Fate Therapeutics (FATE)

Initiating Coverage With an OUTPERFORM Rating and $14 Price Target; Fated to Create Better Stem Cells

- **FATE is developing modified hematopoietic stem cells (HSCs) to improve outcomes in allogeneic transplants.** These modified HSCs could improve outcomes in hematological cancers and lysosomal storage diseases (LSDs), where stem cell transplants are the only potentially curative therapy.
- **FATE's lead product candidate, ProHema, is comprised of *ex vivo* optimized HSCs derived from umbilical cord blood.** A Phase Ib study of ProHema in patients undergoing stem cell transplants showed an improvement of 3 days in median engraftment time, relative to historical controls, and patients also experienced better outcomes in various secondary measures.
- **The company has developed a new formulation of ProHema that appears, based on preclinical models, to be even more effective at improving homing and engraftment of transplanted stem cells.** The improved ProHema formulation is expected to enter into a Phase II study in H1:14.
- **The company is also developing analogs of the Wnt7a protein to stimulate satellite stem cells (SSCs) in order to improve outcomes in muscular dystrophies, having overcome technical barriers that prevented others from developing Wnt-based therapeutics.** We believe FATE's Wnt7a analogs compare favorably to other companies' therapies in relevant animal models and has the advantage of not being limited to a specific genetic subset of patients.
- **In 2014, we expect FATE to begin studies of ProHema for pediatric cancer and LSDs and file an IND for its SSC program in muscular dystrophies.** Data from the studies and the Phase II trial of ProHema in adult cancer should become available in 2015.
- **Initiating coverage with an OUTPERFORM rating and $14 price target.** We arrive at our $14 price target by applying a 6x multiple to an estimated $380M in revenues in 2019, discounted by 35% annually.

FYE Dec	2013E	2014E			2015E		
REV	ACTUAL	CURR.	PREV.	CONS.	CURR.	PREV.	CONS.
Q1 Mar	--	0.0E		N/A	0.0E		N/A
Q2 Jun	0.8E	0.0E		N/A	0.0E		N/A
Q3 Sep	0.0E	0.0E		N/A	0.0E		N/A
Q4 Dec	0.0E	0.0E		N/A	0.0E		N/A
Year*	**0.8E**	**0.0E**		**N/A**	**0.0E**		**N/A**
Change	--	-100%			--		
	2013E	2014E			2015E		
EPS	ACTUAL	CURR.	PREV.	CONS.	CURR.	PREV.	CONS.
Q1 Mar	--	($0.23)E		--	($0.26)E		--
Q2 Jun	($7.41)E	($0.26)E		--	($0.26)E		--
Q3 Sep	($0.33)E	($0.28)E		--	($0.27)E		--
Q4 Dec	($0.21)E	($0.26)E		--	($0.26)E		--
Year*	**($7.94)E**	**($1.03)E**		**--**	**($1.05)E**		**--**
P/E	--	--			--		
Change	--	87%			-2%		

Consensus estimates are from Thomson First Call.
** Numbers may not add up due to rounding.*

October 27, 2013

Price
$6.93

Rating
OUTPERFORM

12-Month Price Target
$14

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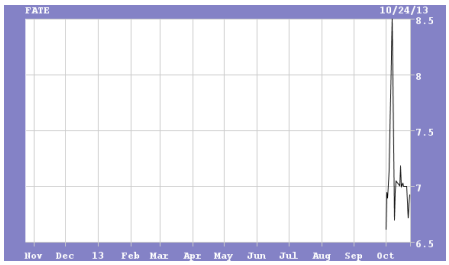
Company Information

Shares Outst (M)	20.6
Market Cap (M)	$142.9
52-Wk Range	$6.06 - $9.19
Book Value/sh	$-3.60
Cash/sh	$2.87
Enterprise Value (M)	$84.4
LT Debt/Cap %	-0.2
Cash Burn (M)	$15.8

Company Description

Fate Therapeutics, Inc., is based in San Diego, California, and is focused on modulating the activity of adult stem cells used in stem cell transplants for the treatment of hematological cancers, rare diseases and muscular dystrophies.



Source: Thomson Reuters

Investment Thesis

Fate Therapeutics is a cell therapy company developing two product platforms that modulate the activity of adult stem cells for the treatment of rare diseases. One platform focuses on the *ex vivo* optimization of hematopoietic stem cells (HSC), and the other focuses on the *in vivo* activation of satellite stem cells (SSC). ProHema, the lead product from the HSC platform, is derived from modified umbilical cord blood (UCB) and should resume a Phase II trial in adult patients with hematological malignancies in H1:14 with an improved formulation relative to its Phase I/II study. ProHema is also in the preclinical stage for pediatric hematological malignancies and for certain lysosomal storage disorders (LSD). Fate's SSC platform is initially focused on analogs of Wnt7a proteins, which are in preclinical testing for muscular dystrophies and neuromuscular disorders.

Valuation

Our price target of $14 is derived from a 6x multiple of 2019 worldwide estimated sales of all products of $380M, discounted by 35%. Our model assumes that with the approval of ProHema, there is significant growth in the use of cord blood as the effective availability of transplantable units should increase, along with ProHema's improvements in efficacy driving use. ProHema's hematologic malignancy indication could garner $175M in 2019 revenues, its rare disease application another $151M, and finally the satellite stem cell program revenues could amount to $53M.

Risks

Risks to the achievement of our price target include failure to gain approval for ProHema, failure to achieve sales estimates for ProHema and failure to achieve earnings estimates.

Key points

- Fate's lead candidate, ProHema, is an *ex vivo* optimized HSC therapeutic derived from umbilical cord blood for the treatment of hematological malignancies and inherited metabolic diseases.
- ProHema has shown superior time-to-engraftment and improvements in other secondary endpoints in an initial cohort of patients in a Phase II trial for hematologic malignancies.
- The company has developed a new formulation of ProHema that appears, based on preclinical models, to be even more effective at improving homing and engraftment of transplanted stem cells, and we expect Fate to enroll patients in a Phase II study with that formulation in H1:14.
- Fate is also developing analogs of the Wnt7a protein to stimulate SSCs for muscular regeneration, having overcome barriers related to the molecular characteristics of Wnt proteins that have prevented other manufacturers from developing therapeutics based on it.
- We expect that Fate will be able to begin a study of ProHema in patients with LSDs in 2014, along with filing its IND for its SSC program in muscular dystrophies.
- Applying a 6x multiple to an estimated $380M in revenues in 2019, discounted by 35% annually, we estimate FATE's value to be $14/share.

Fate Therapeutics, Inc. Overview

Fate Therapeutics is based in San Diego, California and focuses on improving outcomes of hematopoietic stem cell transplants (HSCTs) for oncology and rare diseases and the modification of satellite stem cells (SSC) for use in inherited muscle disorders. The company's lead product candidate, ProHema, is likely to enter its Phase II trial for hematologic malignancies in adults, with additional clinical testing in children and adolescents also likely to begin in 2014. ProHema and second-generation HSC therapeutics are in preclinical testing for treating lysosomal storage disorders. Fate also has Wnt7a protein analogs which promote SSC-driven muscle regeneration in preclinical testing for muscular dystrophies and neuromuscular disorders.

Upcoming milestones

H1:14	Resume enrollment in the Phase II trial of ProHema in adult patients with hematologic malignancies
H2:14	Initiate Phase I trial of ProHema in children and adolescents with hematologic malignancies
H2:14	Initiate Phase I trial of ProHema in pediatric patients with lysosomal disorders
YE:14	File IND for a Wnt7a analog
Mid:15	Data from Phase II trial of ProHema in hematologic malignancies
H2:15	Data from Phase I trial of ProHema in lysosomal disorders
2015	Safety and efficacy data of the Wnt7a analog in muscular dystrophy patients
YE:15	Initiate Phase III trial of ProHema in adult and pediatric patients with hematologic malignancies
2017	File BLA for ProHema

Figure 1: Product Development Table

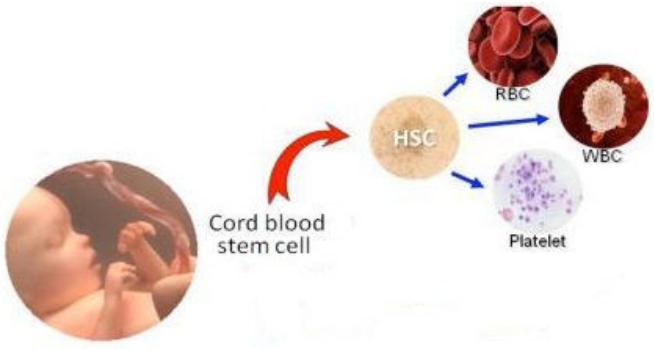
Product	Indication/Field	Stage of Development
ProHema	Adult hematologic malignancies	Phase II
	Pediatric hematologic malignancies	Preclinical
	Lysosomal storage disorders (LSD)	Preclinical
Second Generation HSC Therapeutics	LSD	Preclinical
Wnt7a Protein Analogs	Muscular dystrophy and other neuromuscular disorders	Preclinical

Source: Company data, Wedbush Securities, Inc.

Cord Blood and Hematopoietic Stem Cell Transplantation

Hematopoietic stem cells (HSC) are adult stem cells that can self-renew and differentiate into all other types of blood and immune cells. HSCs are responsible for the constant production of blood during a person's life, and this renewal characteristic provides a therapeutic opportunity for HSCs to treat a variety of disorders affecting the blood and immune systems by replacing the patients' own hematopoietic tissue.

Figure 2: Differentiation of HSCs from Cord Blood

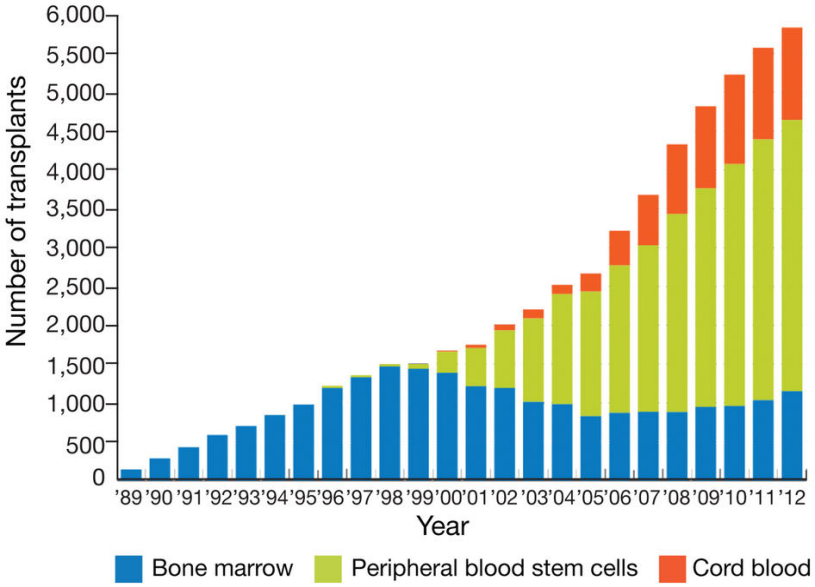


Source: Adapted from cryobank promotional literature

Hematopoietic stem cell transplantation (HSCT) is a commonly used procedure involving the intravenous infusion of stem cells in order to reestablish hematopoietic function in patients with damaged or defective bone marrow or immune systems, most frequently in patients with hematological cancers where the patients' cancerous blood cells are ablated by chemotherapy and/or radiation and replaced with healthy HSCs. These stem cells can come from the patient itself (autologous) or from a donor (allogeneic). For genetic diseases, the transplanted HSCs must be allogeneic.

HSCT is the only curative option for many patients with hematological malignancies like leukemias, lymphomas and myelomas and also certain genetic diseases. The bone marrow has been the classic source of stem cells for HSCT, but its use has decreased over time due to the physical burden harvesting places on the donor. Today, the most common source of stem cells for HSCT is peripheral blood, where stem cells are separated from the circulating blood via apheresis; however, the use of umbilical cord blood has increased dramatically in recent years, with ~1200 cord blood HSCTs being performed in the US (over 20% of allogeneic HSCTs) and over 800 in Europe.

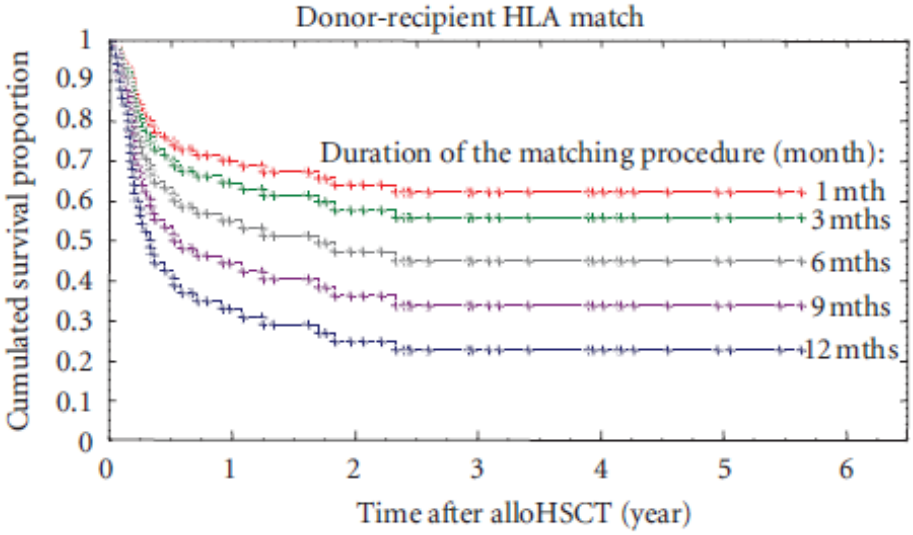
Figure 3: Increasing Use of Cord Blood in Hematopoietic Stem Cell Transplantations

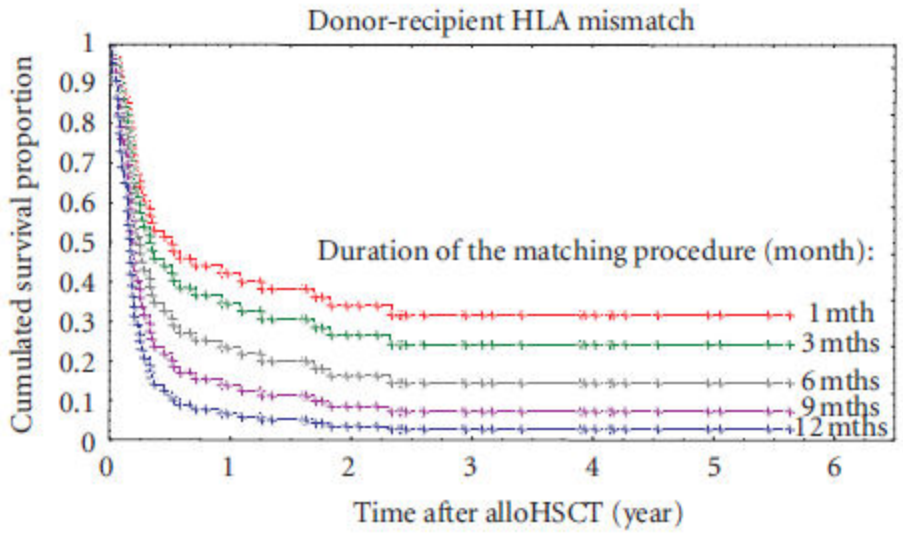


Source: National Marrow Donor Program

Despite the curative potential of HSCT, its use has been limited due to the significant morbidity and mortality associated with the procedure. The mortality rate at one-year post transplant for patients undergoing allogeneic HSCT is over 20%, with higher rates for patients using HSCs from an unrelated donor. The key factors that determine patient outcomes after HSCT include time to hematological reconstitution, conditioning regimen (full- or reduced-intensity bone marrow ablation), and whether the HSCs are human leukocyte antigen (HLA) matched. For allogeneic transplants there always exists the risk of graft versus host disease (GvHD), where the donor stem cells regards the host body as foreign and attacks it. In order to reduce the likelihood of rejection, donor HSCs have to be HLA matched to the patient, with better matches. However, due to the scarcity of matched donors only about 30% of patients are able to find an allogeneic match in the ideal time. With both oncologic applications and LSD transplants, reducing the time to transplant corresponds with improved outcomes.

Figure 4: Time to Transplant Correlates with Survival in HSCTs for Hematologic Malignancies





Source: Mizia et al. (2012) Both Optimal Matching and Procedure Duration Influence Survival of Patients after Unrelated Donor Hematopoietic Stem Cell Transplantation. Bone Marrow Res.

Because of the importance of quickly finding a suitable HSC donor, the use of cord blood in HSCT has increased in recent years since it has less rigorous requirements for HLA matching: four matches out of six antigen markers is acceptable, whereas HSCs from an unrelated bone marrow or peripheral blood donor requires at least five matches out of six markers. HSCs sourced from cord blood account for about 20% of allogeneic HSCTs performed in the US and EU.

Cord blood, which is sourced from either the placenta or umbilical cords of newborns, is 25-50% less likely to cause GvHD since the T-cells of cord blood are immunologically immature and less likely to trigger an immune response. As a result of the reduced risk, patients undergoing HSCT with cord blood can undergo a conditioning regimen that is less intense than with traditional HSCT, which requires high-dose myeloablative chemotherapy prior to transplant in order to provide adequate immunosuppression. Consequently the use of cord blood opens up HSCT procedures for the elderly and other patient groups that cannot undergo intensive therapy. It also increases access to racial minorities, particularly African Americans, who are less likely to find a sibling or matched donor. Today, 44% of all HSCT procedures performed on African-Americans use cord blood, compared to 16% for Caucasians. Additional benefits of cord blood is that it is readily available and non-invasive to collect compared to peripheral or bone marrow, since umbilical cords are a byproduct that would otherwise be discarded post-partum. Because of efforts to promote public banking of cord blood after pregnancies, there are an estimated 600,000 cord blood units in storage around the world that is electronically searchable for quick donor identification and selection.

Figure 5: Comparing Cord Blood to Bone Marrow and Peripheral Blood for use in HSCT

Criteria	Cord Blood	Bone Marrow or Peripheral Blood
Risk of Disease Relapse	Up to 50% reduced.	
Risk of GvHD	Reduced by about 30%.	
Availability	Readily available. Quick search and procurement.	More stringent matching requirements lead to longer match times and decreased availability.
HLA matching	Less stringent.	More stringent.
Racial/ethnic minorities	Higher probability of finding match	Lower probability of matching.
Stress on Donor	None.	Requires collection procedures.

Source: Wedbush Securities, Inc.

A key limitation to using cord blood in adults is that the number of HSCs that can be recovered from umbilical cords is often insufficient to transplant into an average sized adult, resulting in higher rates of graft failure and early mortality than with other stem cell sources. The way the medical community has dealt with this limitation is by combining two cord blood units, but this method adds expenses

(cord blood costs thousands of dollars per unit) and potential complications, including a higher risk for GvHD, as the units are not identical to each other.

Fate intends to address the limitations of cord blood use through its proprietary modulation process, which enhances hematopoietic reconstitution by improving HSC homing and proliferation after transplantation (even potentially with what is currently a sub-optimal cell count in the unit) while maintaining the advantages cord blood transplantation has over peripheral blood and bone marrow sources. Indeed, if Fate's improvements could reduce the cell dose (TNC, total nucleated cells) requirement to 1.5 x 10^7 from 2.5 x 10^7, cord blood match rates would improve by ~50%, so that around 90% of Caucasians and 50%-75% of minorities could find a matched cord unit. Our view is that by reducing this key limitation of cord blood HSCs, use would increase dramatically.

Figure 6: ProHema's Potential to Expand the Use and Improve the Outcome of Umbilical Cord Blood (UCB) Transplants

Criteria	ProHema Cord Blood	Bone Marrow or Peripheral Blood (unrelated)
Time to acquire cells	2-3 weeks	8-12 weeks
Time to engraftment	Reduced	14-22 days
Hospitalization (inpatient stay)	Reduced	24-28 days
Risk of Disease Relapse	Up to 50% reduced	
Risk of GvHD	Reduced by about 30%	

Source: Wedbush Securities, Inc., company reports

ProHema

Fate's HSC platform is based on modifying HSCs *ex vivo* in order to improve its *in vivo* performance. The platform involves inducing changes in the expression of key genes, including CXCR4, in HSCs critical for homing and engraftment in order to enhance hematopoietic reconstitution through a faster and more durable engraftment.

The lead product candidate to come out of their HSC platform is ProHema, which is umbilical cord blood that has been modulated with a prostaglandin E2 analog (16, 16-dimethyl prostaglandin E2, which Fate refers to as FT1050), a key regulator of HSC homeostasis. This analog is resistant to metabolism, giving it a higher duration of activity and the potential to increase the effective dose of HSCs. The enhanced hematopoietic reconstitution that results has the potential to enable greater flexibility with matching donors and the use of less toxic conditioning, while lowering the risk of side effects.

The effect that the prostaglandin E2 (PGE2) pathway has on HSCs was first examined in a paper co-authored by Dr. Leonard Zon, one of Fate's scientific cofounders. The paper, "Prostaglandin E2 regulates vertebrate haematopoietic stem cell homeostasis" (*Nature*, June 2007), detailed studies done on zebrafish and mouse models showing that PGE2 exposure enhanced the quantity of HSCs and improved marrow recovery following irradiation.

Manufacturing Process

ProHema is produced by treating umbilical cord units with FT1050 on the day of the transplant. The process begins after a suitable cryopreserved cord blood unit has been identified based on the patient's HLA type and cell dose requirements. This cord blood unit is thawed and washed, and then incubated with FT1050 in a proprietary modulation process that takes two hours. Afterwards, the modified cord blood unit is washed and undergoes in-lab filtration and final packaging and labeling.

ProHema was initially manufactured using standard processing media, and it was this formulation that was used in the Phase I 'ProHema-01' trial. In Q2:13 Fate completed a series of preclinical studies that supported the use of a nutrient-rich media (NRM) formulation in their ProHema manufacturing process. The studies showed that the potency and efficacy profile of ProHema was significantly improved using the NRM formulation. The new formulation increased the HSC population and expression of PGE2-related genes, which translated into improved levels of homing and engraftment in mouse models.

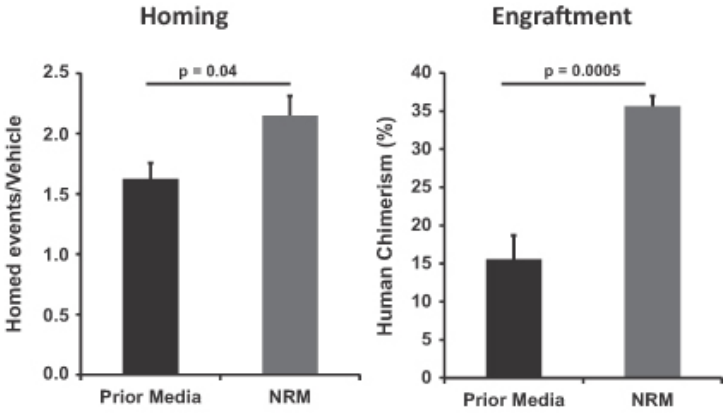
We note that preclinical models of improved homing and engraftment were predictive of the improved clinical outcomes, and so we expect that the better performance of the new formulation in preclinical studies should lead to similarly better performance in the clinic.

Figure 7: Improvement in Homing and Viability with NRM Formulation in *In Vitro* Studies

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%

Source: Fate Therapeutics, Inc.

Figure 8: Improvements in Homing and Engraftment Levels for NRM relative to Phase Ib Formulation in Mouse Model



Source: Fate Therapeutics, Inc.

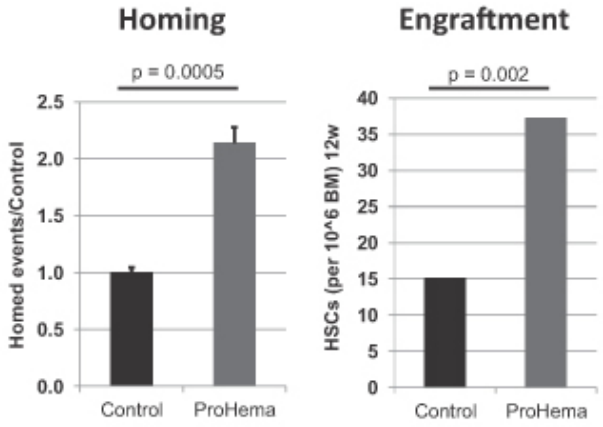
Enrollment in the Phase II 'ProHema-03' trial in adult hematologic malignancies is currently paused while the improved NRM formulation is incorporated into the manufacturing of ProHema. Enrollment is expected to resume during H1:14 when the FDA could approve the IND amendment submitted by Fate to support the use of the NRM formulation.

ProHema for Hematologic Malignancies

Preclinical Studies

Preclinical studies conducted by Fate confirmed the theorized targeted performance and therapeutic benefit of PGE2-modulated HSCs. In a mouse model of HSCT, homing and engraftment was improved with the use of *ex vivo* modulated human HSCs.

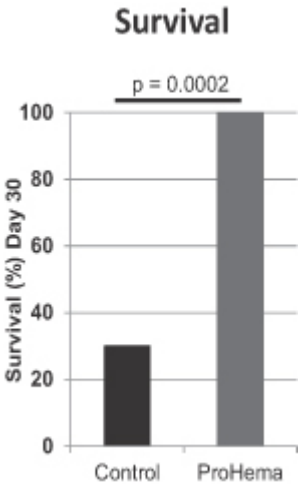
Figure 9: Targeted Properties of ProHema in Mouse Model of HSCT



Source: Fate Therapeutics, Inc. S-1

This superior homing and engraftment translated into improved survival outcomes in mice exposed to lethal amounts of radiation and administered a suboptimal amount of HSCs. Only 30% of mice in the control group were alive at 30 days, compared to all of the mice in the ProHema group.

Figure 10: Survival Benefit of HSCs in Lethally Irradiated Mice



Source: Fate Therapeutics, Inc. S-1

Clinical Study: Phase Ib 'ProHema-01' in Adults

Human proof of concept for ProHema was established in the Phase Ib 'ProHema-01' trial in adult patients with hematological malignancies who underwent a double umbilical cord blood transplant following a conditioning regimen of reduced intensity. Patients were administered an untreated cord blood unit along with either a single ProHema-treated cord blood unit (incubated at 37°C) or a cord blood unit modulated with FT1050 under biologically inactive conditions (incubation at 4°C). The study was conducted at the Dana Farber Cancer Institute and the Massachusetts General Hospital, with results compared to historical outcomes of 53 adult patients with hematologic malignancies that underwent double umbilical cord blood transplants at the same centers. Compared to historical control, the 12 patients in the ProHema group had a statistically significant (p=0.043) improvement in time to neutrophil engraftment, defined as a neutrophil count in peripheral blood above 500 cells per µL. Improvements were also seen in the survival rate at 100 days and the incidence and failure rate of neutrophil engraftment.

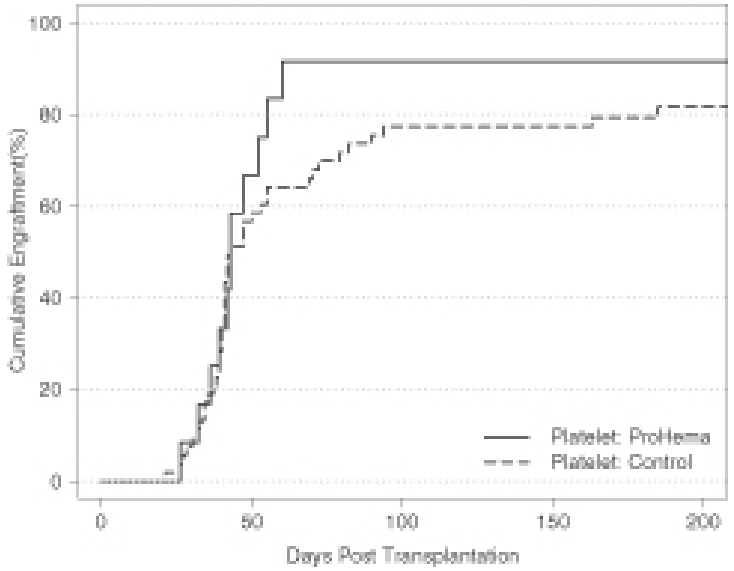
Figure 11: Phase Ib ProHema-01 Trial Results

Cohort	Median Time to Engraftment	Cumulative Incidence of Neutrophil Engraftment at 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%

Source: Fate Therapeutics, Inc. S-1

Patients in the ProHema group also showed improvements in the rate and incidence of platelet engraftment by Day 100. None of the patients in the ProHema cohort experienced secondary graft failure, which is where the graft fails after an initial period of engraftment.

Figure 12: Rate and Incidence of Platelet Engraftment for ProHema versus Historical Control



Source: Fate Therapeutics, Inc. S-1

The incidence of GvHD was also lower with ProHema, with one patient (8%) in the ProHema cohort having acute grade II-IV GvHD compared to 17% for historical control. ProHema was well tolerated, with adverse events consisting of mild-to-moderate infusion-related events.

Current Clinical Study: Phase II 'ProHema-03' in Adults

Fate initiated the Phase II 'ProHema-03' trial in December 2012 in adult patients with hematological malignancies undergoing double umbilical cord blood transfusions after either myeloablative or reduced-intensity conditioning. In the 11 patients that were enrolled, all of which were conditioned using a myeloablative regimen, eight were randomized to receive ProHema plus an untreated cord blood unit and three were randomized to receive two untreated cord blood units. Five of the patients in the ProHema group engrafted prior to the control median of 31 days time-to-engraftment, while two ProHema patients engrafted afterwards (days 40 and 48) and one failed to engraft. None of the patients had secondary graft failure. At a median follow-up period of 156 days, five of the eight ProHema patients remained alive and engrafted, compared to only one of the three control patients.

Although the study remains active for continued follow-up, further enrollment was stopped in order to incorporate ProHema manufactured with the NRM formulation. Fate submitted an amended IND to FDA on Aug. 1, 2013, with the necessary preclinical and product development data to support the use of ProHema formulated with NRM. The company expects to resume enrolling an additional 60 patients in H1:14, with final data available in mid:15.

Pediatric Patients

Fate is also currently conducting the Phase I 'ProHema-02' trial to determine the viability of ProHema in the pediatric patient population, for whom typically only a single cord blood unit is used during umbilical cord blood transplants. The Phase I study has enrolled eight adults with hematologic malignancies who will receive reduced-intensity conditioning followed by a single ProHema cord blood unit. The primary endpoint is safety, with secondary endpoints being rates of engraftment, relapse and survival.

Of the six evaluable patients (median age of 56 years) in the study, two have experienced primary graft failure and none have experienced secondary graft failure. All patients were alive at day 100 and no cases of acute or chronic GvHD has been observed. Based on these results, Fate plans to begin a Phase Ib trial in children and adolescents with hematologic malignancies who will be administered a single ProHema unit. The study will assess safety as defined by neutrophil engraftment and will use the NRM formulation of ProHema. The company could start the study in 2014 under its current IND for ProHema, with the timing delayed if the FDA decides a separate IND for pediatric patients is required.

Future Clinical Study: Phase III trial in adult and pediatric patients

Guidance provided by FDA suggests that a single Phase III trial that enrolls both adults and pediatric patients would be sufficient for approval in both age groups, with a satisfactory primary endpoint being the time to engraftment of neutrophils and/or platelets. Fate could begin a Phase III trial in about 200 patients with hematologic malignancies in 2015.

ProHema for Lysosomal Storage Disorders

HSCT is being used to treat an increasing number of genetic disorders, with the most common use in inherited metabolic diseases such as lysosomal storage disorders (LSD). LSDs are a group of rare autosomal recessive disorders characterized by enzyme deficiencies that result in defective metabolism of macromolecules. The resulting internal accumulation of toxic substances progressively affects the central and peripheral nervous systems, often leading to death in childhood. Although enzyme replacement therapy (ERT) is successful in treating the peripheral manifestations of the disease, the difficulty in crossing the blood-brain barrier has made ERT ineffective for treating the CNS manifestations of the disease. HSCT is a potentially curative option that can provide a constant source of enzyme replacement from the engrafted HSCs, which are not impeded by the blood-brain barrier. The most common LSDs that Fate is likely to target include Hunter's syndrome, Hurler's syndrome, Krabbe's disease (globoid cell leukodystrophy), metachromatic leukodystrophy and Sanfilippo syndrome.

Proof of Concept in Hurler's syndrome

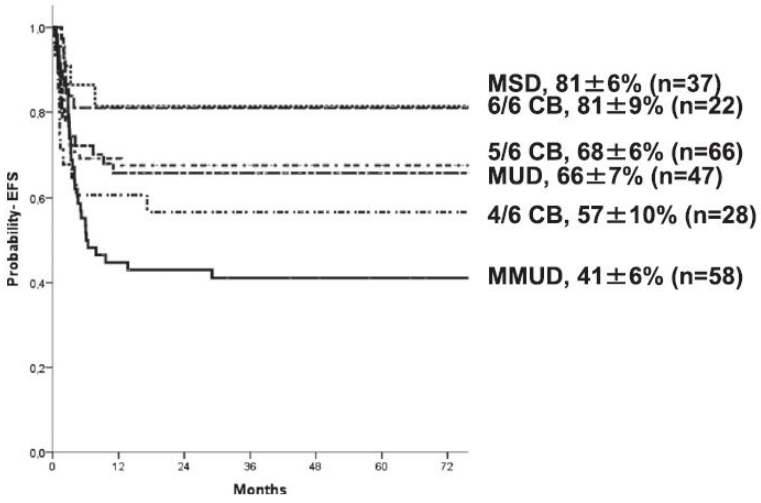
An externally-conducted retrospective analysis of 258 children with Hurler syndrome who underwent HSCT following myeloablative chemotherapy showed that using cord blood in transplants produced similar outcomes to using bone marrow from perfectly matched unrelated donors and siblings. Full-donor chimerism and normal enzyme levels, which are associated with superior neurocognitive outcomes post-engraftment, were higher after using cord blood compared with other graft sources (see Figure 13). Event-free survival (EFS) rates were similar between patients receiving cord blood and those patients with matched unrelated or sibling donors (see Figure 14).

Figure 13: Comparing Donor Chimerism and Leukocyte Enzyme Levels in Alive and Engrafted Patients for Umbilical Cord Blood (UCB) Transplants, Matched Sibling Donors (MSD), Matched Unrelated Donors (MUD) and T-Cell Depleted Matched Unrelated Donors (TCD-MUD).

	MSD (%)	MUD (%)	TCD-MUD (%)	UCB (%)	*P* value
Chimerism	n = 30	n = 35	n = 19	n = 79	
Full (>95%)	21 (70)	26 (74)	9 (50)	69 (93)	.039
Mixed (50-95%)	6	6	4	5	
Mixed (10-50%)	3	3	5	0	
Missing	0	0	1	5	
Enzyme level*					
Normal	16 (54)	23 (66)	5 (53)	64 (98)	.007
Low	10	12	4	1	
Missing	2	0	9	14	

Source: Boelens et al. (2013) "Outcome of Transplantation Using Various Hematopoietic Cell Sources in Children with Hurler Syndrome after Myeloablative Conditioning", Blood

Figure 14: Comparing the Probability of Event-Free Survival (EFS) for Cord Blood (CB) by HLA Matches to Matched Sibling Donors (MSD), Matched Unrelated Donors (MUD) and Mismatched Unrelated Donors (MMUD).

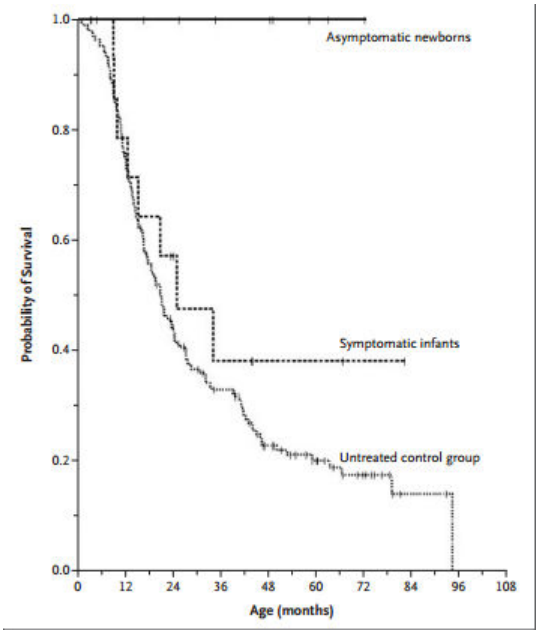


Source: Boelens et al. (2013) "Outcome of Transplantation Using Various Hematopoietic Cell Sources in Children with Hurler Syndrome after Myeloablative Conditioning", Blood

Proof of Concept in Krabbe's disease

An external research study in infants with Krabbe's disease validated the use of umbilical cord blood for HSCT, with superior outcomes seen in early stage patients. In the study, 11 asymptomatic newborns (ages 12 - 44 days) who were diagnosed because of family history and 14 symptomatic infants (ages 142 – 352 days) underwent HSCT with umbilical cord blood from unrelated donors after myeloablative chemotherapy. In the asymptomatic newborns, the rates of engraftment and survival were both 100% at a median follow-up of 3 years, while for the symptomatic infants the rates of engraftment and survival were 100% and 43%, respectively, at a median follow-up of 3.4 years. The natural progression of the disease was favorably altered following transplantation, with the greatest improvements seen in patients who were not yet symptomatic.

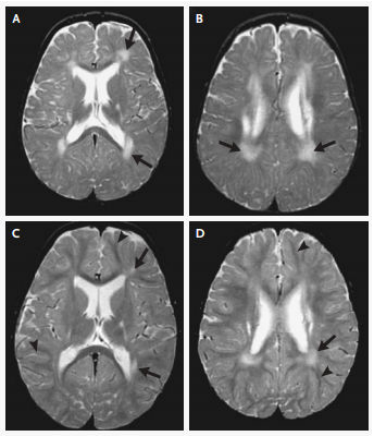
Figure 15: Overall Survival in Infants with Krabbe's Disease after Umbilical Cord Blood Transplants



Source: "Transplantation of Umbilical-Cord Blood in Babies with Infantile Krabbe's Disease". New England Journal of Medicine, 2005; 352:2074

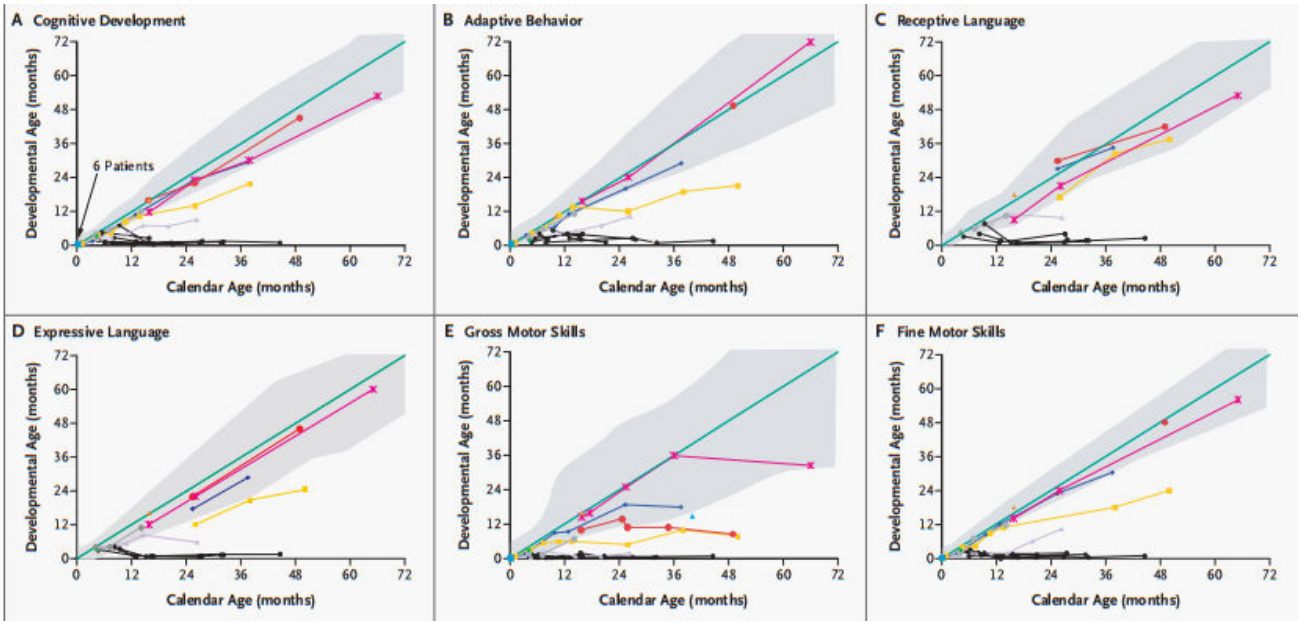
MRI scans verified the progression of myelination and the decrease in the size of abnormal regions of white matter following transplantation (see Figure 15). The importance of early intervention was emphasized with patients who had already progressed to the symptomatic stage at transplantation showing no significant improvement post-transplant in any neurological outcome assessed (see Figure 14). As seen in hematological malignancies, the relatively short time to finding a match is a key advantage for cord blood transplants in this setting, and one that ProHema could build on in these patients by also potentially improving CNS homing.

Figure 16: MRIs of a Newborn Umbilical Cord Blood Transplant Recipient at One Year (panels A and B) and Two Years (panels C and D) Post-transplant Showing Images Through the Level of the Corpus Callosum (panels A and C) and Centrum Semiovale (panels B and D)



Source: "Transplantation of Umbilical-Cord Blood in Babies with Infantile Krabbe's Disease". New England Journal of Medicine, 2005, 352:2076

Figure 17: Comparing Neurodevelopmental Outcomes of Children with Krabbe's Disease who Underwent Cord-Blood Transplantation as Infants (black Line) or Newborns (colored lines) against Typical Healthy Development (green line, with typical variability in shaded area)

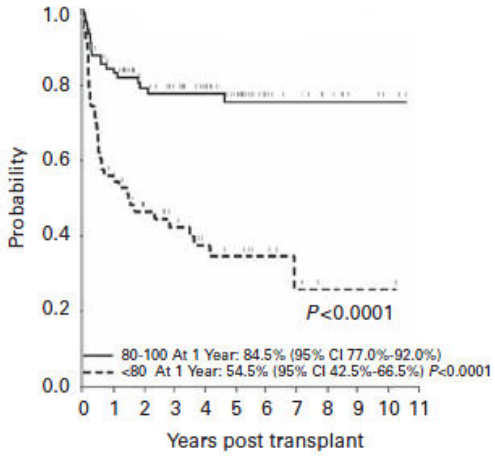


Source: "Transplantation of Umbilical-Cord Blood in Babies with Infantile Krabbe's Disease". New England Journal of Medicine, 2005, 352:2077

Proof of Concept in Multiple LSDs

An externally-conducted retrospective study of 159 patients with a variety of IMDs treated with donor UCB grafts after receiving myeloablative chemotherapy showed that the timing of the transplant in relation to disease stage had a greater impact on outcomes than the specific IMD diagnosis.

Figure 18: Effect of Performance Status (>80% vs. 80-100%) at Time of Transplant on Overall Survival Probability



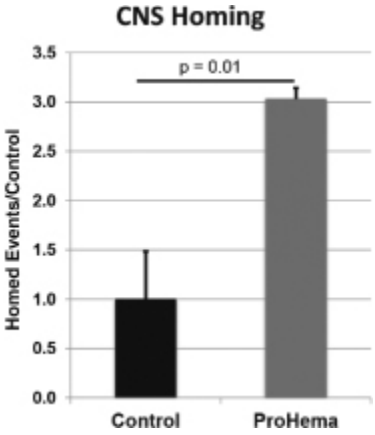
Source: "Umbilical Cord Blood Transplantation for Non-malignant Diseases", Nature.

In an analysis of just the patients with Hunter's (n=6), Hurler's (n=45), Krabbe's disease (n=36), metachromatic leukodystrophy (n=15) and Sanfilippo syndrome (n=19), the probability of overall survival at one and five years was similar. Based on the results of the study, it is reasonable to conclude that early stage success in any LSD should convey similar benefits for ProHema in other LSDs.

Preclinical Study

The improved homing of HSCs across the blood-brain barrier to the central nervous system after FT1050 modulation was established in a study of sub-lethally irradiated immunodeficient mice. The mice were injected with human cord blood-derived HSCs which were treated *ex vivo* with either FT1050 or control. At twenty hours post-injection, the number of human cells in the brain tissue of the mice was three times greater in the FT1050 group compared to control.

Figure 19: Modulated Human HSCs in Immunodeficient Mouse Model



Source: Fate Therapeutics, Inc. S-1

Clinical plan

Fate plans to begin a Phase I trial of ProHema in patients aged 1 to 21 years with demyelinating LSDs in 2014 after filing an IND amendment. Patients in the study will receive a standard myeloablative regimen comprised of multiple high-dose chemotherapeutic agents. Patients in the first cohort will have the dose of one of the agents reduced by 25%, with increasing dose-reductions for subsequent cohorts. The primary endpoint will be neutrophil engraftment, and data is expected to become available in 2015. The company expects to start the study in 2014 under its current IND for ProHema, if FDA agrees.

Second-generation HSC Therapeutic

Fate is also developing second-generation treatments based on its HSC modulation platform with the goal of enhancing the homing potential of HSCs to the CNS and improving the delivery of the essential enzymes patients with LSDs lack.

Competition

There are a number of other products in development for the improvement of cord blood transplants, although most are behind development compared to Fate.

Figure 20: Companies with Products to Assist Umbilical Cord Blood Transplants

Company	Product	Description	Stage
Fate	ProHema	modified HSCs from cord blood	Phase II for hematologic malignancies
Cellerant	CLT-008	*Ex vivo* expanded human myeloid progenitor cells	Phase I/II for hematologic malignancies
Gamida/Teva	StemEx	cord blood cells expanded using a copper chelator and infused with unexpanded portion	Phase III for hematological malignancies (not clear if development will continue)
	NiCord	cord blood expanded using nicotinamide	Phase II for hematological malignancies, sickle cell and myelodysplastic syndrome
Novartis	HSC835	Expanded cord blood HSCs	Phase I/II for hematological malignancies
Tarix Pharmaceuticals	TXA127	Injectable formulation of angiotensin (1-7) peptide to stimulate hematopoietic progenitors	Phase II for hematologic malignancies and inherited metabolic diseases

Source: Wedbush Securities, Inc.

Another competitive threat to the use of modified allogeneic HSCs is the emerging rise of gene therapy. Genetic disorders traditionally required the use of non-defective allogeneic HSCTs, however gene therapy offers the potential for using a patient's own corrected HSCs. The 2012 approval in the EU of Glybera (alipogene tiparvovec) for lipoprotein lipase deficiency was the first approval for a gene therapy product in the Western world. Its manufacturer, the Dutch biotech firm uniQure, has also announced plans to develop a gene therapy treatment for the LSD Sanfilippo syndrome. Another leading gene therapy company, bluebird bio (BLUE, OUTPERFORM), is developing a gene-modified autologous HSC treatment for adrenoleukodystrophy, a peroxisomal storage disorder.

Our view is that there are a range of diseases potentially treated by Fate's ability to improve SCTs that are not commercially viable for other companies to pursue due to small size (the development costs and risks of a new gene therapy would not be recouped) or the pathophysiology of the disease (too-rapid progression to allow for adequate time for a gene therapy to counter the disease's effects). As Fate is improving on an extant technology, the development costs and risks are lessened, and of course Fate's technology is not specific to one disease, allowing it potentially to be used on multiple ultra-rare diseases

Market

In the USA, about 1200 cord blood transplants are performed, with the vast majority occurring in patients with hematological cancers. Rare diseases such as Hunter's, Hurler's, the Sanfilippo diseases and metachromatic leukodystrophy account for over 30 cord blood transplants per year (Source: U.S. Transplant Data by Disease Report, U.S. Health Resources and Services Administration).

In Europe, based on published results of bone marrow transplant centers, there are about 14,500 allogeneic transplants performed.
http://www.ebmt.org/Contents/Research/TransplantActivitySurvey/Results/Documents/Survey_2011_slides_for_website.pdf
Of these, about 13,500 were for hematologic malignancies, and about 500 were for immune disorders (363) and inherited metabolic disorders (146), i.e., rare diseases. Six percent (831) of transplants were with cord blood. We adjust based on population numbers in Japan for ROW market opportunities in our model.

Given that ProHema could significantly increase the effective availability (by 50% as referred to above) and efficacy of cord blood transplants, we assume that there will be continued significant growth in cord blood use with the approval of ProHema, and that ProHema-modified cord blood will become the standard for cord blood transplantation.

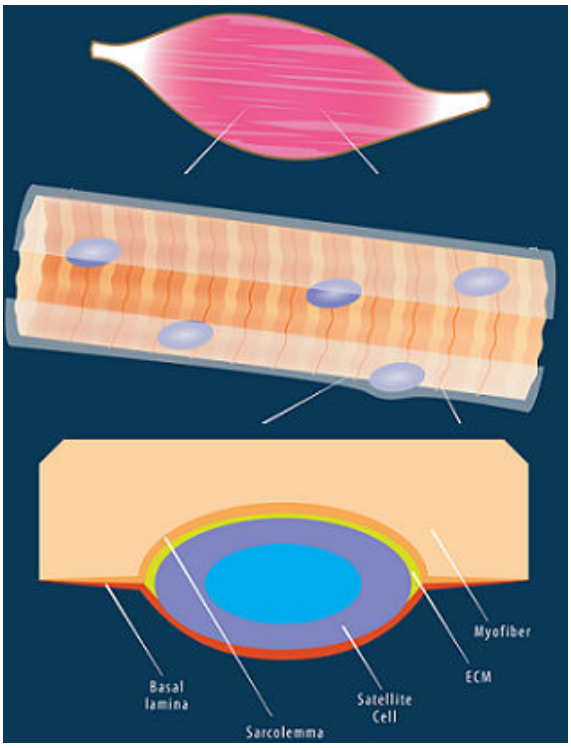
Our worldwide ProHema hematology/oncology estimates in 2019 (our valuation year) are for $176M in sales, with pricing of $50,000 per procedure (about 3500 world-wide cord blood transplants with ProHema). If ProHema administration leads to better overall survival in addition to better engraftment in hematological cancers, we would anticipate on the order of oncology therapies with proven survival benefits ($80,000+).

Because of the dire, immediate need for HSCT in treating rare diseases and the curative potential, we anticipate a much higher price for a specialized CNS-homing ProHema formulation, of $500,000 and a rapid adoption in the market. Converting one-third of estimated current patients who receive cord blood HSCT for inherited, rare diseases, yields our estimated $151M in LSD revenues in our valuation year (~250 patients).

Satellite Stem Cells

Satellite stem cells (SSC) are progenitor cells involved in skeletal muscle regeneration. The main function of SSCs are to proliferate (self renew) and differentiate into muscle tissue precursor cells after exercise or injury. They are called "satellite" cells due to their peripheral location between the basal lamina and the muscle fiber sarcolemma. However, unlike true stem cells, SSCs lack pluripotency and under normal conditions produce cells only of myogenic lineage.

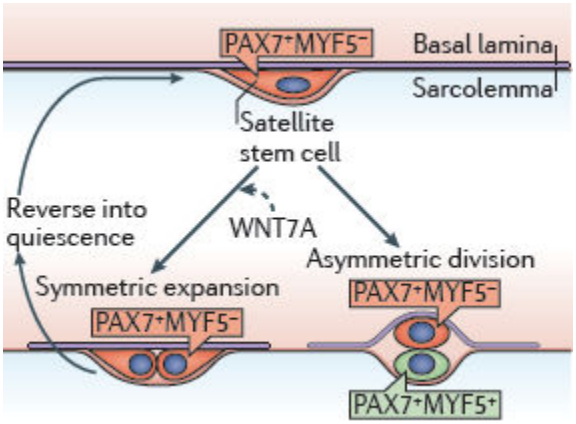
Figure 21: Satellite Cell Localization



Source: LifeMap Sciences, Inc.

Under normal physiological conditions, SSCs are in a state of inactivity (quiescence). SSCs only proliferate and differentiate when they are activated in response to certain external signaling factors triggered by exercise or trauma to the underlying muscle fiber. These factors include two families of transcription factors: myogenic regulatory factors (MRFs), including muscle-specific regulatory factor 4 (MRF4, also known as MYF6), myogenic factor 5 (MYF5), myoblast determination protein (MYOD), and myogenin; and the paired box family (particularly PAX 3/7), which regulates the expression of MRFs. Two signaling pathways that have been found to regulate SSC activity includes Notch, the inhibition of which leads quiescent SSCs to differentiate, and Wnt, which is thought to promote proliferation. In a paper ("Satellite cells, the engines of muscle repair", *Nature Reviews Molecular Cell Biology*) co-authored by a founder of Verio Therapeutics (which Fate acquired in 2010), Wnt protein 7a (Wnt7a) was found to stimulate the expansion of SSCs and muscular hypertrophy. Wnt7a was shown to signal through the Frizzled 7 (FZD7) receptor, which is predominantly expressed in skeletal muscle. Overexpression of Wnt7a enhanced muscle regeneration and increased SSC numbers, while muscle lacking Wnt7a exhibited a decrease in the quantity of SSCs following regeneration.

Figure 22: Self-renewal of SSCs



Source: "Satellite cells, the engines of muscle repair", Nature Reviews Molecular Cell Biology

Wnt7a and muscular dystrophies

Certain degenerative muscle diseases, like muscular dystrophies, creates a cycle of muscle damage and repair that can lead to the depletion of satellite cells and a consequent reduction of a muscle's regenerative capacity. The most common muscular dystrophies are Duchenne and Becker muscular dystrophies (DBMD), which results from a mutation in the DMD gene that encodes dystrophin. Dystrophin is a protein that protects muscle cells by acting as a shock absorber. Partial (Becker) or complete loss (Duchenne) of dystrophin function is characterized by progressive weakening of the skeletal and cardiac muscles. Most children affected by Duchenne will have delayed motor skills and are typically diagnosed between age three and five, with most wheelchair-bound by adolescence. Patients with Becker have a slower progression of disease, with most patients losing the ability to walk after the age of 20. Although advances in antibiotics, respiratory support and cardiac care have increased the life expectancy of DBMD patients, the chronic quality of life impact remains. Since it is an X-linked disorder, patients who suffer from DMBD are almost exclusively male.

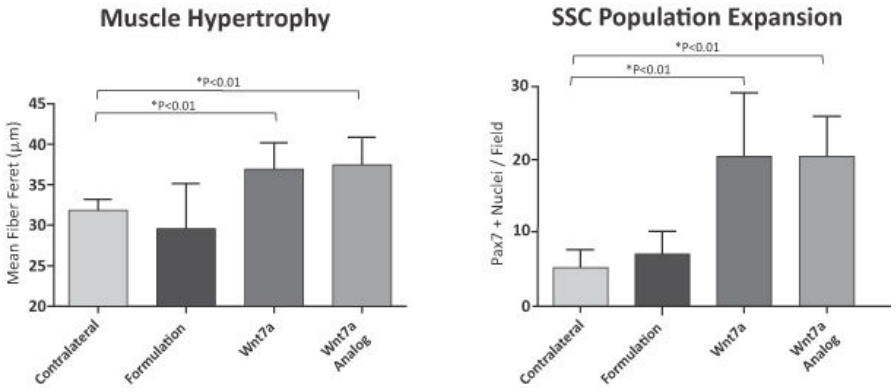
Using their understanding of stem cell modulation, Fate is developing treatments based around Wnt7a analogs that will stimulate SSCs *in vivo* in order to support muscle regeneration. Wnt proteins have not previously been developed as therapeutics due to their molecular characteristics which provide barriers to functional specificity and administration, as well as manufacturing challenges like formulation and scaled production. Fate's Wnt7a analogs are designed to have a higher degree of specificity and a wider therapeutic range on administration, and the compositions are also designed to enable for scaled recombinant manufacturing and protein purification.

Outside of DBMD, there are many other rarer forms of muscular dystrophy that Wnt7a has potential in, including facioscapulohumeral, limb-girdle and myotonic dystrophies. Wnt7a's ability to drive muscle hypertrophy could also have therapeutic benefit for neuromuscular degenerative conditions like sarcopenia.

Proof of Concept

In preclinical studies conducted by Fate in wild type and mdx (point mutation within dystrophin gene) mouse models, a single intramuscular injection of a low µg amount of either Wnt7a or a Wnt7a analog resulted in a dose-dependent and statistically significant hypertrophic effect and increase in the SSC population. Results were assessed at three weeks and compared to muscle injected with formulation (control) and untreated muscle on the opposite side of the body (contralateral control). Compared to contralateral control, muscle treated with either Wnt7a or Wnt7a analog had an approximate 20% increase in muscle fiber cross-sectional diameter (minimal Feret's diameter) and a 3x increase in the number of PAX7 positive cell nuclei, a marker for SSCs.

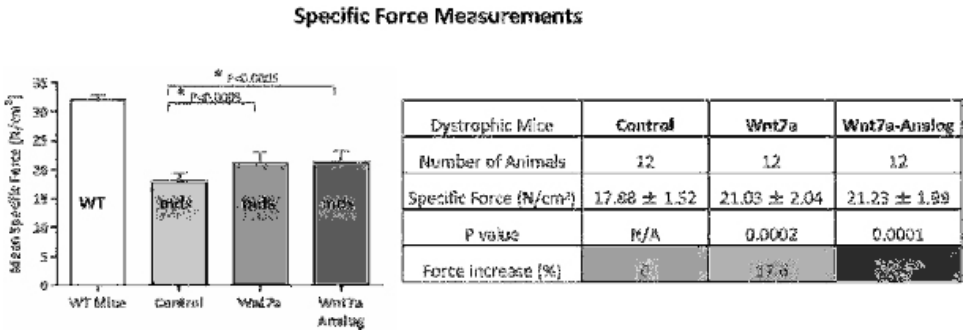
Figure 23: Effect of Wnt7a on Hypertrophy and SSC Population in Mice



Source: Fate Therapeutics, Inc. S-1

This hypertrophic effect was reflected in improved muscle strength as measured by specific force, defined as the normalization of force per cross-sectional area of muscle. The muscle injected with either Wnt7a or its analog generated an approximate 18% increase in specific force compared to muscle injected with formulation control. Although results were still far below the specific force that normal healthy muscle can generate, additional injections or greater doses may reduce the gap.

Figure 24: Effect of Wnt7a on Muscle Strength in Mice

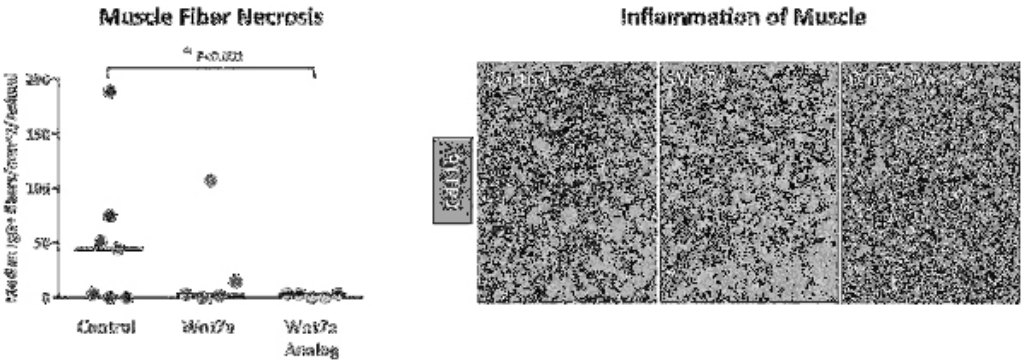


Dystrophic Mice	Control	Wnt7a	Wnt7a-Analog
Number of Animals	12	12	12
Specific Force (N/cm²)	17.88 ± 1.52	21.03 ± 2.04	21.23 ± 1.89
P value	N/A	0.0002	0.0001
Force increase (%)			

Source: Fate Therapeutics, Inc. S-1

Administration of either Wnt7a or Wnt7a analog also reduced the levels of inflammation and muscle damage associated with muscular dystrophy. Levels of inflammation within the muscles of the mice were determined by positive staining of CD11b (also known as integrin αM), a cellular biomarker of inflammation, while levels of muscle fiber necrosis were determined by the mean IgG-positive fibers per unit area of muscle.

Figure 25: Effect of Wnt7a on Muscle Damage and Inflammation in Mice



Source: Fate Therapeutics, Inc. S-1

Competition

Treatment for DMD has thus far been limited to the use of glucocorticoids, which can temporarily (~ two years) preserve walking ability at the expense of a variety of steroid-related side effects. An emerging modality that can potentially exceed the duration and efficacy of glucocorticoids and be safer involves correcting the specific genetic defect underlying DMD. These molecular-based therapies use exon-skipping or nonsense codon suppression to modify the DMD gene. About 60% of DMD cases are due to the deletions of entire exons, which are the regions within the gene that hold instructions on producing dystrophin. The absence of these exons from the exon chain causes the genetic code of DMD to not be translated properly. Exon-skipping is based around the use of splice-switching oligomers that at the pre-mRNA level will omit one or more exons from the genetic code of DMD. By omitting certain exons, the code becomes translatable despite being incomplete. As a result, exon-skipping drugs have the potential to restore partial dystrophin expression, advancing DMD patients into Becker status. Exon-skipping treatments in development include drisapersen from ProSensa (RNA, NEUTRAL) and eteplirsen (AVI4658) from Sarepta (SRPT, OUTPERFORM). Recently drisapersen failed the 6MWD endpoint in its Phase III trial, raising doubts about the future of the compound. Drisapersen, like eteplirsen, excises exon-51.

Nonsense codon suppression involves suppressing premature stop codon mutations (also known as nonsense codons) within the DMD gene. Stop codons terminate translation of the mRNA, so nonsense codons result in the production of a truncated non-functional protein. Nonsense codons are thought to be responsible for about 15% of DMD cases. Ataluren, which PTC Therapeutics (PTCT, OUTPERFORM) is developing for nonsense mutation genetic disorders, is a promising therapy in development, with recently released Phase II data showing a clinically meaningful improvement in the 6 minute walk distance (6MWD) test with ataluren over placebo.

From analyzing preclinical data, the hypertrophic effect of Fate's Wnt7a analogs compares favorably with the exon-skipping approach. A study of eteplirsen in mdx mouse models showed that low doses (5 mg/kg/week) of eteplirsen produced an 8.4% increase in specific force at 20 weeks over control and that high doses (50 mg/kg/week) produced a 7.2% increase in specific force at 50 weeks over control. In comparison, Fate's Wnt7a analog produced an 18.7% increase in specific force in mdx mouse models over control.

Another emerging molecular-based therapy involves up-regulation of the dystrophin analog utrophin. Utrophin is meant to act as a surrogate gene, since it shares 80% of dystrophin's genetic sequence and has been shown to partially restore dystrophin function in animal models. Since DMD patients do express some levels of utrophin (as opposed to none for dystrophin), utrophin-based therapies are less likely to trigger an immune response compared to dystrophin replacement. Summit (UK: SUMM, not covered) is currently developing the utrophin up-regulator SMT-C1100 solo after former partner BioMarin (BMRN, NEUTRAL) withdrew from a collaboration for the drug following disappointing Phase I results. Separately, Tivorsan (private) is developing therapy (TVN102) using recombinant biglycan, a glycoprotein that helps regulate expression of utrophin. Although clinical data for it is not yet available, TVN102 is likely to be less efficacious than SMT-C1100 was in DMD patients since biglycan mainly stabilizes utrophin in muscles that have residual utrophin activity, while SMT-C1100 is designed to promote transcription of utrophin in all muscle cells.

A major benefit Fate's Wnt7a analogs have over molecular-based therapies is that Wnt7a can treat dystrophies irrespective of the underlying genetic mutation. Ataluren is only effective in the subset of DMD patients that are due to nonsense codons, while exon-51 skipping would be effective in only 13% of DMD patients. Fate's Wnt7a analogs could also be used as a complementary therapy with other DMD treatments since its promotion of SSCs can work in parallel with therapies designed to increase dystrophin.

Figure 26: Comparing Wnt7a to DMD Treatments in Clinic

Company	Product	Description	Subset	Status
Fate	Wnt7a analog	Stimulates SSCs	Universal	Preclinical
	Drisapersen	Exon 51 skipping	13%	Failed Phase III primary endpoint, subset analysis continuing.
ProSensa /GSK	PRO 044	Exon 44 skipping	6%	Phase I/II
	PRO 045	Exon 45 skipping	8%	Phase I/II
	PRO 053	Exon 53 skipping	6%	Phase I/II
PTC Therapeutics	Ataluren	Nonsense codon suppressor	15%	Phase III ongoing
Sarepta	Eteplirsen	Exon 51 skipping	13%	Completed Phase II
Summit	SMT C1100	Utrophin modulator	Universal	Completed Phase I
Tivorsan	TVN 102	Recombinant human biglycan	Unknown	Phase I

Source: Wedbush Securities, Inc.

Market

We estimate that Fate's Wnt7a analog could be approved in 2018. We believe Fate, as with ProHema, will seek to market its SSC product by itself since the small patient population could be targeted with a minimal sales force. It is estimated that DMD occurs in one out of 3,500 live births, with approximately 25,000 males living with the disease in the US and Europe. Given a $300,000 price, we estimate that the Wnt7a analogs could generate sales in excess of $50 million in 2019, the beginning of its ramp and our valuation year.

iPSC and IP

Fate is also developing induced pluripotent stem cell (iPSC) related technology for the development of their stem cell modulators. iPSCs are pluripotent stem cells that are generated by inducing the expression of certain genes in mature cells. Fate's iPSC technology, which is built upon discoveries made by two of its scientific cofounders, enables more efficient cellular reprogramming, increased high-throughput generation and large-scale culture expansion. Fate had previously entered into a collaboration agreement, which ended in September, with Becton, Dickinson and Company (BDX, Not Covered) for certain iPSC tools and technologies for use in drug discovery and development. The first commercial product to come out of the collaboration is BD SMC4, a cocktail of small molecules for improving cellular reprogramming which BDX launched in June 2012. Fate is still eligible to receive up to $1.5 million in milestones and $4 million in royalties as part of the collaboration, although the company does not expect that to be likely.

Fate has an exclusive worldwide license with the Children's Medical Center Corporation for rights to therapeutic compositions of modulated HSCs and methods for promoting reconstitution of the hematopoietic system using modulators of the prostaglandin pathway. This includes the issued patent US Patent No. 8,168,428 covering the use of HSC modulators to promote HSC engraftment. The Children's Medical Center is eligible for up to $5 million in milestones per product developed and a low- to mid-single digit royalty on any sales. Fate also has an exclusive worldwide right from Stanford to the use of Wnt7a analogs and inherited rights (through its acquisition of Verio) to methods to promote muscle regeneration from the Ottawa Hospital Research Institute (OHRI). Stanford is eligible for up to $900,000 in milestones and a low- to single-digit royalty on sales of any Wnt7a analogs commercialized, while OHRI is eligible for up to CDN$1.4 million in milestones and a low digit royalty.

Covered Companies Mentioned

COMPANY	TICKER	RATING	PRICE	PRICE TARGET
BioMarin	BMRN	NEUTRAL	$66.24	$63
Prosensa	RNA	NEUTRAL	$4.32	$7
Sarepta	SRPT	OUTPERFORM	$42.78	$60
bluebirdbio	BLUE	OUTPERFORM	$22.95	$40

Financial Model

Ticker: (FATE:Nasdaq)
Fate Therapeutics, Inc

Wedbush PacGrow Life Sciences
David M. Nierengarten, Ph.D.
415-274-6862

	2012	1H:13	Q3	Q4	2013E	2014E	2015E	2016E	2017E	2018E	2019E
Revenues:											
US Product Sales	$0	$0	$0	$0	$0	$0	$0	$0	$0	$43,180	$181,224
ex-US Product Sales	$0	0	0	0	0	0	0	0	0	27,896	198,672
Grant Revenue	$2,670	762	0	0	762	0	0	0	0	0	0
Total Revenues	**2,670**	**762**	**0**	**0**	**762**	**0**	**0**	**0**	**0**	**71,076**	**379,896**
Cost and Expenses:											
Cost of Sales	0	0	0	0	0	0	0	0	0	7,108	37,990
R&D	11,999	5,598	2,855	2,998	11,451	17,332	19,973	23,273	26,279	28,445	30,790
SG&A	4,228	2,789	1,422	1,451	5,662	6,099	6,553	6,835	7,291	37,785	94,964
Total Operating Expenses	**16,227**	**8,387**	**4,277**	**4,449**	**17,113**	**23,431**	**26,526**	**30,109**	**33,570**	**73,338**	**163,743**
Operating Income (Loss)	(13,557)	(7,625)	(4,277)	(4,449)	(16,351)	(23,431)	(26,526)	(30,109)	(33,570)	(2,261)	216,153
Net Interest Income (Expense)	(486)	(187)	42	111	(34)	1,376	1,673	1,990	1,013	1,072	3,180
Other non-operating Income (Expense)	(196)	(1,270)	0	0	0	0	0	0	0	0	0
Income Before Income Taxes	(14,239)	(9,082)	(4,235)	(4,338)	(16,385)	(22,055)	(24,853)	(28,118)	(32,557)	(1,189)	219,332
Provision for Income Taxes	0	0	0	0	0	0	0	0	0	1,190	11,625
Net Income (Loss)	(14,239)	(9,082)	(4,235)	(4,338)	(16,385)	(22,055)	(24,853)	(28,118)	(32,557)	(2,379)	207,708
Non-GAAP EPS	**(13.21)**	**(0.74)**	**(0.21)**	**(0.21)**	**(0.88)**	**(0.98)**	**(1.03)**	**(1.16)**	**(1.28)**	**(0.12)**	**7.81**
GAAP EPS	**(13.06)**	**(7.41)**	**(0.33)**	**(0.21)**	**(7.94)**	**(1.03)**	**(1.05)**	**(1.14)**	**(1.30)**	**(0.11)**	**7.84**
Total Shares Outstanding	1,090	12,490	20,627	20,627	20,627	23,202	24,742	24,762	26,027	26,508	26,508
Cash Burn	(13,274)	0	0	0	(15,753)	(24,971)	(26,271)	(29,814)	(33,286)	(6,140)	200,733
Cash Balance	9,087	21,313	59,245	54,907	54,907	64,216	74,869	47,045	50,024	43,411	234,947

Management

Figure 27: Fate Management

Title	Biography
President and Chief Executive Officer: Christian Weyer, M.D., M.A.S	Dr. Weyer joined Fate from Amylin, where he served as Senior Vice President of R&D until Amylin's acquisition by Bristol-Myers Squibb in Aug. 2012. Prior to that, he was a researcher at the NIH's National Institute of Diabetes and Digestive and Kidney Diseases. Dr. Weyer earned his M.D. from the University of Dusseldorf and has a postdoctoral Masters degree in clinical research from the University of California, San Diego.
Chief Medical Officer: Pratik Multani, M.D., M.S.	Dr. Multani joined Fate from Kalypsys, where he served as Vice President of translational medicine. Prior to that, he served as Senior Vice President of clinical development and CMO at Kanisa Pharmaceuticals. Prior to that, he served as Vice President of clinical development at Salmedix. Prior to that, he served as senior director of medical research at Biogen-Idec. Dr. Multani earned his undergraduate degree from Yale University and his M.D. from Harvard Medical School.
Chief Technology Officer: Daniel Shoemaker, Ph.D	Dr. Shoemaker joined Fate from ICxBiosystems, where he served as Chief Scientific Officer. Prior to that, he served as Chief Scientific Officer of GHC Technologies. Prior to that, he served in several roles at Merck Research Laboratories. Dr. Shoemaker earned his Ph.D. in biochemistry from Stanford University and his B.S. in biochemistry from the University of California, Santa Barbara.
Chief Financial Officer: J. Scott Wolchko	Mr. Wolchko joined Fate from Bocada Inc., where he served as CFO. He previously worked as an investment banker with Morgan Stanley in the Health Care Group. Mr. Wolchko earned his M.S. in biochemical engineering from the University of Virginia and his B.S. in biomedical engineering from the University of Vermont.

Source: Company data, Wedbush Securities, Inc.

Analyst Biography

David Nierengarten, Ph.D.

David is an Analyst covering stocks in the Biotechnology/Biopharmaceuticals/BioDefense sector. His prior sell-side research experience at Robert W. Baird & Co. covered biotechnology companies of all market capitalizations, with a focus on oncology and rare diseases.

David received his B.S. (Biochemistry) from the University of Wisconsin-Madison and Ph.D. (Molecular and Cell Biology) from the University of California-Berkeley.

David's Edge: David's early stage venture capital investing experience gives him a balanced perspective on developmental-stage biotechnology companies and their ultimate risk/reward potential. His experience on the other side of that equation in a clinical-stage, venture backed biotechnology company provides him with insights into corporate operations. The combination of experiences creates a focus on value creation in this event-driven space.

Analyst Certification
I, David M. Nierengarten, Ph.D., Gregory R. Wade, Ph.D., Christopher N. Marai, Ph.D., certify that the views expressed in this report accurately reflect my personal opinion and that I have not and will not, directly or indirectly, receive compensation or other payments in connection with my specific recommendations or views contained in this report.

Disclosure information regarding historical ratings and price targets is available at http://www.wedbush.com/ResearchDisclosure/DisclosureQ313.pdf

Investment Rating System:
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Neutral: Expect the total return of the stock to perform in-line with the median total return of the analyst's (or the analyst's team) coverage universe over the next 6-12 months.
Underperform: Expect the total return of the stock to underperform relative to the median total return of the analyst's (or the analyst's team) coverage universe over the next 6-12 months.

The Investment Ratings are based on the expected performance of a stock (based on anticipated total return to price target) relative to the other stocks in the analyst's coverage universe (or the analyst's team coverage).*

Rating Distribution (as of September 30, 2013)	**Investment Banking Relationships (as of September 30, 2013)**
Outperform:55% Neutral: 41% Underperform: 4%	Outperform:14% Neutral: 2% Underperform: 0%

The Distribution of Ratings is required by FINRA rules; however, WS' stock ratings of Outperform, Neutral, and Underperform most closely conform to Buy, Hold, and Sell, respectively. Please note, however, the definitions are not the same as WS' stock ratings are on a relative basis.

The analysts responsible for preparing research reports do not receive compensation based on specific investment banking activity. The analysts receive compensation that is based upon various factors including WS' total revenues, a portion of which are generated by WS' investment banking activities.

Wedbush Equity Research Disclosures as of October 27, 2013

Company	Disclosure
Fate Therapeutics	1,3,5,7
BioMarin Pharmaceuticals	1
bluebird bio	1,3,5
Prosensa Holding N.V.	1,3,5
Sarepta Therapeutics	1,3,4,5

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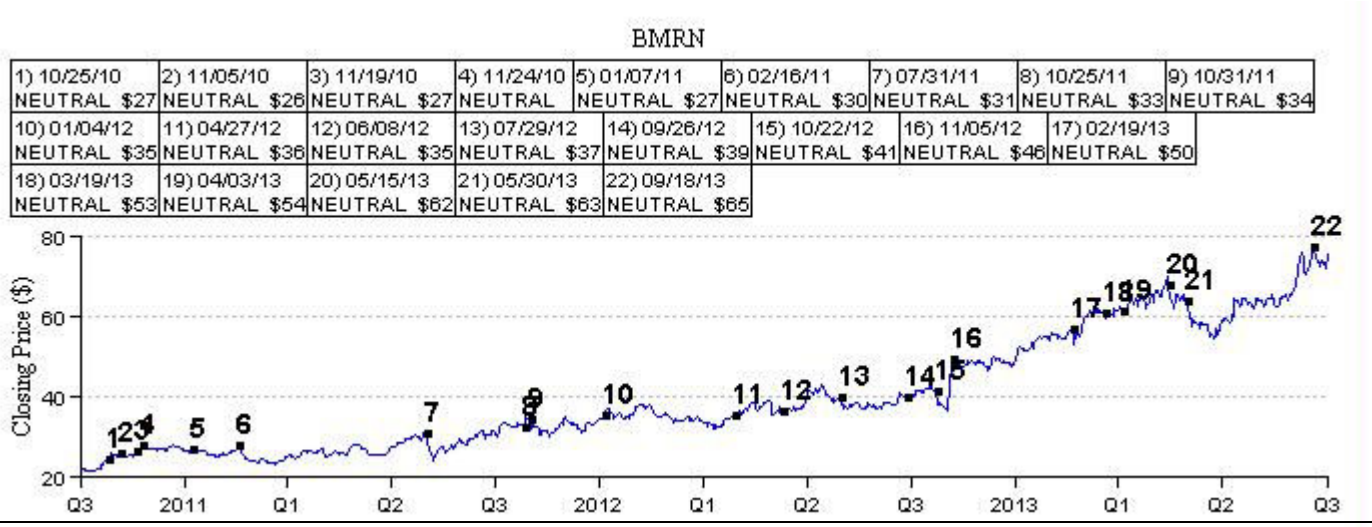
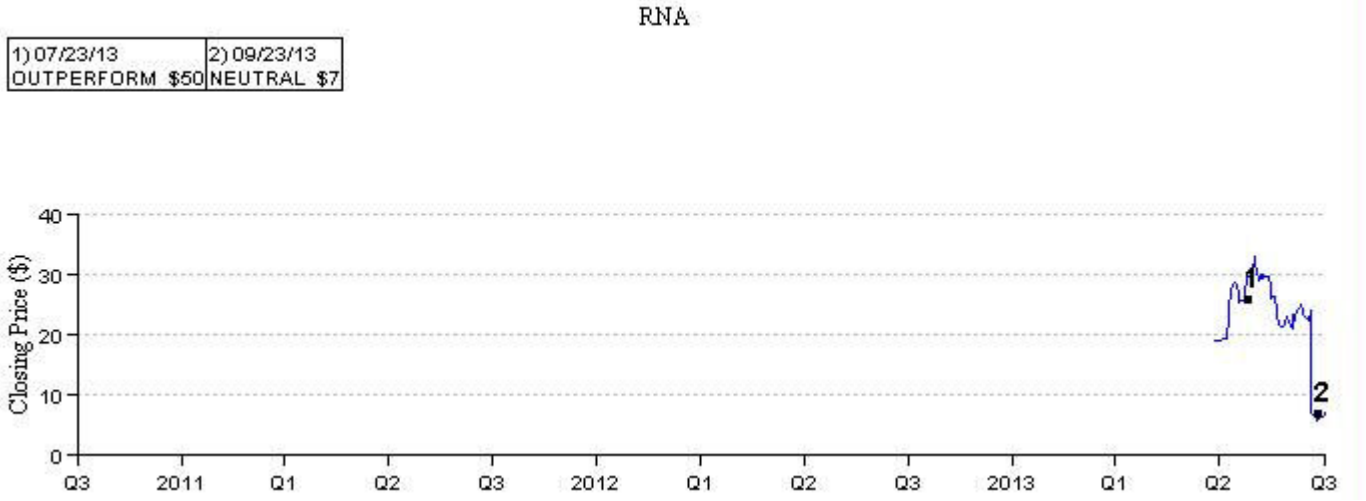
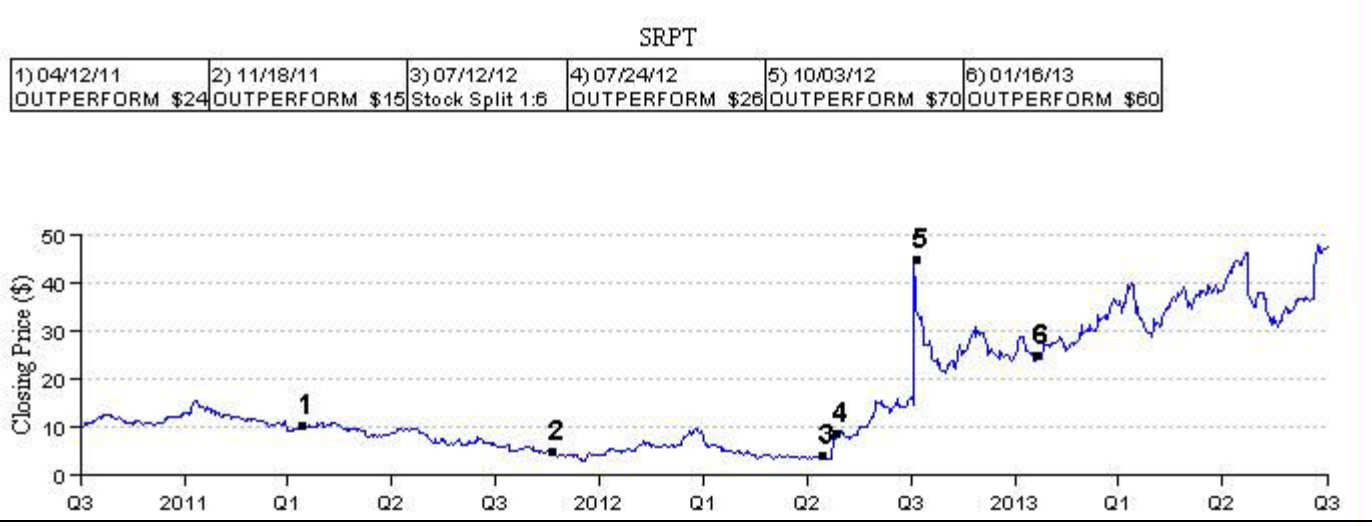
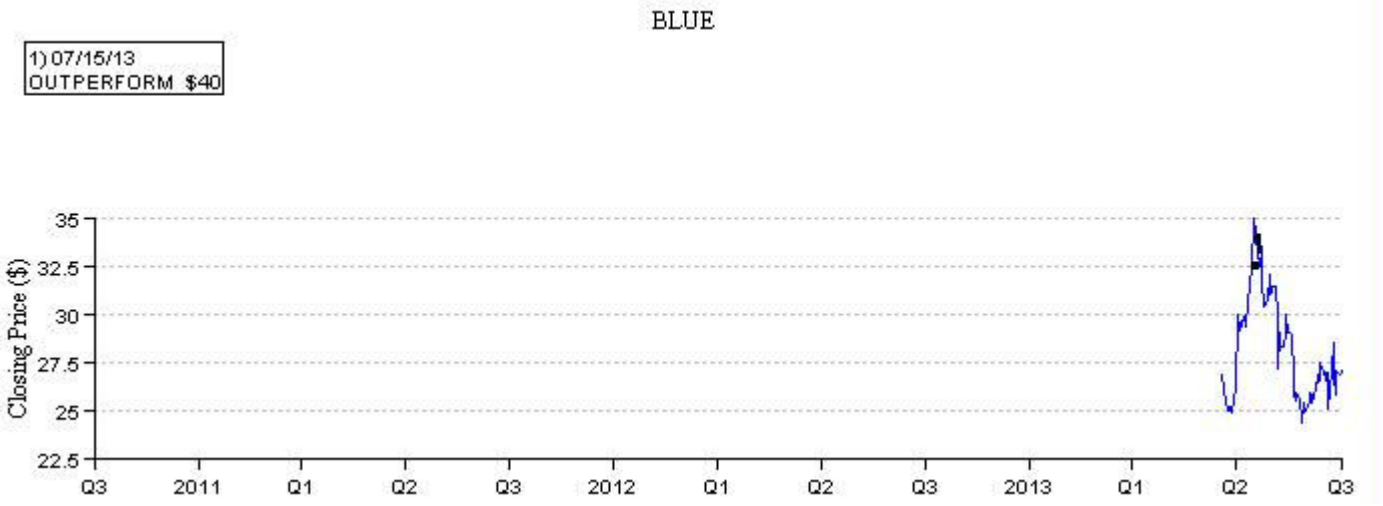
Wedbush disclosure price charts are updated within the first fifteen days of each new calendar quarter per FINRA regulations. Price charts for companies initiated upon in the current quarter, and rating and target price changes occurring in the current quarter, will not be displayed until the following quarter. Additional information on recommended securities is available on request.









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