyte antigen (HLA) matching, is a critical determinant of clinical outcomes for allogenic HSCT. The most ideal donor for any patient would be a related matched donor. If the donor-derived immune system is not sufficiently compatible with the recipient's tissue, a serious complication known as graft-versus-host disease (GvHD) may occur. In GvHD, the donor-derived immune cells recognize host antigens as foreign targets and mount an immune reaction. GvHD may be acute (developing within the first 100 days) or chronic (developing beyond 100 days). Approximately half of patients who undergo HSCT procedures develop acute GvHD and 25-50% of patients develop chronic GvHD (Exhibit 6). According to CIBMTR, GvHD is responsible for approximately 17% of deaths in allogenic HSCT.

Exhibit 6: Risk of Acute and Chronic GvHD in HSCT



Source: Fate Therapeutics

Greater HLA mismatch also increases the risk of engraft failure. Engraftment refers to the localization and integration of HSCs within a targeted tissue where they can produce new cells.

Successful transplants also require a sufficient number of HSCs in order to ensure timely engraftment. While an adequate number of HSCs can usually be collected from healthy adults donating bone marrow or mobilized peripheral blood, some HSC collections may be suboptimal. Cord blood units generally contain fewer HSCs than traditional HSC sources, and as a result, additional cycles of proliferation (cell division) are required to reconstitute the host. This could result in delayed engraftment and a higher risk of failure.

Prior to allogeneic HSCT, it is necessary to eradicate the hematopoietic cells in a patient, either for the purpose of treating the underlying disease or to make room for donor-derived HSCs to reconstitute a healthy hematopoietic system. This process is referred to as patient conditioning or cytoreduction, and could involve chemotherapy, radiation therapy, or immunotherapy.

Traditionally, HSCT has required intense myeloablative conditioning (MAC), a highly toxic procedure associated high rates of transplant-related morbidity. For this reason, only younger (<45-50 years old) and healthier patients are considered eligible for MAC. More recently, investigators have developed reduced-intensity conditioning (RIC) regimens that involve significantly lower doses of chemotherapy or radiation. The lower early morbidity and mortality associated with the RIC regimen have allowed the upper age limit for HSCT to be increased to 65+ years. However, RIC regimens are associated with lower rates of engraftment and higher rates of relapse.

The reconstitution of a patient's hematopoietic system by transplanted HSCs takes several weeks to several months, and different components of the hematopoietic system return to

normal levels at different rates. The engraftment of neutrophils, a type of white blood cell involved in fighting bacterial infections, is the first clinical milestone following HSCT, and is required for patient discharge. More than 70% of patients undergoing HSCT experience serious infections. Failure to achieve neutrophil engraftment by day 30 is the single strongest predictor of infection-related mortality, conferring a 3.9-fold increased risk. Failure to achieve neutrophil engraftment by day 30 also correlates with a >2.5-fold increased risk in 100-day treatment-related mortality. Therefore time to engraftment, especially neutrophil engraftment, correlates with key clinical outcomes, including rates of serious infections and overall transplant-related morbidity and mortality.

Overall, allogeneic HSCT is associated with significant risk, with a 100-day mortality rate of 20%-30%. Therefore there is a significant need to improve patient outcomes in HSCT procedures. A promising strategy that could positively impact outcomes is the pharmacological optimization of HSCs, which is being pursued by Fate through its lead product candidate, ProHema.

Advantages and Limitations of Cord Blood as a Source of HSCs

There are three possible sources of stem cells to use for transplants: bone marrow, peripheral blood, and umbilical cord blood from newborns. While bone marrow was the first source used in stem cell transplant, peripheral blood is the most common source today, and the use of cord blood is rapidly growing.

Bone marrow contains a rich supply of stem cells and was the original source of HSCs used in HSCT. The donor receives general anesthesia and a large needle is put into the back of the pelvic (hip) bone and the thick, liquid marrow is extracted through the needle. The hip bone is preferred because it contains the most bone marrow. The harvested marrow is filtered and stored frozen until the day of transplant. Once transplanted, new blood cells derived from the bone marrow HSCs can be measured in the patient's blood in about 2 to 4 weeks.

Peripheral blood stem cells are the most often used source of HSCs in HSCT. Normally, few stem cells are found in the peripheral blood. However, administration of cytokines (growth factors), such as G-CSF, can increase the number of stem cells released into the blood stream, and the stem cells in the peripheral blood can be harvested in a process called apheresis. In apheresis, blood is removed through a large vein and passed through a machine that removes stem cells and returns the rest of the blood to the donor. Significant number of HSCs can be harvested this way; sometimes more HSCs can be harvested from peripheral blood than from bone marrow. Once transplanted, new blood cells derived from the peripheral blood HSCs are usually found in the patient's blood in about 10 to 20 days, a few days sooner than when bone marrow HSCs are used.

Cord blood now represents approximately 30% of unrelated hematopoietic stem cell transplant. The blood of newborn babies contains a large number of stem cells, and therefore the blood left behind in the placenta and umbilical cord (cord blood) after birth can be stored for later use in HSCT.

Cord blood has several distinct advantages as a source of HSCs compared with peripheral blood or bone marrow.

(1) Cord blood is more accessible, allowing more rapid time-to-transplant. There is no need to harvest cells from donors. Cord blood cells can be stored indefinitely in cryopreservation, whereas bone marrow is used fresh and peripheral blood stem cells are stored for a short period, usually a few days to a few months. The worldwide inventory of publicly banked cord blood units can be searched electronically. Result of the search is available within days and the cord blood can be delivered to the transplant center within 2 to 3 weeks. This overall reduction of time required for donor search could translate into better clinical outcomes, as a study in unrelated allogenic bone marrow/peripheral blood HSCT has demonstrated that the duration of donor search (or time-to-transplant) is an independent predictor of survival, with each month of delay leading to a 10% increase in mortality. Similarly, early transplantation is also beneficial in the treatment of congenital metabolic disorders: HSCT performed in a newborn with Krabbe's disease leads to near normalization of cognitive development, whereas a delay of 6+ months abolishes all therapeutic value.

(2) Cord blood HSCs are immunologically naïve and therefore are more tolerant of HLA mismatch compared with bone marrow or peripheral blood HSCs. Bone marrow and peripheral blood HSCTs typically require at least a 7 out of 8 match in 8 HLA markers (HLA-A, -B, -C and -DRB1 antigens). Cord blood HSCTs are more tolerant of HLA mismatch and the minimum requirement is a 4 out of 6 match in 6 HLA markers (HLA-A, -B and -DRB1). As a result, there is an expanded pool of potential cord blood donors for any one patient. In addition, the incidence and severity of GvHD are also reduced. Studies have demonstrated that cord blood HSCT is associated with a 25%-50% reduction in the risk of GvHD.

(3) A retrospective single-center study has demonstrated a lower risk of relapse (up to 50% reduction) in cord blood HSCT as compared with bone marrow or peripheral blood HSCT.

Despite the significant potential benefits, the use of cord blood in HSCT is currently constrained by the following factors.

(1) Limited number of cells per cord blood unit. The number of cells collected from a single donor (a *unit* of cord blood) is limited. It is commonly held that at least 2.5×10^7/kg cord blood cells is necessary to achieve consistent engraftment. Thus a single cord blood unit cannot provide enough cells for most adult patients. Indeed, most cord blood HSCTs are carried out in young patients as they require less cells because of lower body weight. Strategies have been explored to make cord blood more widely available, including *ex vivo* stem cell expansion and the pooling of two partially HLA-matched cord blood units in circumstances where a single unit is considered inadequate.

(2) Longer time to engraftment compared with bone marrow or peripheral blood. Because cord blood units contain fewer HSCs than traditional HSC sources, additional cycles of

proliferation are required to reconstitute the host. This could result in delayed engraftment and a higher risk of failure, as well as an increased risk of infection in the first two to three months after the transplant.

As summarized in Exhibit 7, when evaluated for their effectiveness in HSCT, cord blood scores high in the aspects of HLA-match requirement, time to acquire cells, relative risk of GvHD, and relative risk of relapse, whereas peripheral blood and bone marrow have a more favorable profile in time to engraftment, average patient stay, and graft failure rates. Fate's ProHema could potentially improve the biological properties of cord blood and expand its use in allogenic HSCT.

Exhibit 7: Comparison of HSC Sources for Use in HSCT

	RELATED	UNRELATED		
Cell Source Considerations	Matched Sibling	mPB / Bone Marrow	Cord Blood	Favors...
Pre-HSCT				
HLA Match Requirement	Full match	7+/8 HLA match	4+/6 HLA match	Cord Blood
Time to Acquire Cells	2-3 weeks	8-12 weeks	2-3 weeks	Cord Blood
Day 0 – Day 100				
Time to Engraftment	14-22 days	14-22 days	21-28 days	mPB / BM
Average Inpatient Stay	24-28 days	24-28 days	35 days	mPB / BM
Graft Failure Rates	3-5%	3-5%	10-15%	mPB / BM
Long-Term Outcomes				
Relative Risk of GvHD	Reduced	Reference	25-50% reduction [1]	Cord Blood
Relative Risk of Relapse	Reference	Reference	Up to 50% lower [2]	Cord Blood

Source: Fate Therapeutics

Rationale for PGE2-mediated Modulation of HSC

ProHema is a pharmacologically modulated HSC therapeutic derived from umbilical cord blood. ProHema has the potential to address the limitations of allogenic HSCT and enhance its curative potential across a broad range of hematologic malignancies and rare genetic disorders.

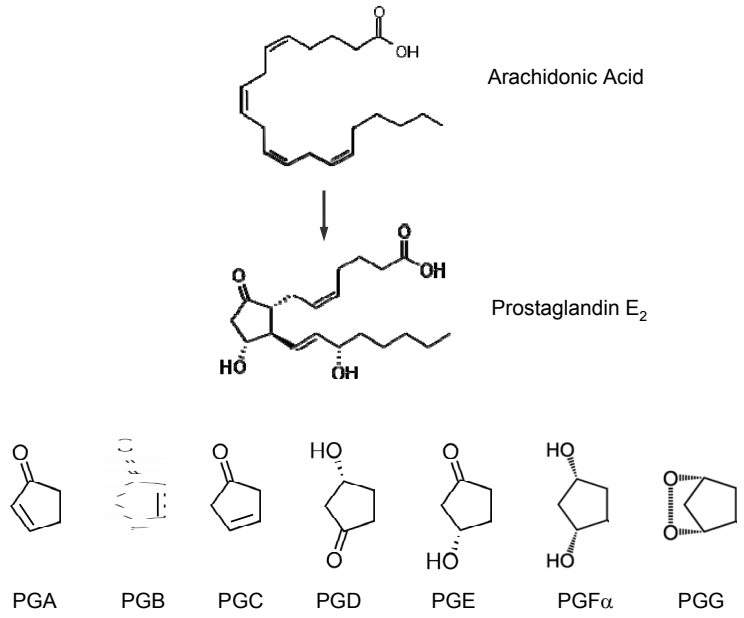
In 2007, Dr. Leonard Zon, one of the scientific founders of Fate, and his colleagues at Harvard Medical School identified 16, 16-dimethyl prostaglandin E2 (referred to as FT1050) as a potent regulator of hematopoiesis (blood cell formation). They demonstrated that *ex vivo* treatment of mouse bone marrow with FT1050 led to an approximately 4x increase in HSC frequency when transplanted into irradiated mice. These findings were later confirmed by others in the field.

Fate has translated the initial academic discovery into clinical settings, optimizing the incubation conditions and performing extensive preclinical characterization studies. The first product candidate generated is ProHema, cord blood HSCs modulated with FT1050.

Prostaglandins are lipid mediators that participate in the regulation of multiple biological processes, including inflammation, fever, pain, vascular tone, platelet function, and fertility. Although initially thought to be a product of the prostate gland, and hence the name, prostaglandins are now known to be produced by virtually all tissues of the body.

Prostaglandin molecules are the cyclooxygenase (COX) metabolite of arachidonic acid (a 20-carbon polyunsaturated fatty acid), which is in turn derived from membrane phospholipids (usually phosphatidylinositol). Prostaglandins can be categorized based on their chemical structures, taking into account the number of double bonds present in the molecule and the fatty acid from which they are derived. A group letter (e.g., A, B, C, D, E, F, G, H) is assigned to distinguish functional substitutions in the cyclopentanone nucleus, and a numeric (e.g., 1, 2, 3) is assigned to note the number of double bonds. The structure of prostaglandin E2, or PGE2, as well as the functional substitutions of common prostaglandin molecules, is shown in Exhibit 8.

Exhibit 8: Structures of Prostaglandins



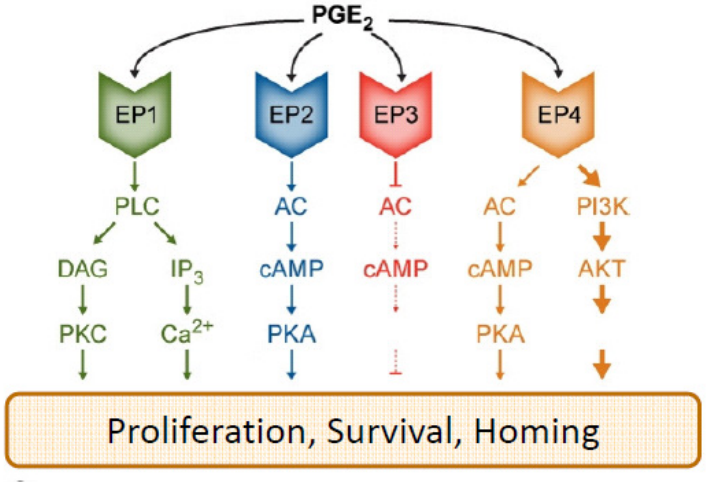
Source: BMO Capital Markets

Prostaglandins are signaling molecules, but they cannot act over long distances (as hormones do) because they are rapidly degraded. As a result, prostaglandins mainly act locally, affecting either the same cell that makes them (autocrine) or nearby cells (paracrine).

Prostaglandin E2, or PGE2, is among the best characterized prostaglandins. PGE2 signals in an autocrine/paracrine manner through four specific G-protein coupled E-prostanoid (EP) receptors. Signaling through EP1 triggers mobilization of intracellular Ca2+, whereas signaling

through EP2 or EP4 stimulates cyclic AMP (cAMP) production. EP3 acts as a negative effector to inhibit cAMP (Exhibit 9).

Exhibit 9: Prostaglandin E2 Signaling Pathways



Source: Fate Therapeutics

The work carried out by Leonard Zon's laboratory and other groups has demonstrated that treatment with FT1050, which is a longer-lasting form of PGE2, increases HSC engraftment. Ex vivo treatment of mouse bone marrow with FT1050 led to an approximately 4x increase in HSC frequency when transplanted into irradiated mice. Similarly, in a model system to evaluate human HSC function *in vivo*, FT1050 treatment enhanced engraftment of human umbilical blood cord HSC in immunodeficient mice (NOD/SCID-IL2Rγ null mice).

The enhancement in the engraftment of FT1050-treated HSCs could be attributed to three effects of FT1050 treatment on HSCs: increased homing efficiency, increased survival, and increased self-renewal.

Treatment with FT1050 resulted in a 2x increase in homing of HSCs to the bone marrow microenvironment. The increase in homing efficiency is a result of increased expression of chemokine receptor CXCR4 on HSCs. CXCR4 and its cognate ligand, SDF-1α, are believed to play a major role in HSC trafficking to bone marrow. In fact, Sanofi's MOZOBIL, which is licensed to increase stem cell mobilization, is an inhibitor of CXCR4.

In addition, treatment with FT1050 also resulted in reduced apoptosis and increased proliferation of HSCs. Treatment with FT1050 blocked caspase 3 activation and protected HSCs from apoptosis in reduced serum conditions (an experimental model to induce apoptosis). Exposure of HSCs to FT1050 also led to increased proliferation and entry into the cell cycle both *in vitro* and *in vivo*.

ProHema's Potential Advantages Over Standard of Care HSCT

Modulation of HSCs with PGE2 has the potential to enhance the biological properties of HSCs from any source, including cord blood, peripheral blood and bone marrow. Potential advantages afforded by PGE2 modulation include enhancement of engraftment and allowing the use of less toxic conditioning regimens across all HSCT, and overcoming limitations in cell dose for cord blood HSCT in particular.

Modulation of HSCs with PGE2 could improve patient outcomes by increasing engraftment success rates, accelerating the time to reconstitution, and improving the durability of engraftment. These potential benefits apply to HSCT using any source of HSCs, including bone marrow, peripheral blood, and cord blood.

Because ProHema enhances the rate of engraftment, it may be possible to improve the feasibility of conducting HSCT under the less toxic RIC regimen as opposed to MAC. This may help broaden the patient populations for allogeneic HSCT.

ProHema may significantly overcome the limitations associated with cord blood HSCT and expand its use. Cord blood has many benefits, such as increased likelihood of identifying an HLA-compatible HSC source and reduced incidence of GvHD and relapse. Given that there are currently more than 600,000 publicly-banked cord blood units available world-wide, it is possible to rapidly identify a well HLA-matched cord blood unit for most patients. By enhancing the biological properties of cord blood HSCs, ProHema could help reduce the number of cells needed in HSCT. This could overcome the limitation of lower HSC dose for chord blood units, and help expand the use of cord blood HSCs. This could be particularly beneficial to the 80% of patients undergoing HSCT who do not have the ideal option of a sibling transplant.

Exhibit 10: Potential of ProHema in Allogeneic Transplant

Transplant Variable	TODAY	Tomorrow
HSC Source	Related matched donor (when possible) Unrelated mPB/BM: Inferior outcomes	ProHema (Cord Blood-Derived Ex Vivo Modulated HSCs)
Degree of HLA Match	Best outcomes require 8/8 HLA-match	4-6/6 matching due to immunonaïve cells in cord blood
Time to Transplant	Months due to donor searches for best match	Rapid access to banked cord blood units with onsite manufacture of ProHema
Conditioning Regimen	Myeloablative	Reduced Intensity
Engraftment	Graft failure and time to engraftment a challenge in some transplants	Low graft failure with rapid time to engraftment
Relative Risk of GvHD	Up to 50%	25-50% reduction with cord blood[1]
Disease Free Survival/Relapse	~50-60% at 2 years with unrelated BM	Up to 50% improved with cord blood[2]

[1] Brunstein, Blood 2010
[2] Milano. ASH 2012

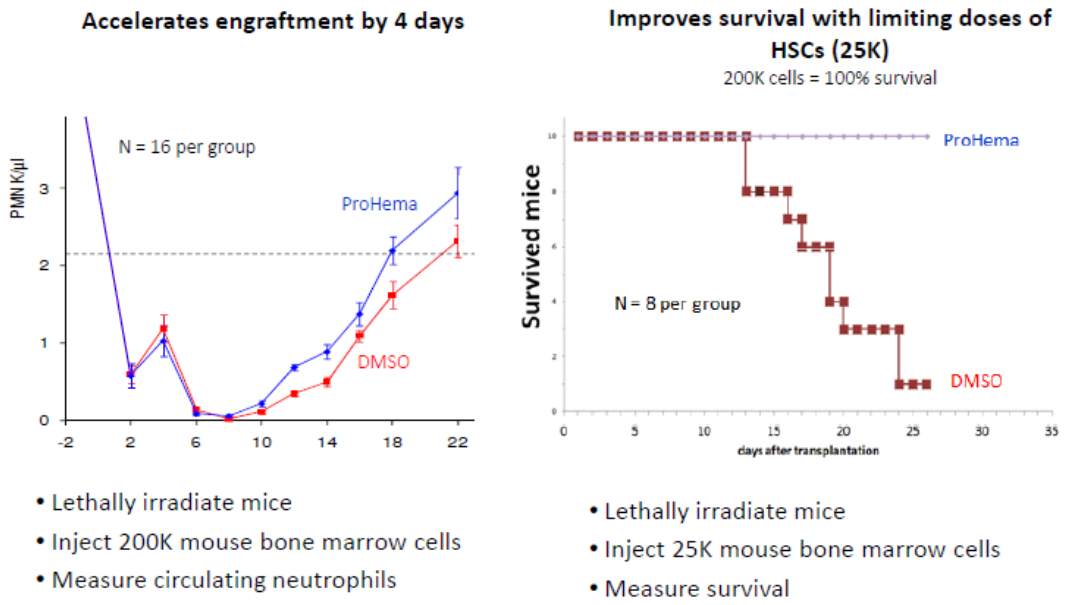
Source: Fate Therapeutics

Review of Preclinical Results

In preclinical studies, treatment of FT1050 resulted in a 2x increase in homing of HSCs to bone marrow and a 4x increase in long-term HSC engraftment. Treatment of FT1050 also increased the survival outcomes in a mouse model of HSCT.

In a mouse model of HSCT, mice were irradiated at a high radiation dose to eliminate all HSCs, and then received FT1050-treated or untreated mouse bone marrow cells. The recipient mice were then monitored for circulating neutrophil counts or for survival. As shown in Exhibit 11, engraftment was accelerated by four days in the group that received 200,000 FT1050-treated bone marrow cells compared with the group that received the same number of untreated cells. To evaluate survival, a reduced number (25,000) of bone marrow cells were transplanted. At 25 days after the transplantation, 90% of the mice that received untreated bone marrow cells had died, whereas all mice that received FT1050-treated bone marrow cells were alive.

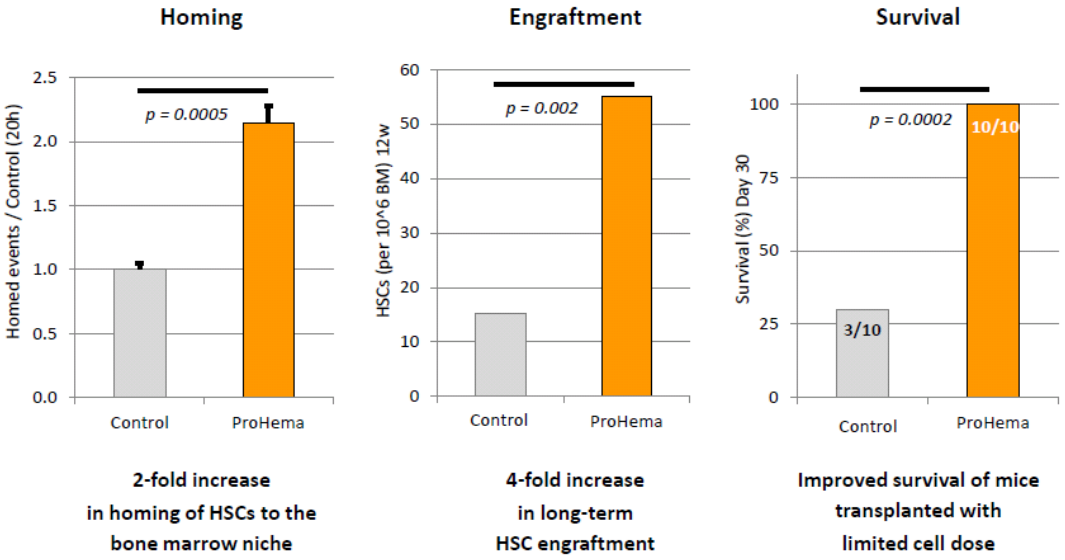
Exhibit 11: HSC Modulation Accelerates Engraftment and Enables Rescue with Fewer HSCs



Source: Fate Therapeutics

Fate also demonstrated that ProHema increased homing of HSCs by two-fold and increased engraftment of HSCs by four-fold. In addition, in experiments where a suboptimal dose of HSCs was transplanted, 70% of mice in the control group died, whereas all of the mice in the ProHema group survived at day 30 (Exhibit 12).

Exhibit 12: Enhanced Properties of HSCs by FT1050 Treatment



Source: Fate Therapeutics

Review of Phase 1b Results in Hematologic Malignancies

In phase 1b experience, ProHema has demonstrated human proof-of-concept that *ex vivo* pharmacologic modulation of HSCs has the potential to improve the key clinical measures of neutrophil engraftment. ProHema demonstrated a median time to neutrophil recovery of 17.5 days (versus 20.5 days historical) in double-umbilical cord blood transplants (UCBT) in patients with hematologic malignancies following reduced-intensity conditioning therapy. ProHema also demonstrated preferential reconstitution compared with unmanipulated cord blood. ProHema was described as well tolerated in the phase 1b experience.

The phase 1b trial, known as ProHema-01, was conducted at the Dana Farber Cancer Institute and the Massachusetts General Hospital and was completed in September 2011. The trial evaluated ProHema in adult patients with hematologic malignancies undergoing double UCBT following a reduced-intensity conditioning (RIC) regimen. Adult patients with acute leukemia, non-Hodgkin's lymphoma or myelodysplastic syndrome received sequential infusion of two cord blood units, a ProHema unit and an unmanipulated cord blood unit. The ProHema unit was infused 2-6 hours before the unmanipulated cord blood unit. The primary objective of the trial was safety and the secondary objectives were time to engraftment and 100-day survival.

In the ProHema-01 trial, a total of 21 patients were enrolled into two cohorts. In Cohort 1 (n=9), patients received ProHema prepared by incubating cord blood cells with FT1050 at 4°C. For Cohort 2 (n=12), the incubation was performed at 37°C. Cohort 1 served as a control group, because the FT1050 treatment was biologically inactive at 4°C. The results of the trial were also compared with a historical control group of 53 adult patients with hematologic malignancies undergoing double UCBT at the two clinical sites of the ProHema-01 trial.

As shown in Exhibit 13, treatment with ProHema demonstrated a statistically significant improvement in time to neutrophil engraftment (peripheral blood neutrophil count ≥500 cells per microliter) as compared with the historical control (p=0.043). The mean time to engraftment was 17.5, 22.0, and 20.5 days for ProHema cohort (Cohort 2), ProHema inactive cohort (Cohort 1), and historical control, respectively. ProHema also improved the cumulative incidence of neutrophil engraftment and the cumulative incidence of platelet engraftment (peripheral blood platelet count ≥20,000 platelets per microliter). In addition, the 100-day survival in the ProHema cohort (100%) compared favorably to both the inactive cohort (89%) and the historical control (87%).

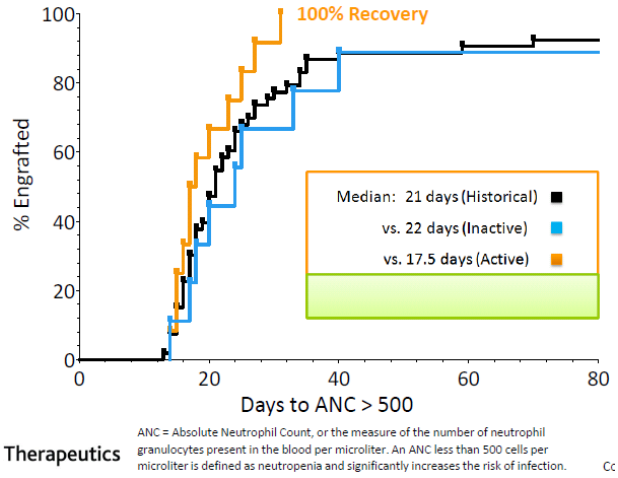
There was also a low incidence of acute GvHD in the ProHema cohort. By day 100, there was an 8% incidence of Grade II-IV acute GvHD in the ProHema cohort as compared with 17% in the historical control group. One patient in the ProHema cohort experienced mild chronic GvHD.

Exhibit 13: ProHema Phase 1b Results

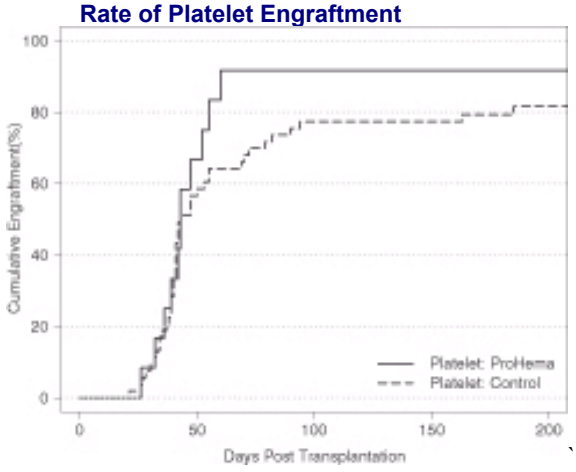
Summary of Key Results

Cohort	Mean Time to Engraftment	Cumulative Incidence of Neutrophil Engraftment by Day 26	Rate of Failure to Achieve Neutrophil Engraftment	100-Day Survival
ProHema	17.5 days (range 14 - 31 days)	83%	0%	100%
Inactive	22.0 days (range 14 - 40 days)	67%	11%	89%
Historical	20.5 days (range 13 - 70 days)	70%	6%	87%

Rate of Neutrophil Engraftment



Rate of Platelet Engraftment



Source: Fate Therapeutics

Double-umbilical cord blood transplantation is known to have a competitive engraftment dynamic, whereby one of the two cord blood units becomes dominant over a short period of time and is responsible for long-term hematopoiesis. The competitive dynamic provided an opportunity to evaluate whether ProHema could out-compete unmanipulated HSCs in Cohort 2.

In this cohort, ProHema-derived HSCs dominated the hematopoietic reconstitution in 83% of patients (10 of 12) at day 100. In contrast, at day 100, the profile of hematopoietic reconstitution in the historical control was substantially diverse: 34% of patients engrafted with the first cord administered; 34% of patients engrafted with the second cord administered; and 8% of patients demonstrated dual chimerism, where both cords contributed to hematopoietic reconstitution (the remainder either experienced graft failure or died prior to day 100).

With a median follow-up among survivors of 24.6 months, no secondary graft failure (defined as a graft failure following an initial period of engraftment) was observed in the ProHema cohort (Cohort 2).

ProHema was described as well tolerated in the phase 1b experience. The trial met all safety criteria and adverse events attributed to ProHema consisted of mild to moderate infusion-related events including rash, nausea, chills, flushing, abdominal pain, and cough, all of which are considered common transplant-related side effects. One patient with known coronary artery disease experienced transient myocardial ischemia that resolved promptly after completion of the infusion.

In summary, the ProHema-01 trial demonstrated proof-of-concept that *ex vivo* pharmacologic modulation of HSCs has the potential to improve the key clinical measures of neutrophil engraftment, including time to engraftment and durability of engraftment.

Potential Positive Impact of Media Change

In the ProHema-01 trial, ProHema was manufactured using a standard processing media, which is commonly used in the clinical setting for thawing and washing umbilical cord blood units. In 2Q13, Fate demonstrated that a new nutrient-rich media formulation (referred to as NRM) improved the potency and efficacy of ProHema.

The use of the NRM formulation resulted in a 9- to 126-fold increase in the expression of FT1050-induced genes, compared with a 2- to 6-fold increase with standard processing media. The new manufacturing conditions also improved cell viability, as measured by HSC recovery. The homing potential of HSCs, as measured by an *in vitro* transwell migration assay, was also improved from 7% (standard media) to 34% (NRM). NRM also improved the performance of HSCs in an *in vivo* homing assay involving a human xenograft model (Exhibit 14).

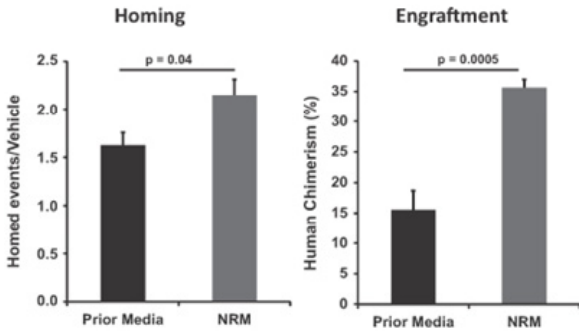
Fate intends to incorporate the improved NRM formulation into its future clinical development program for ProHema, including the ongoing phase 2 trial. Based on the data presented, this new media formulation has the potential to improve ProHema's potency and efficacy profile in the clinical setting.

Exhibit 14: Optimization of ProHema Using Nutrient-rich Media (NRM)

Summary of Key Measures

Biologic Measure of Activity	Prior Media	NRM
Expression of relevant genes	2-6 fold	9-126 fold
Homing potential	7%	34%
Viable HSC Recovery	88%	107%
Increase in HSC population	62%	131%

In Vivo Homing Assay



Source: Fate Therapeutics

Review of Ongoing Phase 2 Development in Adult Hematologic Malignancies

Fate initiated a phase 2 trial of ProHema in December 2012, but recently paused the enrollment of the trial to enable the manufacture of ProHema using the NRM formulation. The trial is currently active at eight major allogeneic HSCT centers but is not recruiting. Eleven patients had been treated in the phase 2 trial prior to the pause of enrollment. Seven of 8 patients in the ProHema arm engrafted, with a median time to engraftment of 28 days, as compared with 3 of 3 patients who engrafted in the control arm, with a median time of 31 days.

This phase 2 trial, referred to as ProHema-03, is a randomized, controlled, multi-center trial of ProHema in adult patients undergoing double UCBT for hematologic malignancies using both MAC and RIC regimens. Before Fate elected to pause enrollment of the trial, eight patients had been randomized to receive ProHema plus an unmanipulated cord blood unit, and three patients had been randomized into the control arm to receive two unmanipulated cord blood units. All 11 patients were conditioned using a MAC regiment and no patients conditioned using a RIC regimen were enrolled.

The three subjects in the control arm engrafted at days 30, 31 and 40, yielding a control median time to engraftment of 31 days. Five of the 8 subjects in the ProHema arm engrafted prior to the control median, at days 14, 19, 24, 28 and 30. Two of the 8 subjects in the ProHema arm engrafted post the control median at days 40 and 48, and 1 of the 8 subjects in the ProHema arm failed to engraft. No patients experienced secondary graft failure.

As of September 30, 2013, of the eight subjects in the ProHema arm, four subjects survived to day 100, two subjects died before day 100, and two subjects engrafted and remain alive and have not yet reached day 100. The three subjects in the control arm survived to day 100. With a median follow-up period of 156 days, five of eight subjects in the ProHema arm remain alive and have engrafted, as compared to one of three subjects in the control arm.

One subject in the ProHema arm experienced Grade IV acute GvHD, and one subject in the control arm experienced Grade III acute GvHD. Adverse events attributed to ProHema were primarily limited to common infusion-related side effects. Fate believes these safety results are consistent with expected outcomes in adult patients undergoing HSCT using umbilical cord blood after a MAC regimen without ProHema.

As mentioned earlier, Fate paused the enrollment of the phase 2 trial to enable the manufacture of ProHema incorporating the NRM formulation and to generate data to qualify the optimized manufacturing process that incorporates the NRM formulation. On August 1, 2013, Fate submitted an IND amendment to FDA. The IND contained preclinical and product development data supporting the incorporation of NRM formulation for the manufacture of ProHema. The data also suggested that the use of the NRM formulation should not result in additional safety risks. In addition, Fate submitted an amended protocol for the ProHema-03 trial that defined how it will resume enrollment with ProHema as manufactured using the NRM formulation.

Specifically, Fate stated in its IND amendment that it would enroll 60 subjects under the revised ProHema manufacturing process and that the previously enrolled subjects would be followed and analyzed separately. Fate expects to resume enrollment of the ProHema-03 trial in 1H14, subject to the consent of FDA, and expects full data from this trial in mid-2015.

Exhibit 15: Comparison of ProHema Phase 1b and Phase 2 Trials

Design considerations	Prior Formulation		New Formulation
ProHema preclinical potency	+++		+++++
Study	Phase 1b	Phase 2	Phase 2
Randomization	Non-randomized (historical control)	Randomized (concurrent control)	Randomized (concurrent control)
Conditioning regimen	RIC (partial myeloablation)	MAC (full myeloablation)	RIC & MAC (stratified randomization)
# of sites	2 sites	8 sites	10 sites
# of pts on ProHema (vs. control)	12 pts (53 control)	8 pts (3 control)	40 pts (vs. 20)
Patient data			
% engraft before control median	67% of pts	63% of pts	*80% power (one-sided) to demonstrate that 70% of ProHema pts engraft prior to control median*
Δ median time to engraftment	3 Days (17.5 vs. 20.5)	3 Days (28 vs. 31)	
Alive & engrafted, Day 100 F/U	12/12 pts (vs. 76%)	6/8 pts (vs. 3/3)	
Alive & engrafted, Day 156 median F/U		5/8 pts (vs. 1/3)	

Source: Fate Therapeutics

Upon resuming enrollment, a total of 60 adult patients across both MAC and RIC regimens will be enrolled and randomized, at a 2:1 ratio, to ProHema plus an unmanipulated cord blood unit or two unmanipulated cord blood units. Prior to randomization, patients will be stratified based on conditioning regimen (RIC vs. MAC). The primary endpoint of the trial is the cumulative incidence of neutrophil engraftment by a pre-specified control median engraftment time, which will be adjusted based upon the median times calculated for subjects enrolled to the control arm. The study is designed to demonstrate that 70% of the subjects in the ProHema arm achieve neutrophil engraftment before the control median engraftment time. Secondary endpoints include time to neutrophil engraftment, cumulative incidence of neutrophil engraftment by day 42, time to platelet engraftment, cumulative incidence of platelet engraftment by day 180, as well as rates of graft failure and of GvHD and event-free and overall survival.

If the ProHema-03 trial is successful, Fate plans to seek additional regulatory guidance with the goal of initiating a phase 3 pivotal trial of ProHema, which may include both adult and pediatric patients, undergoing UCBT for hematologic malignancies.

In the end-of-phase 1 meeting, FDA provided guidance on potential phase 3 clinical trial endpoints and other trial design issues. The guidance suggested that time to engraftment of neutrophils, platelets, or both may be a sufficient primary endpoint. FDA also encouraged the use of survival as a secondary endpoint, and discouraged the use of duration of hospitalization or transfusion as primary endpoints due to higher risk of bias. FDA strongly encouraged the use of concurrent controls (as opposed to historical controls). FDA's feedback also suggested that a single phase 3 trial, enrolling both adult and pediatric subjects, may be sufficient for approval in both age groups, depending on the results.

Based on the regulatory guidance and preliminary statistical power calculations, Fate believes that the phase 3 program could consist of a single trial enrolling approximately 200 patients, with time to engraftment of neutrophils, platelets, or both as an endpoint to support approval.

Review of Ongoing Phase 1b Development in Pediatric Hematologic Malignancies

Fate intends to explore the potential of ProHema in pediatric patients in the setting a single UCBT. As a first step, a phase 1 trial has been initiated in the setting of single UCBT in adult patients with hematologic malignancies. Of the six adult subjects that are evaluable to date, four subjects engrafted at days 17, 19, 22, and 37, and two subjects had primary graft failure; the 100-day survival rate was 100%. Fate plans to initiate a phase 1b pediatric trial in the single UCBT setting in 2014, subject to FDA consent.

For pediatric patients, the standard of care in UCBT for the treatment of hematologic malignancies only requires a single cord blood unit. While the cell dose received by pediatric patients from a single cord blood unit can be sufficient, data suggest that these patients still suffer from delayed engraftment, high rates of graft failure and high rates of transplant-related morbidity and mortality.

To explore ProHema in a pediatric patient population, Fate first initiated a phase 1 clinical trial to determine safety in the setting of single UCBT in adults with hematologic malignancies. In this phase 1 trial, referred to as ProHema-02, qualifying patients receive a RIC regimen followed by a single ProHema cord blood unit. The primary endpoint is safety. Engraftment measures and rates of GvHD, relapse and survival are also measured.

The phase 1 trial has enrolled eight subjects, of which six subjects are evaluable (median age 55.9 years; four subjects with acute myelogenous leukemia, one with myelodysplastic syndrome and one with multiple myeloma). Four of the six evaluable subjects engrafted at days 17, 19, 22 and 37, and two experienced primary graft failure. Survival at 100 days was 100%. No acute or chronic GvHD has been observed and no patients experienced secondary graft failure. Adverse events attributed to ProHema were limited to common transplant-related side effects.

Based on these results, Fate engaged in a preliminary review of the ProHema-02 data with FDA with the intent to conduct a phase 1b trial in children and adolescents with hematologic malignancies. FDA indicated that it was open to such a pediatric trial, but requested a written summary of the ProHema-02 trial as well as a synopsis of proposed phase 1b pediatric trial with justifications for the trial design.

Subject to Fate's submission of the requested information and FDA approval, Fate plans to initiate the phase 1b clinical trial in children and adolescents with hematologic malignancies, in which patients would receive a single ProHema unit. The primary endpoint of the trial is expected to be safety as defined by neutrophil engraftment. Secondary endpoints will include additional measures of engraftment, including time to neutrophil engraftment, cumulative incidence of neutrophil engraftment by day 42, time to platelet engraftment, cumulative incidence of platelet engraftment by day 180, as well as rate of graft failure, rate of GvHD, event-free survival, and overall survival.

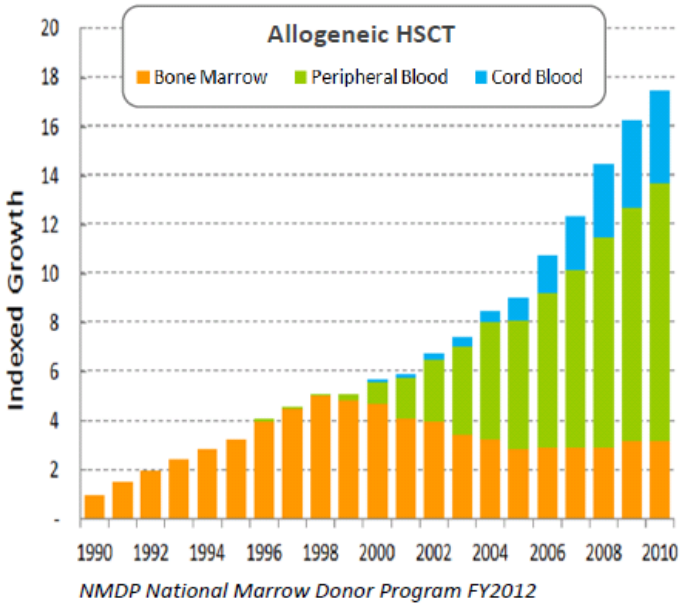
Fate expects commencing enrollment for the planned phase 1b trial in pediatric patients during 2014 and conducting the trial at one to three clinical centers in the US. The NRM formulation will be used to manufacture ProHema in this trial. Fate believes that it can conduct this trial under its current IND for ProHema (with necessary amendment); however, Fate still needs to submit clinical development plans to the FDA for this trial, and the FDA may require a new IND.

Market Opportunity in ProHema in Hematologic Malignancies

Over the past two decades, the number of HSCT procedures has increased steadily and the growth is expected to continue. According to a global survey conducted by the Worldwide Network for Blood and Marrow Transplantation, a total of 56,739 HSCT procedures were performed worldwide in 2010, including 26,241 such procedures in the allogeneic setting. In the US, more than 5,800 allogenic HSCT procedures were performed in 2012 according to the National Marrow Donor Program, and 20% of the procedures used cord blood as the cell

source. It is estimated that approximately 95% of HSCT procedures are performed for the treatment of hematologic malignancies.

Exhibit 16: Growth of Allogeneic HSCT and Shift in Cell Sources



Source: Fate Therapeutics

Rationale for ProHema in Orphan Metabolic Diseases

There has been a steady increase in the use of HSCT for the treatment of rare genetic disorders. The rationale for HSCT treatment is that donor-derived cells can correct the underlying genetic defect by direct repopulation of the hematopoietic and immune systems or by indirect delivery of the missing enzymes or other cellular components.

For example, in a group of 50+ genetic disorders collectively known as lysosomal storage diseases (LSDs), certain enzyme in the lysosomes (cellular organelles responsible for degradation of waste products) is absent or does not work properly because of a genetic defect, and as a result, certain metabolites cannot be degraded, and accumulate in various tissues and organs (referred to as *storage*). Examples of these metabolites include sphingolipids, glycogen, mucopolysaccharides, and glycoproteins. Through HSCT, healthy HSCs of the donor colonize the bone marrow of the patient, and differentiate into various blood cells. In addition to residing in the bone marrow and the blood, donor-derived cells can also reach other tissues. This is because circulating monocytes (a type of white blood cells) can migrate into various tissues and become tissue resident macrophages. In the blood and other tissues, donor HSC-derived cells produce and secrete the enzyme that is missing in the patient. The secreted enzyme is then internalized by surrounding host cells and is delivered into the lysosomes (through the mannose-6-phosphate receptor mediated pathway), where it degrades the stored, undigested metabolites.

HSCT has several advantages over the traditional therapy for LSDs, known as enzyme replacement therapy (ERT). In ERT, the missing enzyme is synthesized using recombinant technologies in therapeutic amounts and administered to patients through periodic infusions or injections. Because intravenously administered enzymes do not cross the blood-brain barrier, ERT is not effective in certain LSDs that primarily involve the nervous system. HSCT, on the other hand, is not limited in this aspect, because donor-derived monocytes can migrate into the brain and differentiate into microglia cells (tissue-resident macrophages), which then produce the missing enzyme in the brain.

Two examples of LSDs with primary neuronal involvement are Krabbe's disease and Hurler's syndrome. Krabbe's disease is caused by deficiency of the lysosomal enzyme galactocerebrosidase. Krabbe's disease is characterized by a breakdown of myelin in the central and peripheral nervous systems, rapidly progressive neurologic deterioration, and death. In a small study involving 11 asymptomatic newborns (12 to 44 days old) and 14 symptomatic infants (142 to 352 days old) with infantile Krabbe's disease, treatment with cord blood HSCT favorably altered the natural history of the disease. Survival rate was 100% for the asymptomatic newborns (median follow-up 3.0 years) and 43% for the symptomatic infants (median follow-up 3.4 years). Clinical benefit was more pronounced in newborns who underwent transplantation before the onset of symptoms. These newborns had progressive central myelination and continued gains in development skills, and most had age-appropriate cognitive function and receptive language skills.

Hurler's syndrome is caused by deficiency of α-L-iduronidase and results in accumulation of heparan and dermatan sulfate substrates in various tissues. Hurler's syndrome is characterized by progressive deterioration of the central nervous system and death in childhood. Allogeneic bone marrow transplant before the age of two years halts disease progression and prolongs life, but many children lack appropriately matched bone marrow donor. Cord blood HSCT favorably altered the natural history of the disease in a study involving 20 children with Hurler's syndrome. Seventeen of the 20 children were alive a median of 905 days after transplantation, and transplantation improved neurocognitive performance. The investigators of the study concluded that cord blood from unrelated donors was an excellent source of HSCs for transplantation in patients with Hurler's syndrome.

Besides the ability to address the neurologic manifestations of LSDs, HSCT also has the advantages of being a one-time therapy and being able to treat multiple disorders with the same treatment approach. In contrast, ERT is a continuous, life-long therapy and different therapeutic agents have to be developed for each disease.

The use of HSCT is in genetic disorders is not limited to LSDs. To date, more than 50 genetic disorders have been treated with allogeneic HSCT. Many of these diseases are life-threatening and do not have alternative therapeutic options. Examples include peroxisomal storage disorders, such as adrenoleukodystrophy; hemoglobinopathies, such as sickle cell disease and certain thalassemias; inherited bone marrow failure syndromes, such as Fanconi anemia and Diamond-Blackfan anemia; and inherited immune deficiencies, such as Wiskott-Aldrich syndrome.

It has been recognized that, compared with bone marrow or peripheral blood HSCT, cord blood HSCT has several advantages in treating LSDs: (1) Compared with the hematologic malignancy setting, even more patients lack a suitable related donor or matched unrelated donor; (2) Cord blood can be rapidly accessed, a critical factor for patient outcomes in certain LSDs that have rapid and irreversible progression, such as infantile Hurler syndrome or Krabbe disease; and (3) There is growing evidence that the proportion of patients achieving normal enzyme levels is higher following allogeneic HSCT with cord blood than with other HSC sources.

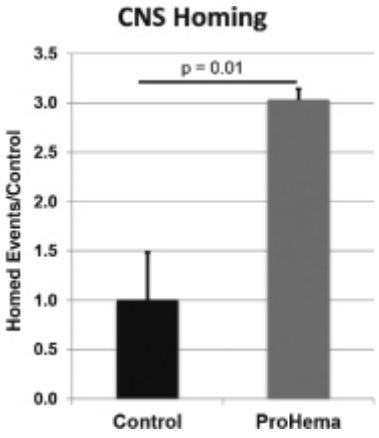
ProHema, or similar product candidates from Fate's hematopoietic HSC modulation platform, has considerable potential in allogeneic HSCT in rare genetic disorders, given preclinical data demonstrating enhanced homing and engraftment, as well as clinical proof-of-concept in the hematologic malignancy setting. By improving engraftment, ProHema may also be able broaden the use of less toxic RIC regimens in patients with rare genetic diseases, who are often infants or young children. So far, HSCT in rare genetic disorders are routinely performed using MAC regimens, because attempts to utilize RIC regiments have resulted in unacceptably high graft failure rates. The use of the highly toxic MAC regimens in infants and young children is of significant concern and ProHema's enhanced engraftment potential could enable the broader adoption of RIC regimens.

Preclinical Data and Development Plans for ProHema in Orphan Metabolic Diseases

Fate is planning to develop an *ex vivo* pharmacologically-modulated HSC therapeutic in pediatric patients with demyelinating LSDs. Fate plans to explore this potential through an initial clinical trial of ProHema, as well as through a focused research program to identify other product candidates.

In a preclinical study, Fate demonstrated that *ex vivo* modulated cord blood increased the number of donor cells that home and migrate across the blood-brain barrier into the CNS. Human cord blood-derived HSCs were treated with FT1050, or vehicle control for two hours at 37°C and injected into sub-lethally irradiated NSG mice. The number of human cells in the brain tissue of mice was determined 20 hours later (as measured by genomic DNA). As shown in Exhibit 17, the CNS homing property of HSCs derived from human cord blood was significantly improved by *ex vivo* modulation with FT1050.

Exhibit 17: Enhanced CNS Homing by ProHema



Source: Fate Therapeutics

Fate plans to initiate a first clinical trial of ProHema in pediatric patients with demyelinating LSDs in 2014 after filing an amendment to the existing IND (subject to FDA consent), with the goal of generating data from this trial in 2015. The primary objective of the trial will be to evaluate the potential of *ex vivo* enhanced HSCs to enable robust engraftment under RIC regimens, a setting in which unmodulated cord blood units did not perform well according to previous studies. This trial is expected to enroll patients between the ages of 1 to 21 years.

The trial will be conducted at up to three centers that specialize in pediatric cord blood transplantation for rare genetic disorders. After conditioning, patients will receive a ProHema unit in combination with an unmodulated unit. The first cohort of subjects will receive a conditioning regimen using a combination of high-dose chemotherapy agents that comprise a standard myeloablative regimen (MAC) used for such transplants but in which one agent has been dose-reduced by 25%. Subsequent cohorts will receive conditioning regimens that are successively dose-reduced. The primary endpoint of the study will be neutrophil engraftment. Subjects will also be followed for other measures of engraftment and safety, as well as cognitive and functional evaluations to measure the impact of treatment on developmental milestones.

Fate also plans to develop second generation therapeutics specifically designed to enhance the homing of HSCs to the CNS to improve delivery of essential enzymes that are deficient in patients with LSDs.

Pipeline Focus on Wnt7a Protein Analogs for the Treatment of Muscular Dystrophies

Fate's SSC modulation platform focuses on the *in vivo* (within the body) pharmacological activation of satellite stem cells (SSCs) for the treatment of muscle degeneration conditions, such as muscular dystrophies. Fate has identified Wnt7a as a driver of muscle regeneration and has developed injectable analogs of Wnt7a as recombinant human protein therapeutics with muscle regenerative activity.

Wnt drives muscle regeneration through two independent mechanisms: expansion of the SSC compartment and promotion of hypertrophy (increase in size) in differentiated muscle cells.

In preclinical studies, injection of a Wnt7a analog led to a three-fold expansion of SSC population, a 20% increase in muscle hypertrophy, and an 18% increase in muscle strength in both wild-type mice and mdx mice. (Mdx mice harbor a premature termination codon in the dystrophin gene and are the most commonly used mouse model for muscular dystrophy.) A significant reduction in disease-related muscle damage was also observed in the mdx mice. Wnt7a analogs demonstrated similar potency as naturally-occurring Wnt7a. In experiments involving *in vitro* cultures of myotubes derived from healthy volunteers and patients with Duchenne, Becker and facioscapulohumeral dystrophies, Wnt7a analogs also induced muscle cell hypertrophy.

Fate's initial clinical focus for the Wnt7a analog program is to assess safety and demonstrate human proof-of-concept in X chromosome-linked dystrophy patients. Fate is currently selecting proprietary Wnt7a analog with preferred efficacy, manufacturing and formulation characteristics, and will conduct an IND-enabling study in a canine model of muscular dystrophy in 2014. Fate plans to initiate a phase 1 clinical trial in healthy volunteers, with the goal of data readout in 2015. Fate plans to assess biomarkers and measures of muscle strength by electromyography in the phase 1 trial. Based on the results of the phase 1 trial, Fate plans to initiate a dose-escalation study in an X chromosome-linked muscular dystrophy patient population, such as DMD.

Overview of Muscular Dystrophies

Muscular dystrophies are a group of rare genetic diseases characterized by progressive degeneration and weakness of skeletal muscle. There are many distinct types of muscular dystrophies and the underlying genetic defects are diverse, involving in over 30 distinct genes that encode structural proteins as well as enzymes that modify these structural proteins.

The most prevalent and well-characterized forms are the X chromosome-linked muscular dystrophies, including Duchenne muscular dystrophy (DMD) and Becker muscular dystrophy (BMD). These diseases occur primarily in males (X-linked recessive inheritance).

DMD, which accounts for approximately half of all muscular dystrophy cases, is caused by genetic defects that lead to an absence of a protein called dystrophin (see Primer on Muscle

Structure and Dystrophin below). Symptoms of DMD usually appear in children between ages two and three years, with weakness starting near the trunk and spreading to the extremities. A child may have difficulty running, jumping, and may use their hands to push upright from squatting or sitting positions (called Gower's sign). Boys with DMD continue to develop in strength and muscle function, albeit at a lower rate than healthy boys, until they are approximately seven years old. Afterwards, afflicted children begin to lose muscle function and generally become wheel-chair bound by age 12 or 13. DMD may also cause heart problems, including irregular heartbeat and enlargement of heart tissue. Lung function may also be affected because of abnormal curvature of the spine (scoliosis) combined with muscle weakness. A minority of patients also show mental impairment.

BMD is a less severe variant of DMD, in which mutations in the dystrophin gene lead to the expression of truncated, but partially functional, dystrophin protein. The age of onset for BMD is usually later compared with that of DMD, and the symptoms are usually milder. Children can usually walk until age 16, and some patients continue to walk as adults.

Other types of muscular dystrophies include facioscapulohumeral muscular dystrophy (FSHD), limb-girdle dystrophies and myotonic dystrophy. FSHD is thought to be caused by a defect in the expression of the double Homeobox protein 4 (DUX4) gene and is characterized by muscle weakness in the face, shoulders, and upper arms. The DUX gene is normally expressed in germ line tissue and is epigenetically repressed in somatic cells.

FSHD affects both boys and girls and is usually inherited in an autosomal dominant pattern. FSHD usually progresses slowly, but the severity and age of onset can be extremely variable. Symptoms usually start between 20 and 30 years of age, with a slow progression and near normal life span. The facial muscles are involved initially, with inability to close the eyes tightly or to smile, although the facial weakness can be mild and remain mild for many years. The muscles of the shoulders and upper arms are also usually involved. In the more severe infant form of FSHD, symptoms appear within the first few years of life and progresses rapidly to involve the shoulders and hips. Most children with the early onset FSHD require a wheelchair by the age of 9 or 10 years.

Limb-girdle muscular dystrophy is a group of disorders that affect the areas surrounding the shoulders and/or hips. The age of onset varies from early childhood to adulthood, and the progression of disease is typically slow.

Myotonic dystrophy is the most common adult-onset muscular dystrophy, and symptoms typically appear in adolescence or adulthood. Myotonic dystrophy can affect both males and females. Muscles in multiple locations can be affected, particularly in the face, the arms, and the legs. In addition, myotonic dystrophy may also cause problems in heart and glands and affect normal intellectual functioning.

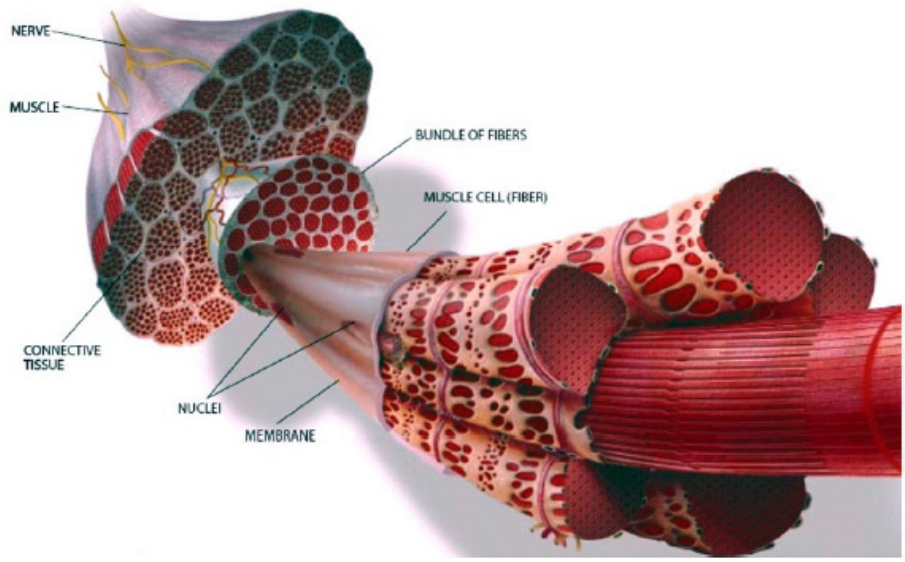
Primer on Muscle Structure and Dystrophin

The functional unit of skeletal muscle is the muscle fiber, which are long, cylindrical cells, each with numerous nuclei. Muscle fibers (also called myofibers) result developmentally from the

fusion of many mononucleated precursor cells called myoblasts. Myoblasts are generated by asymmetric division and differentiation of satellite cells.

In addition to typical cellular organelles, the cytoplasm (sarcoplasm) of a muscle fiber contains an array of myofibrils, which consist of longitudinally arranged actin-containing thin filaments, myosin-containing thick filaments, along with structural and regulatory proteins. Sarcomeres are contractile units of a myofibril arranged as repeating units along the length of the myofibril.

Exhibit 18: Muscle Structure



Source: Fate Therapeutics

Dystrophin is a large, rod-shaped protein that is important for maintaining the stability of muscle fibers. It is encoded by the *DMD* gene, the largest gene in the human genome, located on the X chromosome. Dystrophin is part of the dystrophin-glycoprotein complex (DGC), a multisubunit complex that connects the cytoskeleton of a muscle fiber to its surrounding extracellular matrix. A significant number of muscular dystrophies result from mutations that affect the normal assembly of the DGC at the sarcolemma in muscle.

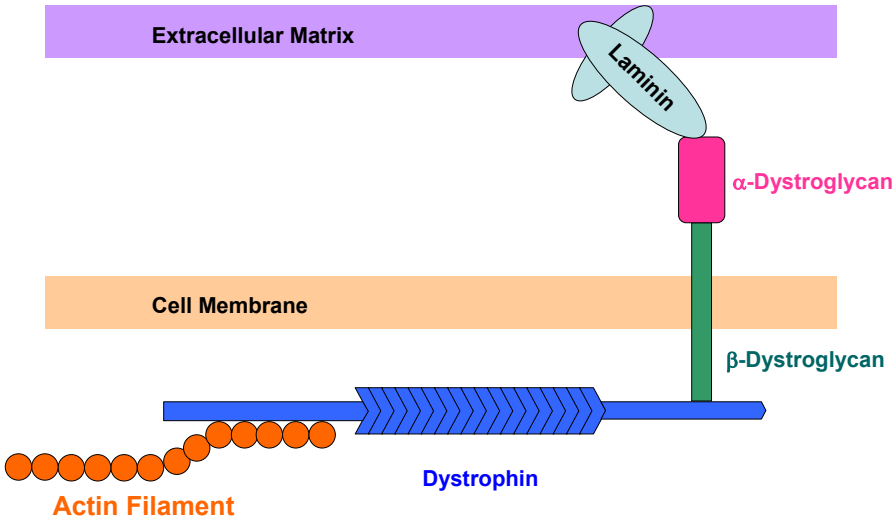
Dystrophin forms a tight link with the actin cytoskeleton at its N-terminus and another tight link with β-dystroglycan at its C-terminus. The transmembrane protein β-dystroglycan interacts strongly with the extracellular α-dystroglycan, which in turn interacts with laminin α2, a major constituent of muscle fiber basement membrane. Therefore, the backbone formed by dystrophin, dystroglycans and laminin α2 connects the actin cytoskeleton with the extracellular matrix. In doing so, the DGC provides structural support to the plasma membrane, protecting it from the mechanical stress of contractile activity by transmitting the lateral tension generated by muscle contraction to the extracellular matrix.

With a defective DGC, the backbone structure falls apart, exposing the cell membrane to muscle contraction stresses. As a consequence, focal ruptures may occur at the cell membrane,

leading to transient intracellular calcium influx, which triggers a series of pathogenic events that result in muscle degeneration and the dystrophic phenotype typical of DMD and BMD.

DMD is caused by mutations in the dystrophin gene that results in a severe reduction or complete absence of the dystrophin protein. The most common mutations underlying DMD are deletions (72%), followed by point mutations (20%) and duplications (7%). Most deletions occur between exons 44 and 55. (There are 79 exons in the dystrophin gene). If a deletion mutation alters the reading frame of dystrophin (out-of-frame mutation), no functional dystrophin can be made and the patient develops DMD. BMD, on the other hand, is caused by in-frame deletions that result in the expression of truncated, but partially functional forms of dystrophin.

Exhibit 19: Structure of the Dystrophin-Glycoprotein Complex



Source: BMO Capital Markets

Primer on Satellite Cells and Satellite Stem Cells

Adult skeletal muscle is a stable tissue under normal physiological conditions but has remarkable ability to regenerate after injury. Reestablishment of full power occurs in as soon as three weeks even after severe damage that has caused widespread muscle cell death.

The cells responsible for generating myoblasts in postnatal skeletal muscle are the satellite cells, which are located on a niche on the surface of muscle fibers. In development, satellite cells provide myoblasts for muscle growth. In adults, satellite cells are mitotically quiescent but can be recruited to supply myoblasts for routine maintenance (homeostasis) of muscle fibers or for sporadic hypertrophy or repair.

The satellite cell population is maintained by a subpopulation of stem cells, termed satellite stem cells (SSC), which accounts for 10% of the adult satellite cell pool. Both SSCs and committed satellite cells express lineage-specific transcription factor paired-box 7 (Pax7), but

SSC can be distinguished by their lack of expression of a myogenic regulatory protein, Myf5. Thus, SSCs are $Pax7^{+}$ $Myf5^{-}$, whereas committed satellite cells are $Pax7^{+}$ $Myf5^{+}$.

Satellite cells, along with SSCs, reside between the basal lamina and the sarcolemma of muscle fibers. The basal lamina is a layer of extracellular matrix closely apposed to the sarcolemma. In response to natural molecular triggers from exercise, injury or disease, committed satellite cells become activated, upregulate myoblast determination protein, MyoD ($Pax7^{+}$ $Myf5^{+}$ $MyoD^{+}$), and reenter cell cycle to proliferate as myoblasts (myogenic precursor cells). Myoblasts undergo multiple rounds of division before committing to terminal differentiation, fusing with the host fibers or generating new muscle fibers to reconstruct damaged tissue.

In muscular dystrophies, muscle degeneration leads to continuous compensatory satellite cell activation and differentiation to effect regeneration. This constant cycle of muscle damage and repair eventually results in exhaustion of the regenerative capacity, leading to accelerated tissue degeneration and significant loss of muscle function.

Restoring the balance between muscle degeneration and regeneration to induce tissue repair is a promising approach for the treatment of muscular dystrophies, irrespective of the causative genetic mutation.

Rationale for Wnt7a-mediated SSC Modulation in the Treatment of Muscular Dystrophies

Fate is developing recombinant analogs of Wnt7a as a pan-therapeutic agent for the treatment of muscular dystrophies irrespective of genetic background. Wnt7a is a natural signaling molecule involved in satellite stem cell (SSC) biology. Fate and one of its scientific founders have demonstrated Wnt7a's role in driving the expansion of SSCs and promoting hypertrophy (increase in size) of differentiated muscle fibers. In an animal model of muscular dystrophy, Wnt7a treatment led to a significant increase in muscle strength and a reduction in contractile damage.

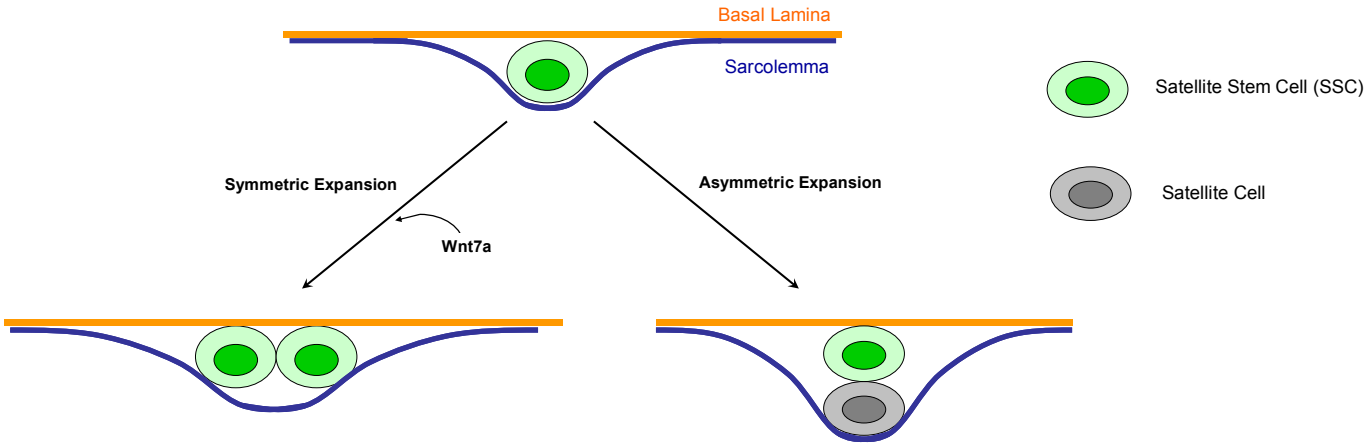
Although Wnt7a-based therapeutics do not directly address the underlying genetic defects of muscular dystrophy, the improvement of muscle strength and reduction of muscle damage could provide significant therapeutic benefits to patients. For example, the most common cause of death in DMD is respiratory failure due to loss of muscle strength in the muscles essential for respiration, leading to complications such as pneumonia. Furthermore, Wnt7a-based therapeutics could potentially be combined with other therapies that directly correct the underlying genetic defects.

One of Fate's scientific founders, Michael Rudnicki Ph.D., has made numerous seminal discoveries in SSC research, including identification of Pax7 as a transcription factor required for the specification of satellite cells. Dr. Runicki's ground-breaking discovery of the role of Wnt7a in stimulating muscle cell growth serves as the basis for Fate's SSC modulation program.

SSCs undergo self-renewal to allow tissue homeostasis and regeneration throughout the lifespan of an individual. Similar to other adult stem cells, SSCs follow the traditional symmetric and

asymmetric paradigms of self-renewal. In symmetric division, the two daughter cells are identical; in asymmetric division, one daughter remains a stem cell whereas the other daughter differentiates. For SSCs, the fate of daughter cells is determined by the orientation of the mitotic axis in relation to the muscle fiber. Divisions parallel to the muscle fiber (planar) result in symmetric expansion of satellite stem cells. Divisions perpendicular to the muscle fiber follow the asymmetric paradigm and give rise to two distinct fates in the daughter cells: one daughter cell becomes a committed satellite cell ($Myf5^{+}$), and the other daughter cell remains an SSC (Exhibit 20). Therefore symmetric expansion, but not asymmetric expansion, leads to an increased number of SSCs.

Exhibit 20: Symmetric and Asymmetric Satellite Stem Cell Expansion



Source: BMO Capital Markets; adapted from Wang and Rudnicki, *Nat Rev Mol Cell Biol* 2012, 13:127

Work from Dr. Rudnicki's laboratory has demonstrated that Wnt7a, a member of the Wnt protein family, drives symmetric expansion of SSCs and enhances muscle regeneration. The Wnt family of proteins plays an important role in cell fate determination during embryogenesis and in adult stem cells. The family includes 19 cysteine-rich, secreted Wnt proteins that bind to Frizzled (Fzd) receptors. Binding of Wnt to Fzd can trigger various downstream signaling cascades, including the canonical pathway that leads to the activation of β-catenin, and the non-canonical pathways, which include the planar cell-polarity (PCP) pathway and the Ca2+ pathway.

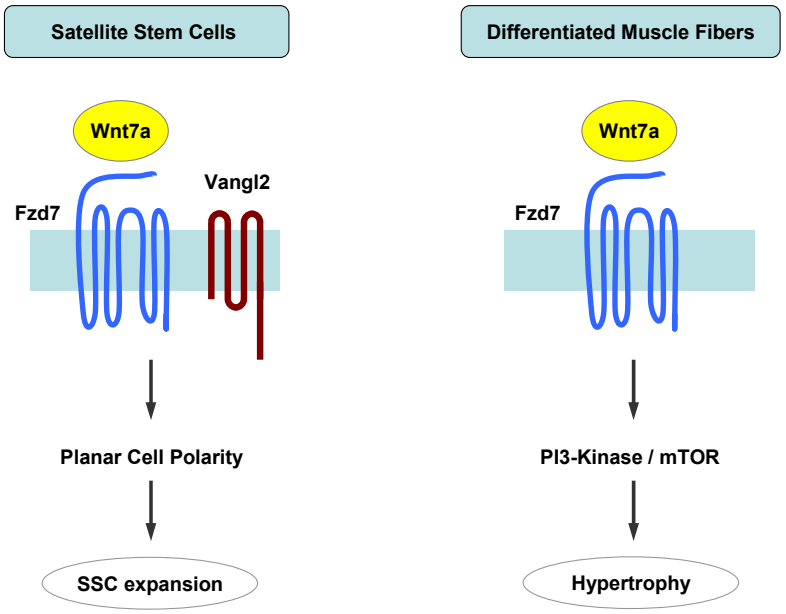
Dr. Rudnicki's laboratory has demonstrated that Wnt7a is expressed during muscle regeneration and that it drives symmetric expansion of SSCs through activating the PCP pathway by interacting with Fzd7 and Vangl2. In animal studies, Wnt7a overexpression increased the number of satellite cells and the proportion of SSCs, while deficiency in Wnt7a resulted in a marked decrease in the number of satellite cells following regeneration. Therefore, Wnt7a signaling through the PCP pathway controls the homeostatic level of SSCs and as a result regulates the regenerative potential of muscle.

Another line of studies from Dr. Rudnick's laboratory has demonstrated that Wnt7a signaling also affects differentiated muscle fibers. Binding of Wnt7a to Fzd7 in differentiated muscle

fibers activates the Akt/mTOR growth pathway and leads to an increase in the size of muscle fibers, a phenomenon referred to as hypertrophy. Hypertrophy results from the production of more contractile proteins.

Dr. Rudnick's laboratory also examined the effect of Wnt7a in mdx mice. Mdx mice harbor a premature termination codon in the dystrophin gene and are the most commonly used mouse model of muscular dystrophy. Wnt7a treatment resulted in a significant increase in muscle strength, which was independent of the increase in muscle mass (the hypertrophy effect of Wnt7a). Furthermore, treatment with Wnt7a reduced the level of contractile damage, likely by changing the composition of fiber types. Treatment with Wnt7a led to a shift in fiber types toward an increased amount of slow-twitch fibers. Slow-twitch fibers are known to be less susceptible to contractile damage compared to fast-twitch fibers.

Exhibit 21: Dual Function of Wnt7a in Adult Skeletal Muscle



Source: Fate Therapeutics and BMO Capital Markets

Fate's SSC Modulation Platform with Wnt7 Analogs

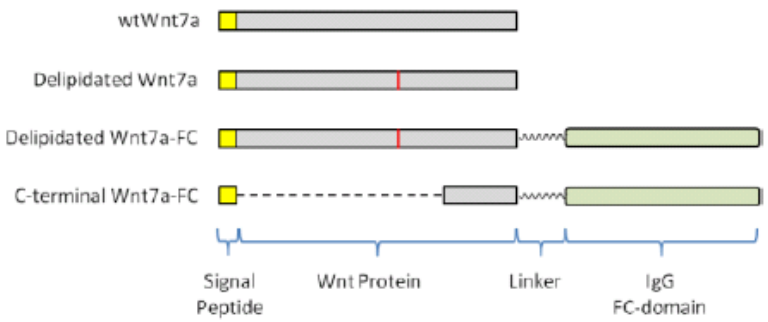
Fate is developing agents targeting the Wnt7a non-canonical signaling pathways using its SSC modulation platform, with an initial focus on muscular dystrophy. To our knowledge, Fate's SSC modulation platform is the first to demonstrate in preclinical studies that SSCs can be pharmacologically targeted to improve muscle regeneration.

Although the regenerative potential of Wnt proteins is well known, their use as therapeutics has historically been hampered by the molecular characteristics of the Wnt family. In particular, Wnt proteins undergo posttranslational lipid modifications. Lipid modifications have been

demonstrated to be critical for Wnt signaling through the canonical pathway. However, lipid modifications present challenges for scaled production, formulation, and administration of Wnt for clinical use.

Fate has developed pharmacologically optimized analogs of Wnt proteins. These rationally designed Wnt analogs have incorporated structure features such as removal of lipid modification (delipidation), N-terminus truncation, and fusion to the Fc domain of IgG (Exhibit 22). As a result, Fate's Wnt analogs have improved pharmaceutical development properties, including scaled production, therapeutic formulation, and effective delivery. In addition, as delipidation abolishes the ability of Wnt to signal through the canonical pathway, Wnt analogs have increased selectivity for non-canonical pathways. To our knowledge, Fate is the first company to produce Wnt analogs that are amenable to therapeutic development and *in vivo* administration.

Exhibit 22: Rationally Designed Wnt7a Analogs



Source: Fate Therapeutics

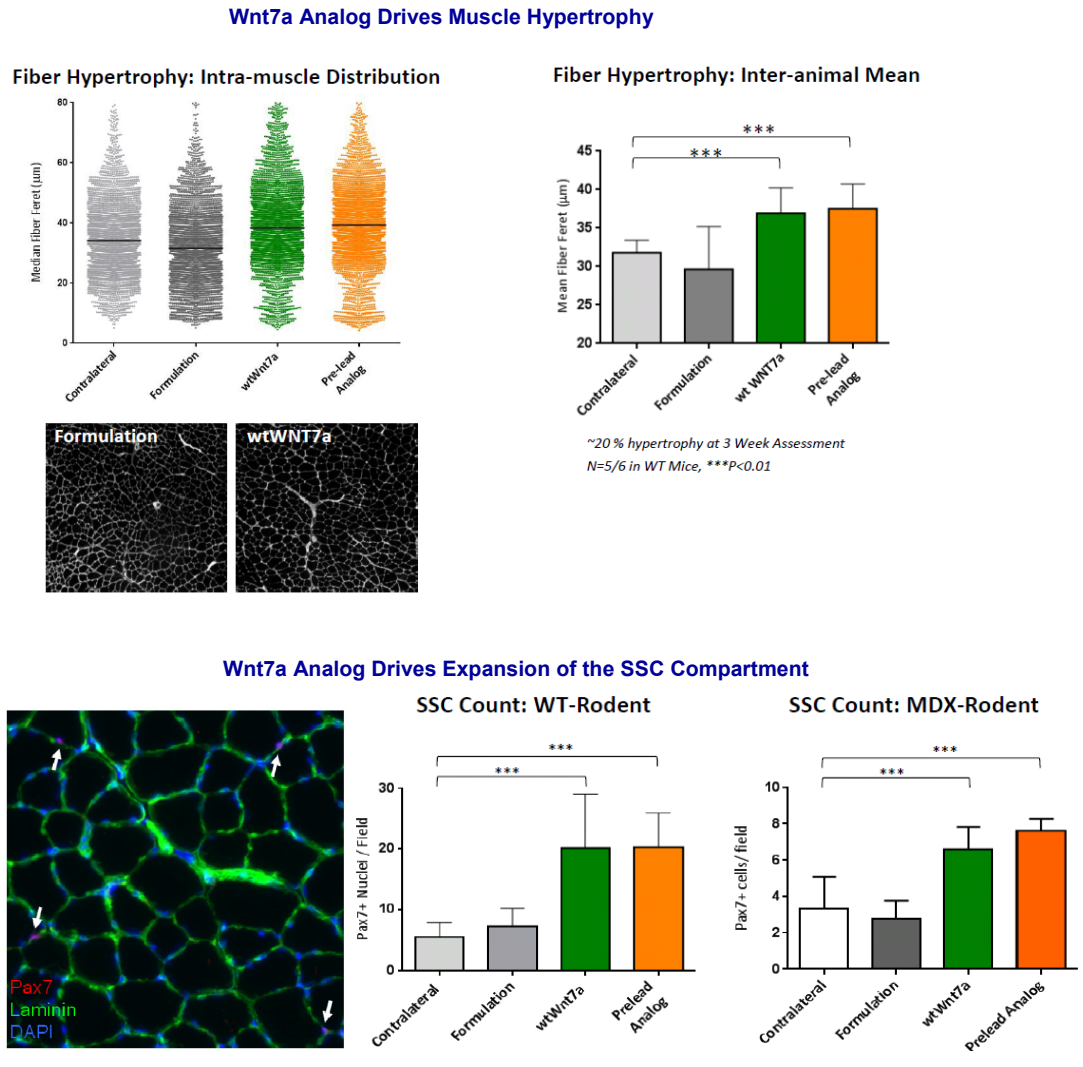
Most approaches to treat muscular dystrophies seek to slow muscle degeneration in distinct subtypes of the disease. Wnt7a analogs, on the other hand, promote muscular regeneration and therefore could be used to treat a broader spectrum of muscular dystrophies irrespective of the underlying defective genes. Another attractive feature of Fate's Wnt7a analogs is that they could serve as either a stand-alone therapy or an add-on therapy to disease-specific muscular dystrophy therapeutics.

Fate believes its SSC modulation platform has potential beyond muscular dystrophies and could be effective in restoring the balance between muscle degeneration and regeneration for other neuromuscular disorders, such as cachexia, atrophy, trauma and sarcopenia. Fate also intends to assess Wnt-based biologic modulators other than Wnt7a for use in broader regenerative medicine applications.

Review of Preclinical Data for Wnt7a Analogs

Fate has demonstrated the therapeutic potential of Wnt7a analogs in various preclinical models. Injection of a Wnt7a analog led to a 3-fold expansion of SSC population, a 20% increase in muscle hypertrophy, and an 18% increase in muscle strength in both wild-type mice and mdx mice, a rodent model of muscular dystrophy. A significant reduction in disease-related muscle damage was also observed. Wnt7a analogs demonstrated similar potency as naturally-occurring Wnt7a. In experiments involving *in vitro* cultures of myotubes derived from healthy volunteers and patients with Duchenne, Becker and facioscapulohumeral dystrophies, Wnt7a analogs also induced muscle cell hypertrophy.

Exhibit 23: Muscle Hypertrophy and SSC Expansion Following a Single Wnt7a Analog Injection

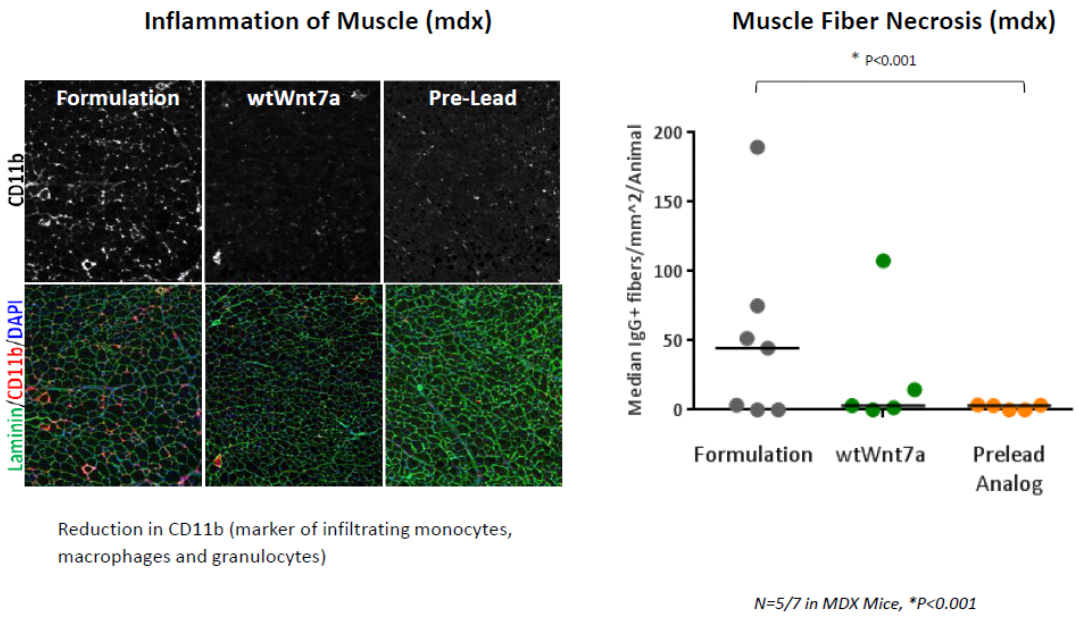


Source: Fate Therapeutics

In both wild-type and mdx mice, a single injection of low microgram amounts of Wnt7a or a Wnt7a analog to the *tibialis anterior* muscle induced muscle hypertrophy and a significant expansion of the SSC population in a dose dependent manner at three weeks following the injection. Compared with equivalent untreated muscle on the opposite side of the body in the wild-type mice (contralateral control), Wnt7a and the Wnt7a analog demonstrated a statistically significant hypertrophic effect, represented by an approximately 20% increase in the median muscle fiber minimum cross-sectional diameter (Exhibit 23, top row). Fate also observed a statistically significant increase in the number of muscle SSCs, represented by an approximately three-fold increase in the number of Pax7 positive cell nuclei, a marker for SSCs, in the treated muscle relative to the contralateral control (Exhibit 23, bottom row).

In human muscular dystrophies as well as the mdx mouse model, muscle fiber necrosis, and inflammation contribute to tissue fibrosis and a reduction in muscle strength and regenerative capacity. Inducing muscle regeneration in the mdx mouse resulted in increased muscle fiber integrity and reduced inflammatory cell infiltration of the tissue. Following a single injection of Wnt7a or a Wnt7a analog to mdx mice, Fate observed a reduction in positive staining of CD11b, a biomarker of inflammation and a statistically significant reduction in disease-specific muscle fiber necrosis, measured by IgG-positive fibers per unit area of muscle, as compared to formulation control (Exhibit 24).

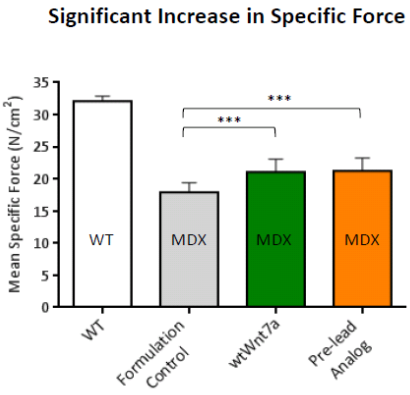
Exhibit 24: Wnt7a Analog Reduced Inflammation and Muscle Damage in MDX Mouse Model



Source: Fate Therapeutics

The mdx mice are significantly weaker than wild-type mice, as measured by specific force, or normalized force per cross-sectional area of muscle. Fate demonstrated that a single administration of Wnt7a or a Wnt7a analog to mdx mice induced a significant increase of approximately 18% in the specific force generated by the *tibialis anterior* muscle (Exhibit 25).

Exhibit 25: Wnt7a Analog Improved Muscular Strength in MDX Mouse Model



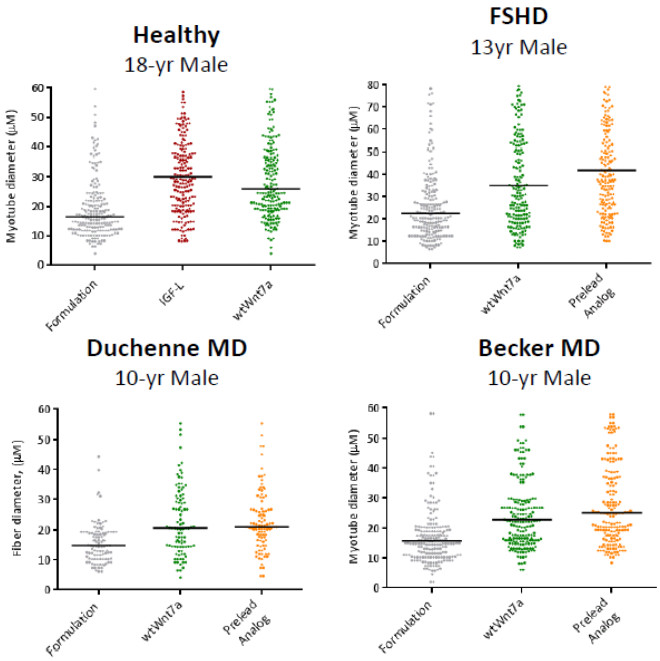
In Situ Force Measurements				
	WT	Formulation Control MDX	wtWNT7a MDX	Pre-Lead Analog MDX
Number of Animals	5	12	12	12
CSA (mm²)	4.99±0.50	7.67±0.68	7.02±0.52	7.26±0.70
Tetanic Force	1599±147	1372±172	1476±169	1536±167
Specific Force (N/cm²)	32.09±0.79	17.88±1.52	21.03±2.04	21.23±1.99
P value	<0.0001	N/A	0.0002	0.0001
Force Increase (% Control)	79.5	0	17.6%	18.7%

*N=12 MDX Mice, ***P<0.0005*

Source: Fate Therapeutics

In experiments involving *in vitro* cultures of myotubes derived from healthy volunteers and patients with Duchenne, Becker and facioscapulohumeral dystrophies, Wnt7a analogs also induced muscle cell hypertrophy (Exhibit 26). (The IGF treatment in the top left panel represents a positive control; the IGF-IGFR-PI3K-Akt-mTOR signaling axis is a key regulator of skeletal muscle hypertrophy). These studies support the potential for Wnt7a analogs to drive muscle hypertrophy across different types of muscular dystrophies irrespective of the underlying genetic cause.

Exhibit 26: Wnt7a Analog Induced Hypertrophy of Human Muscle Cells in Vitro



Source: Fate Therapeutics

Development Plans for Wnt7a Analogs in Muscular Dystrophies

Fate is currently expanding preclinical assessments of Wnt7a analogs to include dose and regimen optimization in rodent models. Fate also plans to initiate efficacy and pharmacokinetic assessments in a well characterized canine model of muscular dystrophy, which allows assessment of larger muscle groups and a more predictable transition of dose and administration regimen to human trials. Preliminary non-GLP toxicology assessments with dose escalation are also underway and will inform future IND-enabling toxicology studies.

Fate has identified potential Wnt7a-specific pharmacodynamic biomarkers to accelerate the clinical development process. These include both cellular markers, such as muscle hypertrophy and SSC population expansion, and molecular markers based on whole genome expression analysis of Wnt7a-treated muscle. The measurement of these biomarkers can be attained through a pre- and post-treatment punch biopsy.

Fate's initial clinical focus for the Wnt7a analog program is to gain safety information and demonstrate human proof-of-concept in X chromosome-linked dystrophy patients with local administration of therapeutic protein to targeted muscle groups.

Upon the completion of IND-enabling studies, Fate plans to file an IND in 2014 to initiate a phase 1 clinical trial in healthy volunteers, with the goal of generating phase 1 data in 2015. Fate plans to assess biomarkers and measures of muscle strength by electromyography in the phase 1 trial.

Based on the results of the phase 1 trial, Fate plans to initiate a dose-escalation study in an X chromosome-linked muscular dystrophy patient population, such as DMD. Fate believes the results of the phase 1 trial in healthy volunteers and the dose escalation trial in DMD patients, as well as the establishment of effective pharmacodynamic biomarkers, will provide a solid ground for further discussions with FDA regarding the path to approval in muscular dystrophies.

Competitive Landscape for Muscular Dystrophy Therapies

Besides Fate's unique strategy of stimulating muscle regeneration for the treatment of muscular dystrophy, other promising therapeutic strategies have also emerged. These include oligonucleotide exon-skipping of specific mutations (e.g., Prosensa's drisapersen and Sarepta's eteplirsen), stop-codon override strategies (e.g., PTC Therapeutics' ataluren), and utrophin up-regulation (Summit's SMT C1100).

These therapeutic strategies differ significantly with Fate's strategy because they primarily focus on preventing muscle degeneration, and, with the exception of utrophin up-regulation, aim at a subset of the muscular dystrophy patient population, such as DMD patients with specific types of mutations. In contrast, Fate's strategy seeks to increase the regenerative potential of muscle and could potentially address many types of muscular dystrophies regardless of underlying genetic defect.

Because of the differences in mechanisms of action, it is possible that Fate's Wnt7a analogs could be used in combination with the other therapeutics, such as exon-skipping, to provide additive or even synergistic therapeutic effects.

Prosensa's Drisapersen

Prosensa and partner GSK are developing drisapersen (formerly GSK2402968/PRO051), an antisense oligonucleotide that binds specifically to exon 51 of the dystrophin gene's pre-mRNA. The mechanism through which drisapersen corrects the genetic mutations underlying DMD is known as "exon skipping."

The dystrophin gene consists of 79 exons. Approximately 72% of the mutations in DMD are deletion mutations that occur between exons 44 and 55. If a deletion alters the reading frame of dystrophin (out-of-frame mutation), no functional dystrophin can be made and the patient develops DMD.

The specific binding of drisapersen to exon 51 causes exon 51 to be skipped when dystrophin mRNA is created from the pre-mRNA. The skipping of exon 51 may correct the transcription reading frame in approximately 13% of all DMD patients, leading to the production of shortened but functional dystrophin proteins which could improve or maintain muscle function in these patients.

Drisapersen has been studied in several clinical studies by Prosensa and partner GSK and was granted Breakthrough Therapy designation by FDA. Although phase 2 results were promising, with expression of new dystrophin observed in the majority of patients and positive readouts from 6-minute walk tests (6MWT) in certain cohorts, a recently completed 186-patient phase 3 study failed to meet its primary endpoint of improvement in 6MWT versus placebo.

In a phase 1/2 study (PRO051-02) first initiated in March 2008, 12 patients received weekly subcutaneous injections of drisapersen in four dosing cohorts (0.5, 2.0, 4.0 and 6.0 mg/kg) for five weeks. The primary endpoint was safety, and secondary endpoints included PK and molecular and clinical effects. Changes in RNA splicing and dystrophin protein levels were assessed in muscle biopsies at baseline (for 0.5mg/kg cohort), two weeks (all cohorts), and five weeks (all cohorts except the 0.5mg/kg cohort). New dystrophin expression was observed between approximately 60% and 100% of muscle fibers in 10 of the 12 patients, in a dose-dependent manner to up to 15.6% of the expression in healthy muscle.

After an interval of 6 to 15 months following the last dose, each patient restarted treatment with a dose of 6.0 mg/kg/week (open-label extension). Twelve weeks after the start of the extension study, there was a modest improvement of 35.2m on the 6-minute walk test (6MWT). The extension phase is still ongoing. At the latest assessment time-point of 141 weeks, 8 of 10 subjects were able to perform the walking evaluation, and Prosensa suggested the results appeared to demonstrate delay of disease progression in these patients. (The other two patients were not able to complete 6MWT at the start of the extension study).

Prosensa's partner GSK initiated a placebo-controlled phase 2 study (DMD114117) of drisapersen at a 6 mg/kg subcutaneous dose in September 2010. The study enrolled 53 DMD subjects (aged 5 and above with a time to rise from floor of less than 7 seconds) into three arms:

a continuous dosing regimen of 6 mg/kg/week (n=18); an intermittent dosing regimen of 9 doses of 6 mg/kg spread over 6 weeks, followed by 4 weeks off drug (n=17); and a placebo arm (n=18). The primary endpoint was efficacy at 24 weeks. Although this study was not statistically powered to evaluate efficacy, GSK reported that drisapersen demonstrated a statistically significant difference in 6MWD between the continuous dosing arm and the placebo arm at 24 weeks, with a mean difference in 6MWD of 35.09 meters (p=0.014), which was considered a clinically meaningful benefit. At 48 weeks, the mean difference in 6MWD was 35.84 meters (p=0.051). Intermittent dosing did not demonstrate a clinical benefit at 24 weeks, but resulted in a mean difference in 6MWD of 27.08 meters (p=0.147) at 48 weeks. There was little change in muscle strength at either time point for either treatment arm. Preliminary data suggested that treatment with drisapersen was in general associated with an increase in dystrophin expression over pre-treatment levels.

Another ongoing phase 2 trial conducted by GSK (DMD114876) is comparing two doses of drisapersen (3mg/kg/week vs. 6mg/kg/week) in 51 patients over 48 weeks. The primary endpoint is 6MWD at 24 weeks. Enrollment was completed in November 2012 and results are expected in 1Q14.

GSK recently completed a randomized, double-blind, placebo-controlled phase 3 trial (DMD114044). The study assessed once-weekly subcutaneous drisapersen at 6 mg/kg in boys over 5 years of age and with a minimum 6MWD of 75 meters at enrollment. A total of 186 boys were randomized to either drisapersen (n=125) or placebo (n=61) injection for over 48 weeks. The goal of the study was to demonstrate a mean improvement of 30 meters in 6MWD at 48 weeks compared with placebo. The phase 3 trial failed to meet its primary endpoint, with a mean difference in 6MWD of 10.33m (confidence interval: -14.65, 35.31; p=0.415) between the two arms. There was also no treatment difference in key secondary assessments of motor function.

Three additional exon skipping product candidates are in Prosensa's pipeline, targeting exons 44, 45, and 53 of the dystrophin gene. These product candidates are currently in phase 1/2 development.

Sarepta's Eteplirsen

Sarepta's lead drug candidate for DMD, eteplirsen, is also an exon-skipping agent targeting exon 51 of the dystrophin gene. It is currently in a phase 2b study, and results from this study have demonstrated exon-skipping in all patients evaluated, an average increase in dystrophin-positive fibers to 47% of normal level, and significant treatment benefit of 70.8 meters in 6MWT in evaluable patients. Eteplirsen is based on Sarepta's phosphorodiamidate morpholino oligomer (PMO)-based chemistry. This chemistry allows eteplirsen to achieve doses that are 5x to 8x greater than those used for drisapersen in its clinical studies.

Sarepta has completed three clinical trials of eteplirsen in DMD, with promising results in new dystrophin protein expression and statistically significant differences in 6MWT.

In a randomized, double-blind, placebo-controlled phase 2b study (Study 201), 12 boys between the ages of 7 and 10 years with deletion mutations correctable by skipping exon 51 were

randomized to weekly intravenous injections of eteplirsen 30 mg/kg (n=4), eteplirsen 50 mg/kg (n=4), or placebo (n=4) for 24 weeks. After 24 weeks, all placebo-treated patients received weekly eteplirsen treatment at 30 mg/kg (n=2) or 50 mg/kg (n=2). After Week 28, all patients entered an open-label extension study (Study 202) designed to assess the long-term safety and efficacy of eteplirsen. The primary efficacy endpoint in Study 201 and Study 202 was the change from baseline in the percent of dystrophin-positive fibers present in muscle biopsies. A key secondary endpoint in the studies, and the principal clinical outcome measure, was the 6MWT.

The primary endpoints of Studies 201 and 202 were met. Exon skipping was observed in all patients at all time-points post eteplirsen treatment. At 48 weeks, eteplirsen administered at either dose resulted in a statistically significant increase ($p \leq 0.001$) in dystrophin-positive fibers to 47% of normal level. The placebo/delayed-treatment cohort, which had received 24 weeks of eteplirsen following 24 weeks of placebo also showed a statistically significant increase in dystrophin-positive fibers to 38% of normal ($p \leq 0.009$). In patients who were evaluable for 6MWT, eteplirsen-treated patients achieved a 67.3 meter benefit compared to placebo/delayed patients ($p < 0.001$). (Two identical twin boys in the 30mg/kg cohort lost ambulation due to rapid disease progression at or beyond week 24 and were excluded from this analysis).

At 96 weeks, a statistically significant treatment benefit of 70.8 meters ($p \leq 0.001$) was observed in Study 202 in eteplirsen-treated patients evaluable on the 6MWT (n=6) compared with the placebo/delayed-treatment cohort (n=4). Patients in the 30 mg/kg and 50 mg/kg cohorts who were able to perform the 6MWT experienced less than a 5% decline (17.5 meters) from baseline, and the placebo/delayed-treatment cohort, after experiencing a substantial decline earlier in the study, also demonstrated stabilization in walking ability from week 36 through 96. These results were in contrast to what could be expected based on natural history studies of DMD.

According to Sarepta, ongoing discussion with the FDA suggests that FDA is open to an NDA filing based on data from the phase 2b extension study alone, without a confirmatory study. Sarepta plans to submit an NDA in 1H14. At the same time, Sarepta is also planning for a confirmatory study, and expects to finalize the study design upon additional discussion with FDA. Sarepta's proposed design of the confirmatory study is an open-label, 48-week study with a treatment arm containing 60 patients who have deletion mutations correctable by skipping exon 51, and a control arm with 60 patients who have deletion mutations amenable to exon skipping through exons 44, 45, 50, and 53. Patients in the control arm will not receive eteplirsen treatment. The primary endpoint is 6MWT, with dystrophin positive muscle fibers being a key secondary efficacy endpoint. First patient dosing of the confirmatory study is expected in 1Q14.

In addition to eteplirsen, Sarepta has three additional exon-skipping drug candidates currently in preclinical testing and these drug candidates target the exons 45, 50 and 53 of the dystrophin gene.

PTC Therapeutics' Ataluren

Ataluren (PTC124) is an orally administered drug that is being developed for the treatment of DMD as well as cystic fibrosis. It targets a specific type of genetic mutation called nonsense mutation, in which a mutation in a single nucleotide introduces a premature stop codon in the middle of a gene, resulting in the production of a truncated protein. It is estimated that 10% to 15% of DMD patients have this type of mutation.

The proposed mechanism of action for PTC124 is that it causes the ribosome to skip over the premature stop, thus achieving the translation of the full length protein (a process referred to as readthrough). However, some researchers have raised questions on whether ataluren has any true readthrough activity at all. These researchers have argued that the identification of ataluren as a readthrough agent might have been an artifact derived from an off-target effect of ataluren on the luciferase reporter. The luciferase reporter was part of the high-throughput screen used in ataluren's discovery.

A pivotal phase 2b trial of ataluren in DMD was completed in 2009. In a randomized, double-blind, placebo-controlled phase 2b trial, 174 patients (≥5 years of age; ≥75 meters in 6MWT at baseline) with nonsense mutation DMD were randomized to either a low dose of ataluren (10mg/kg in the morning, 10mg/kg at midday, and 20mg/kg in the evening), a high dose of ataluren (20mg/kg in the morning, 20mg/kg at midday, and 40mg/kg in the evening), or placebo for 48 weeks. At 48 weeks, the difference in average change in 6MWD between the low-dose ataluren group and the placebo group was 29.7 meters (p=0.149). Numerically, the difference of 29.7 meters can be considered clinically meaningful, because marketed drugs for other genetic disorders that affect muscle activity were approved on the basis of a difference in 6MWT of approximately 30 meters. However, the p value of 0.149 was not statistically significant. In addition, there was no difference between the high-dose group and the placebo group.

PTC Therapeutics conducted a post hoc analysis of the phase 2b clinical data to address what it believed to be issues with the pre-specified statistical analysis. In the post hoc analysis, the difference between the low dose group and the placebo group in 6MWT was 31.3 meters, with a nominal p value of 0.0281, or an adjusted p value of 0.0561.

In 1Q13, PTC Therapeutics initiated a phase 3 study of ataluren. This randomized, double-blind, placebo-controlled study evaluates ataluren at a dose identical to the low dose used in the phase 2b study (10mg/kg in the morning, 10mg/kg at midday, and 20mg/kg in the evening). Primary endpoint is the change in 6MWT. The study plans to enroll 220 patients with DMD or BMD caused by nonsense mutation. Results are expected in mid-2015.

Summit's SMT C1100

SMT C1100 is a drug candidate developed by the UK biotech company, Summit. SMT C1100 is designed to upregulate and maintain the production of utrophin. Utrophin has similar function to dystrophin. Utrophin is highly expressed in fetal and regenerating muscle fibers, but its expression decreases as the muscle fibers mature and is eventually replaced by dystrophin.

SMT C1100 has the potential to be broadly effective in all DMD patients, regardless of the underlying genetic defects.

SMT C1100 is currently in phase 1 development. Top-line data of a healthy volunteer phase 1 study was announced in 4Q12. The trial examined a new nano particle aqueous suspension of SMT C1100, and Summit concluded that the new formulation was safe and well-tolerated at all doses tested. No data was reported regarding utrophin expression in the study subjects.

Market Opportunity in Duchenne/Becker Muscular Dystrophies

In the US, it is estimated that Duchenne/Becker Muscular Dystrophies (DBMD) occur in one out of 3,500 live births, resulting in a prevalence of approximately 10,000 males. Approximately 80% of patients suffering from DBMD were wheelchair-bound by 14 years of age, according to a 2007 study.

In early studies, most patients with DMD died in their late teens or twenties, with respiratory infections or cardiomyopathy as the major cause of death. Newer studies have reported a median survival to age 35 years, and the improvement in survival is thought to be a result of advances in respiratory and cardiac care. Patients with BMD, which is a milder variant of DMD, usually survive into their mid 40s, with the most common cause of death being heart failure from cardiomyopathy.

There are no drugs specifically approved for the treatment of muscular dystrophies. DMD in boys over the age of five is primarily treated with steroids, such as prednisone or deflazacort. Steroids have been demonstrated to temporarily preserve muscle function and delay disease progression, such as time to wheelchair dependency, by approximately two years. Steroid treatment has serious side effects, negatively impacting growth, bone mineral density and immune functions. Treatment options other than steroids have not been extensively studied or proven to be safe and effective. For BMD, prednisone may be used as a treatment, although little is known about its effect on the symptoms and disease progression.

Intellectual Properties

Fate's intellectual property portfolio currently consists of 46 issued patents and 174 patent applications licensed from academic and research institutions and 40 patent applications that it owns.

Regarding the HSC modulation platform, Fate owns six families of pending US and foreign patent applications, including 14 pending applications relating to ProHema and other therapeutic compositions of stem cells that have been pharmacologically modulated to enhance their therapeutic properties, and methods of manufacturing the cellular compositions. Any US patents issued from these applications will expire between 2030 and 2034.

Fate has an exclusive license to a portfolio consisting of two families of issued patents and pending applications granted by the Children's Medical Center Corporation and The General Hospital Corporation. Patents within this portfolio that have issued or may yet issue will expire

in 2027. In this portfolio, Fate has exclusive rights to 9 issued patents and 27 pending patent applications relating to methods for promoting tissue growth or regeneration using modulators that up-regulate the prostaglandin signaling pathway, including US Patent 8,168,428, which claims a method for promoting HSC engraftment through the *ex vivo* modulation of cord blood, bone marrow, or peripheral blood HSCs using FT1050.

Fate has also licensed exclusive rights to two families of patent applications from the Indiana University Research and Technology Corporation claiming methods of enhancing HSCT procedures by altering prostaglandin activity in HSCs and progenitor cells and methods for enhancing gene transduction efficacy in stem cell gene therapy. These applications are currently pending in the US and in certain foreign jurisdictions; if issued, the US patents could have terms expiring in 2029 or 2030.

Regarding the SSC modulation platform and Wnt analogs, Fate owns pending patent applications covering compositions of matter, including Wnt polypeptide analogs having production and formulation advantages, as well as formulations containing such Wnt analogs suitable for local and systemic administration, and methods of preparing such Wnt proteins and formulations. Any US patents that may issue from these applications will expire in 2032 or 2033.

Fate also licenses exclusive rights from Stanford University to a PCT application directed to novel Wnt proteins that provide enhanced characteristics for producing therapeutic formulations of Wnt proteins, formulations of such proteins, and methods of manufacturing such proteins. If issued, patent protection is expected to extend to 2032.

Fate has also obtained rights, as the successor in interest to Verio Therapeutics, to a portfolio of US and international patents and patent applications owned by the Ottawa Hospital Research Institute that supports the program for the treatment of muscle degeneration. This portfolio includes patent applications directed to a novel population of SSCs, enhanced Wnt protein analogs, and the modulation of SSCs to promote muscle regeneration. Any patents that may issue from these pending patent applications will expire between 2022 and 2033.

Management

Christian Weyer, M.D., M.A.S. has served as president and chief executive officer and a director since October 2012. Prior to joining Fate, Dr. Weyer spent 12 years at Amylin Pharmaceuticals, most recently as senior vice president of Research and Development until the completion of Amylin's acquisition by Bristol-Myers Squibb in August 2012, and previously as vice president of Medical Development and vice president of Corporate Development. Prior to joining Amylin, Dr. Weyer spend three years, from 1997 to 2000, at the National Institutes of Health, NIDDK, in Phoenix, Arizona, where he conducted clinical research on the pathogenesis of obesity and type 2 diabetes. Dr. Weyer holds an MD from the University of Düsseldorf, Germany, and a postdoctoral master's degree in clinical research from the University of California, San Diego.

J. Scott Wolchko has served as chief financial officer since the commencement of Fate's operations in September 2007 and as chief operating officer since February 2013. From July 2001 to September 2007, Mr. Wolchko served as the chief financial officer of Bocada, an enterprise software company that specializes in data protection management. Mr. Wolchko began his career in 1994 at Morgan Stanley's Investment Banking Health Care Group, where he assisted emerging growth companies in the life sciences sector complete capital-raising and M&A transactions. Mr. Wolchko holds an MS in biochemical engineering from the University of Virginia, and a BS in biomedical engineering from the University of Vermont.

Pratik S. Multani, M.D., M.S. has served as chief medical officer since May 2013 and was previously senior vice president of Clinical Development from May 2011 to May 2013, and vice president of Clinical Development from April 2009 to May 2011. From August 2007 to March 2009, Dr. Multani was vice president of Clinical Development at Kalypsys, a pharmaceutical company, where he advanced the development of multiple compounds in the therapeutic areas of pain and inflammation and metabolic diseases. From 2005 to 2007, he served as senior vice president of Clinical Development and then chief medical officer at Kanisa Pharmaceuticals. From 1999 to 2004, Dr. Multani worked at Biogen-Idec, advancing from Associate Director of Oncology and Hematology to Senior Director of Medical Research. Dr. Multani holds an MS in epidemiology from Harvard School of Public Health, an MD from Harvard Medical School and a BS in chemistry and biology from Yale University. He completed his Internal Medicine residency at the Massachusetts General Hospital followed by a medical oncology fellowship at the Dana Farber/Partners joint program, after which he was a member of the transplant unit at Massachusetts General Hospital.

Daniel D. Shoemaker, Ph.D. has served as chief technology officer since February 2009 and leads the drug discovery efforts. From 2003 to 2009, Dr. Shoemaker was chief scientific officer of ICxBiosystems, a biotechnology firm that develops advanced detection technologies for use in biodefense, cancer and prenatal diagnostics. From 2003 to 2005, he was chief scientific officer of GHC Technologies, a biotechnology company. From 1998 to 2003, Dr. Shoemaker held several positions at Merck Research Laboratories, including director of Target Discovery, senior director at Rosetta Inpharmatics and research fellow in the Department of Molecular Neurosciences, where his main focus was on target identification and biomarker discovery. Dr. Shoemaker holds a Ph.D. in biochemistry from Stanford University and a BS in biochemistry from the University of California, Santa Barbara.

Peter Flynn, Ph.D. has served as senior vice president, Early Program Development since February 2013. He served as vice president of Biologic Therapeutics and iPSC Technology from May 2011 to February 2013 and senior director of Protein Discovery from May 2009 to May 2011. Prior to joining Fate, from January 2007 to May 2009, he was vice president of Research for Ren Pharmaceuticals, a renal and cario-renal therapeutics company. Prior to Ren, from March 2001 to January 2007, Dr. Flynn was director of Biochemistry Research at KaloBios Pharmaceuticals, an antibody therapeutics company. Prior to the formation of KaloBios, Dr. Flynn was a researcher at UCSF Comprehensive Cancer Center. Dr. Flynn holds a Ph.D. from the ICRF London (Cancer Research UK) and a B.Sc. in molecular biology from University College London.

FATE Income Statement 2013E-2020E

INCOME STATEMENT ($M)	1Q13A	2Q13A	3Q13E	4Q13E	2013E	2014E	2015E	2016E	2017E	2018E	2019E	2020E
REVENUES												
Product Revenue	$ -	$ -	$ -	$ -	$ -	$ -	$ -	$ -	$ -	$ 12.2	$ 42.6	$ 143.7
Collaboration Revenue	0.2	0.2			0.4	-	-	-	-	-	-	-
Other Revenue	0.2	0.2	-	-	0.3	-	-	-	-	-	-	-
TOTAL REVENUES	**$ 0.4**	**$ 0.4**	**$ -**	**$ -**	**$ 0.8**	**$ -**	**$ -**	**$ -**	**$ -**	**$ 12.2**	**$ 42.6**	**$ 143.7**
EXPENSES (GAAP)												
Cost of Goods Sold (COGS)	$ -	$ -	$ -	$ -	$ -	$ -	$ -	$ -	$ -	$ 0.8	$ 4.3	$ 10.8
R&D Expense	2.8	2.8	2.8	2.8	11.1	20.0	30.0	30.0	30.0	30.0	30.0	30.0
SG&A Expense	1.4	1.4	1.9	1.9	6.6	10.1	14.1	18.1	22.1	26.1	30.1	34.1
Other	-	-	-	-	-	-	-	-	-	-	-	-
TOTAL EXPENSES	4.2	4.2	4.7	4.7	17.7	30.1	44.1	48.1	52.1	56.9	64.4	74.9
Operating Income	**(3.8)**	**(3.8)**	**(4.7)**	**(4.7)**	**(16.9)**	**(30.1)**	**(44.1)**	**(48.1)**	**(52.1)**	**(44.7)**	**(21.7)**	**68.8**
Depreciation and amortization	-	-	-	-	-	-	-	-	-	-	-	-
EBIT	(3.8)	(3.8)	(4.7)	(4.7)	(16.9)	(30.1)	(44.1)	(48.1)	(52.1)	(44.7)	(21.7)	68.8
Interest and other income	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Interest and other expense	(0.1)	(0.1)	(0.1)	(0.1)	(0.4)	(0.4)	(0.4)	(0.4)	(0.4)	(0.4)	(0.4)	(0.4)
Other Income (Expense)	(0.6)	(0.6)	-	-	(1.3)	-	-	-	-	-	-	-
Interest and Other Income (Expense)	(0.7)	(0.7)	(0.1)	(0.1)	(1.6)	(0.4)	(0.4)	(0.4)	(0.4)	(0.4)	(0.4)	(0.4)
Pre-Tax Income	(4.5)	(4.5)	(4.7)	(4.7)	(18.6)	(30.5)	(44.5)	(48.5)	(52.5)	(45.1)	(22.1)	68.4
Income Taxes	-	-	-	-	-	-	-	-	-	-	-	-
Net Income (GAAP)	**$ (4.5)**	**$ (4.5)**	**$ (4.7)**	**$ (4.7)**	**$ (18.6)**	**$ (30.5)**	**$ (44.5)**	**$ (48.5)**	**$ (52.5)**	**$ (45.1)**	**$ (22.1)**	**$ 68.4**
EPS (GAAP) (basic)	$ (0.57)	$ (0.57)	$ (0.23)	$ (0.23)	$ (1.60)	$ (1.43)	$ (1.82)	$ (1.62)	$ (1.56)	$ (1.22)	$ (0.57)	$ 1.68
EPS (GAAP) (diluted)	**$ (0.57)**	**$ (0.57)**	**$ (0.23)**	**$ (0.23)**	**$ (1.60)**	**$ (1.43)**	**$ (1.82)**	**$ (1.62)**	**$ (1.56)**	**$ (1.22)**	**$ (0.57)**	**$ 1.68**
Total of Reconciliation Items	0.1	0.1	-	-	0.2	-	-	-	-	-	-	-
Net Income (Non-GAAP)	$ (4.4)	$ (4.4)	$ (4.7)	$ (4.7)	$ (18.4)	$ (30.5)	$ (44.5)	$ (48.5)	$ (52.5)	$ (45.1)	$ (22.1)	$ 68.4
Impact of Adjustments to EPS	0.01	0.01	-	-	0.02	-	-	-	-	-	-	-
EPS (Non-GAAP) (basic)	$ (0.56)	$ (0.56)	$ (0.23)	$ (0.23)	$ (1.57)	$ (1.43)	$ (1.82)	$ (1.62)	$ (1.56)	$ (1.22)	$ (0.57)	$ 1.68
EPS (Non-GAAP) (diluted)	$ (0.56)	$ (0.56)	$ (0.23)	$ (0.23)	$ (1.57)	$ (1.43)	$ (1.82)	$ (1.62)	$ (1.56)	$ (1.22)	$ (0.57)	$ 1.68
Weighted average shares outstanding (basic)	8.0	8.0	20.8	20.8	14.4	21.4	24.6	30.1	33.7	37.1	39.0	40.6
Weighted average shares outstanding (diluted)	8.0	8.0	20.8	20.8	14.4	21.4	24.6	30.1	33.7	37.1	39.0	40.6

Source: Company reports and BMO Capital Markets

FATE Balance Sheet 2012A-2020E

BALANCE SHEET (M)	2012A	2013E	2014E	2015E	2016E	2017E	2018E	2019E	2020E
Current Assets									
Cash and cash equivalents	$ 9.1	$ 57.0	$ 26.5	$ 57.0	$ 83.5	$ 31.1	$ 61.0	$ 38.9	$ 107.3
Short-term investments	-	-	-	-	-	-	-	-	-
Total cash, cash equivalents, and short-term investments	$ 9.1	$ 57.0	$ 26.5	$ 57.0	$ 83.5	$ 31.1	$ 61.0	$ 38.9	$ 107.3
Accounts Receivables	-	-	-	-	-	-	-	-	-
Restricted Cash	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1
Inventories	-	-	-	-	-	-	-	-	-
Prepaid and other current assets	0.7	0.4	0.4	0.4	0.4	0.4	0.4	0.4	0.4
Total Current Assets	$ 9.9	$ 57.5	$ 27.0	$ 57.5	$ 84.0	$ 31.6	$ 61.5	$ 39.4	$ 107.8
Leasehold improvements	-	-	-	-	-	-	-	-	-
Property and equipment, net	1.2	0.9	0.9	0.9	0.9	0.9	0.9	0.9	0.9
Patents and licensed technology	-	-	-	-	-	-	-	-	-
Intangibles, net	-	-	-	-	-	-	-	-	-
Other assets	-	-	-	-	-	-	-	-	-
TOTAL ASSETS	**$ 11.1**	**$ 58.4**	**$ 27.9**	**$ 58.4**	**$ 85.0**	**$ 32.5**	**$ 62.4**	**$ 40.3**	**$ 108.8**
Current Liabilities									
Accounts payable	2.3	3.1	3.1	3.1	3.1	3.1	3.1	3.1	3.1
Accrued payroll	-	-	-	-	-	-	-	-	-
Accrued expenses	-	-	-	-	-	-	-	-	-
Accrued interest	-	-	-	-	-	-	-	-	-
Payables to related parties	-	-	-	-	-	-	-	-	-
Income taxes payable	-	-	-	-	-	-	-	-	-
Current portion of other long-term obligations	1.9	4.7	4.7	4.7	4.7	4.7	4.7	4.7	4.7
Current portion of deferred rent	0.3	0.3	0.3	0.3	0.3	0.3	0.3	0.3	0.3
Current portion of deferred revenue	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Other current liabilities	0.3	0.3	0.3	0.3	0.3	0.3	0.3	0.3	0.3
Total Current Liabilities	$ 4.9	$ 8.4	$ 8.4	$ 8.4	$ 8.4	$ 8.4	$ 8.4	$ 8.4	$ 8.4
Convertible notes payable	-	-	-	-	-	-	-	-	-
Accrued interest on convertible notes payable	-	-	-	-	-	-	-	-	-
Other long-term obligations, less current portion	1.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7
Deferred revenue, less current portion	-	-	-	-	-	-	-	-	-
Deferred rent	0.1	-	-	-	-	-	-	-	-
Other liabilities	0.7	2.0	2.0	2.0	2.0	2.0	2.0	2.0	2.0
TOTAL LIABILITIES	$ 7.4	$ 11.1	$ 11.1	$ 11.1	$ 11.1	$ 11.1	$ 11.1	$ 11.1	$ 11.1
Shareholder's Equity									
Convertible preferred stock	56.5	56.5	56.5	56.5	56.5	56.5	56.5	56.5	56.5
Common stock, at par	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Additional paid-in capital	12.8	75.0	75.0	150.0	225.0	225.0	300.0	300.0	300.0
Accumulated other comprehensive income	-	-	-	-	-	-	-	-	-
Accumulated deficit	(65.6)	(84.2)	(114.7)	(159.1)	(207.6)	(260.1)	(305.1)	(327.2)	(258.8)
TOTAL SHAREHOLDER'S EQUITY (DEFICIT)	$ 3.7	$ 47.3	$ 16.9	$ 47.4	$ 73.9	$ 21.4	$ 51.4	$ 29.3	$ 97.7
TOTAL LIABILITIES AND SHAREHOLDER'S EQUITY	**$ 11.1**	**$ 58.4**	**$ 27.9**	**$ 58.4**	**$ 85.0**	**$ 32.5**	**$ 62.4**	**$ 40.3**	**$ 108.8**

Source: Company reports and BMO Capital Markets

FATE Cash Flow Statement 2012A-2020E

CASH FLOW STATEMENT (M)	2012A	2013E	2014E	2015E	2016E	2017E	2018E	2019E	2020E
Cash Flow From Operating Activities									
Net Income	$ (14.2)	$ (4.7)	$ (8.0)	$ (11.5)	$ (12.5)	$ (13.5)	$ (9.5)	$ (3.3)	$ 28.8
Adjustments to reconcile net income to cash from operations									
Depreciation & amortization	0.6	0.3	0.3	0.3	0.3	0.3	0.3	0.3	0.3
Amortization of premium on investments, net	0.1	-	-	-	-	-	-	-	-
Gain on disposal of property and equipment	-	-	-	-	-	-	-	-	-
Stock-based compensation	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2
Deferred income taxes	-	-	-	-	-	-	-	-	-
Other	0.4	(0.2)	(0.2)	(0.2)	(0.2)	(0.2)	(0.2)	(0.2)	(0.2)
Working Capital Adjustments									
Prepaids and other assets	(0.4)	-	-	-	-	-	-	-	-
Accounts payable	0.4	-	-	-	-	-	-	-	-
Accrued payroll	-	-	-	-	-	-	-	-	-
Accrued expenses	-	-	-	-	-	-	-	-	-
Accrued interest	-	-	-	-	-	-	-	-	-
Receivable from collaborative partners	-								
Payable to related parties	-	-	-	-	-	-	-	-	-
Income taxes payable	-	-	-	-	-	-	-	-	-
Deferred revenue	(0.1)	-	-	-	-	-	-	-	-
Deferred rent	(0.2)	-	-	-	-	-	-	-	-
Other assets and liabilities, net	-	-	-	-	-	-	-	-	-
Total Working Capital Increase (Decrease)	$ (0.3)	$ -	$ -	$ -	$ -	$ -	$ -	$ -	$ -
TOTAL CASH FROM OPERATIONS	**$ (13.3)**	**$ (4.4)**	**$ (7.7)**	**$ (11.2)**	**$ (12.2)**	**$ (13.2)**	**$ (9.2)**	**$ (3.0)**	**$ 29.1**
Cash From Investing Activities									
Purchases of short-term investments	-	-	-	-	-	-	-	-	-
Maturities and sales of short-term investments	-	-	-	-	-	-	-	-	-
Purchases of property and equipment	(0.7)	-	-	-	-	-	-	-	-
Acquisitions of patents	-	-	-	-	-	-	-	-	-
Acquisitions of licenses	-	(0.3)	(0.3)	(0.3)	(0.3)	(0.3)	(0.3)	(0.3)	(0.3)
Increase in patents, deposits and other assets	-	-	-	-	-	-	-	-	-
TOTAL CASH FROM INVESTING	$ (0.7)	$ (0.3)	$ (0.3)	$ (0.3)	$ (0.3)	$ (0.3)	$ (0.3)	$ (0.3)	$ (0.3)
Cash From Financing Activities									
Proceeds from long-term debt borrowings	16.7	-	-	-	-	-	-	-	-
Repayment of borrowings	(0.3)	-	-	-	-	-	-	-	-
Payments of financing costs for an initial public offering	-	-	-	-	-	-	-	-	-
Proceeds from exercise of common stock options	-	-	-	-	-	-	-	-	-
Payments under capital lease obligation	-	-	-	-	-	-	-	-	-
Common stock issuance	0.2	-	-	-	-	-	-	-	-
TOTAL CASH FROM FINANCING	$ 16.7	$ -	$ -	$ -	$ -	$ -	$ -	$ -	$ -
Increase (decrease) in cash and cash equivalents	2.7	(4.7)	(8.0)	(11.5)	(12.5)	(13.5)	(9.5)	(3.3)	28.8
Cash and cash equivalents at beginning of year	6.4	61.7	34.5	68.5	96.0	44.6	70.5	42.2	78.5
Cash and cash equivalents at end of year	$ 9.1	$ 57.0	$ 26.5	$ 57.0	$ 83.5	$ 31.1	$ 61.0	$ 38.9	$ 107.3

Source: Company reports and BMO Capital Markets

ProHema U.S. Market 2013E-2025E

ALLOGENEIC TRANSPLANT	2013E	2014E	2015E	2016E	2017E	2018E	2019E	2020E	2021E	2022E	2023E	2024E	2025E
U.S. MARKET													
Stem Cell Transplant - Global Market	60,000	64,800	69,984	75,583	81,629	88,160	95,212	102,829	111,056	119,940	129,535	139,898	151,090
Growth Rate		8.0%	8.0%	8.0%	8.0%	8.0%	8.0%	8.0%	8.0%	8.0%	8.0%	8.0%	8.0%
% Allogeneic													
Global Allogeneic Hematopoietic Stem Cell Transplant (HSCT) Patients	24,000	25,920	27,994	30,233	32,652	35,264	38,085	41,132	44,422	47,976	51,814	55,959	60,436
% of Patients in U.S.													
U.S. Allogeneic HSCT Patients	9,600	10,368	11,197	12,093	13,061	14,106	15,234	16,453	17,769	19,190	20,726	22,384	24,174
% Cord Blood													
U.S. Eligible Patients	1,920	2,851	3,919	5,140	6,530	8,463	9,140	9,872	10,661	11,514	12,435	13,430	14,505
ProHema Penetration	0.0%	0.0%	0.0%	0.0%	0.0%	2.5%	5.0%	7.5%	10.0%	12.5%	15.0%	17.5%	20.0%
ProHema Patients	0	0	0	0	0	122	377	639	970	1,342	1,753	2,237	2,779
Pricing	$ 100,000	$ 100,000	$ 100,000	$ 100,000	$ 100,000	$ 100,000	$ 100,000	$ 100,000	$ 100,000	$ 100,000	$ 100,000	$ 100,000	$ 100,000
ProHema U.S. Sales ($M)	**$ -**	**$ -**	**$ -**	**$ -**	**$ -**	**$ 12.2**	**$ 37.7**	**$ 63.9**	**$ 97.0**	**$ 134.2**	**$ 175.3**	**$ 223.7**	**$ 277.9**
Royalty Rate 100.0%													
ProHema U.S. Royalties (M)	**$ -**	**$ -**	**$ -**	**$ -**	**$ -**	**$ 12.2**	**$ 37.7**	**$ 63.9**	**$ 97.0**	**$ 134.2**	**$ 175.3**	**$ 223.7**	**$ 277.9**

Source: Company reports and BMO Capital Markets

ProHema Ex-U.S. Market 2013E-2025E

ALLOGENEIC TRANSPLANT	2013E	2014E	2015E	2016E	2017E	2018E	2019E	2020E	2021E	2022E	2023E	2024E	2025E
EX-U.S. MARKET													
Stem Cell Transplant - Global Market	60,000	64,800	69,984	75,583	81,629	88,160	95,212	102,829	111,056	119,940	129,535	139,898	151,090
Growth Rate		8.0%	8.0%	8.0%	8.0%	8.0%	8.0%	8.0%	8.0%	8.0%	8.0%	8.0%	8.0%
% Allogeneic													
Global Allogeneic Hematopoietic Stem Cell Transplant (HSCT) Patients	24,000	25,920	27,994	30,233	32,652	35,264	38,085	41,132	44,422	47,976	51,814	55,959	60,436
% of Patients in Ex-U.S.													
Ex-U.S. Allogeneic HSCT Patients	14,400	15,552	16,796	18,140	19,591	21,158	22,851	24,679	26,653	28,786	31,089	33,576	36,262
% Cord Blood													
Ex-U.S. Eligible Patients	2,880	4,277	5,879	7,709	9,796	12,695	13,711	14,807	15,992	17,271	18,653	20,145	21,757
ProHema Penetration	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	1.3%	2.5%	3.8%	5.0%	6.3%	7.5%	8.8%
ProHema Patients	0	0	0	0	0	0	99	305	517	785	1,335	1,420	1,812
Pricing	$ 50,000	$ 50,000	$ 50,000	$ 50,000	$ 50,000	$ 50,000	$ 50,000	$ 50,000	$ 50,000	$ 50,000	$ 50,000	$ 50,000	$ 50,000
ProHema Ex-U.S. Sales ($M)	**$ -**	**$ -**	**$ -**	**$ -**	**$ -**	**$ -**	**$ 4.9**	**$ 15.3**	**$ 25.9**	**$ 39.3**	**$ 66.7**	**$ 71.0**	**$ 90.6**
Royalty Rate 100.0%													
ProHema Ex-U.S. Royalties (M)	**$ -**	**$ -**	**$ -**	**$ -**	**$ -**	**$ -**	**$ 4.9**	**$ 15.3**	**$ 25.9**	**$ 39.3**	**$ 66.7**	**$ 71.0**	**$ 90.6**

Source: Company reports and BMO Capital Markets

Wnt7a U.S. Market 2013E-2025E

PROTEIN ANALOG	2013E	2014E	2015E	2016E	2017E	2018E	2019E	2020E	2021E	2022E	2023E	2024E	2025E
U.S. MARKET													
DMD Prevalence	14,000	14,420	14,853	15,298	15,757	16,230	16,717	17,218	17,735	18,267	18,815	19,379	19,961
Growth Rate		3.0%	3.0%	3.0%	3.0%	3.0%	3.0%	3.0%	3.0%	3.0%	3.0%	3.0%	3.0%
Wnt7a Penetration	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	2.0%	4.0%	6.0%	8.0%	10.0%	10.0%
Wnt7a Patients	0	0	0	0	0	0	0	861	2,306	3,836	5,456	7,170	7,984
Pricing	$ 300,000	$ 300,000	$ 300,000	$ 300,000	$ 300,000	$ 300,000	$ 300,000	$ 300,000	$ 300,000	$ 300,000	$ 300,000	$ 300,000	$ 300,000
Wnt7a U.S. Sales ($M)	$ -	$ -	$ -	$ -	$ -	$ -	$ -	$ 64.6	$ 172.9	$ 287.7	$ 409.2	$ 537.8	$ 598.8
Royalty Rate 100.0%													
Wnt7a U.S. Royalties (M)	$ -	$ -	$ -	$ -	$ -	$ -	$ -	$ 64.6	$ 172.9	$ 287.7	$ 409.2	$ 537.8	$ 598.8

Source: Company reports and BMO Capital Markets

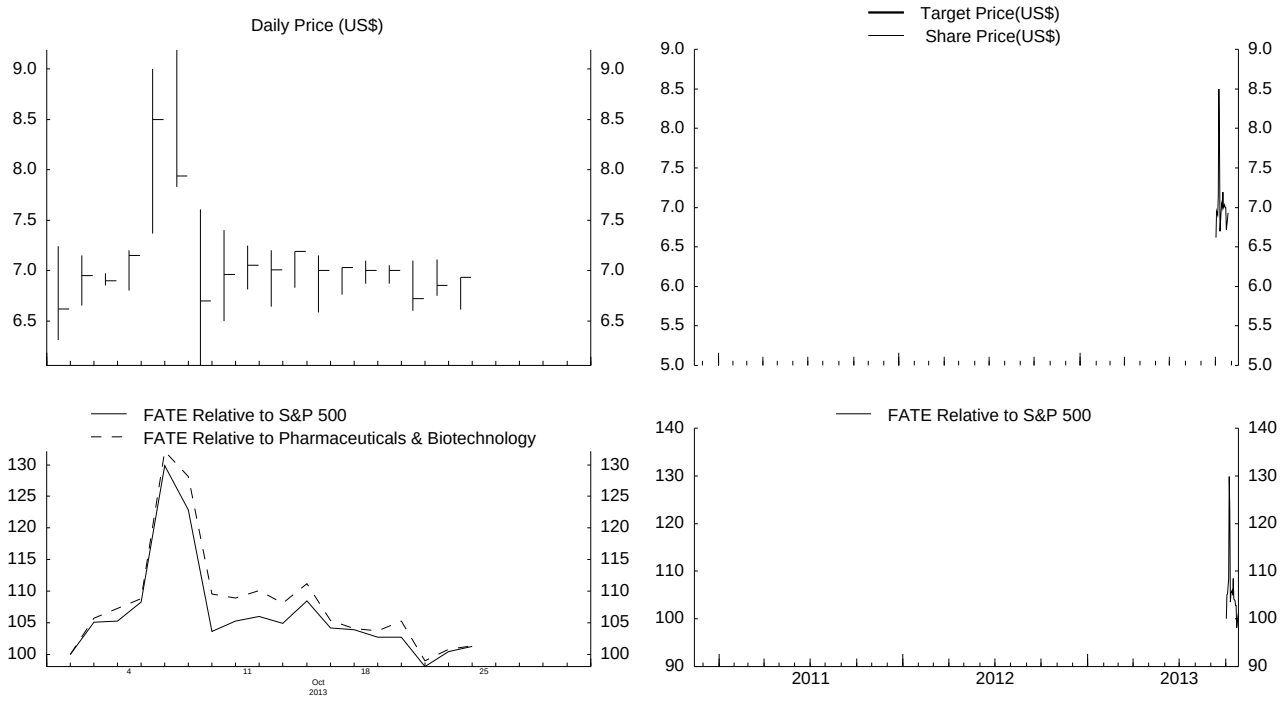
Wnt7a Ex-U.S. Market 2013E-2025E

PROTEIN ANALOG	2013E	2014E	2015E	2016E	2017E	2018E	2019E	2020E	2021E	2022E	2023E	2024E	2025E
EX-U.S. MARKET													
DMD Prevalence	23,000	23,690	24,401	25,133	25,887	26,663	27,463	28,287	29,136	30,010	30,910	31,837	32,793
Growth Rate		3.0%	3.0%	3.0%	3.0%	3.0%	3.0%	3.0%	3.0%	3.0%	3.0%	3.0%	3.0%
Wnt7a Penetration	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	1.0%	2.0%	3.0%	4.0%	5.0%
Wnt7a Patients	0	0	0	0	0	0	0	0	728	1,951	3,246	4,616	6,067
Pricing	$ 300,000	$ 300,000	$ 300,000	$ 300,000	$ 300,000	$ 300,000	$ 300,000	$ 300,000	$ 300,000	$ 300,000	$ 300,000	$ 300,000	$ 300,000
Wnt7a Ex-U.S. Sales ($M)	$ -	$ -	$ -	$ -	$ -	$ -	$ -	$ -	$ 54.6	$ 146.3	$ 243.4	$ 346.2	$ 455.0
Royalty Rate 100.0%													
Wnt7a Ex-U.S. Royalties (M)	$ -	$ -	$ -	$ -	$ -	$ -	$ -	$ -	$ 54.6	$ 146.3	$ 243.4	$ 346.2	$ 455.0

Source: Company reports and BMO Capital Markets

Other companies mentioned (priced as of the close on October 25, 2013):
GlaxoSmithKline (GSK, $52.05, not rated)
Prosensa (RNA, $4.32, not rated)
Sarepta Therapeutics (SRPT, $42.78, not rated)
PTC Therapeutics (PTCT, $20.71, not rated)

Fate Therapeutics (FATE)



FATE - Rating as of 9-Oct-13 = NR

Last Daily Data Point: October 24, 2013

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Hold	Market Perform	59.4%	13.1%	51.1%	56.9%	50.2%	41.7%
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