Initiation
Overweight

PTCT, PTCT US
Price: $16.94

Price Target: $20.00

PTC Therapeutics

Ataluren Has Blockbuster Potential in DMD and CF; Initiate at OW

We are initiating coverage of PTC Therapeutics (PTCT) with an Overweight rating and $20 Dec 2014 PT. PTC is focused on therapies for orphan genetic diseases with the most advanced compound being ataluren, currently in phase 3 development for Duchenne muscular dystrophy (DMD; data 1H15) and cystic fibrosis (CF; beginning second study 1H14). Notably, these are two "hot" orphan markets that each offers a robust opportunity. Of note, other companies are also developing agents for DMD and CF, but ataluren has no direct competitors given it specifically targets patients with nonsense mutations. We believe that insights gained in prior ataluren clinical studies maximize the probability for success in phase 3. While ataluren, which is unpartnered, has potential in both indications we conservatively include revenues only for DMD with peak WW sales of $700M, which could easily double should CF also be successful. Of note, ataluren could receive conditional approval in Europe for DMD in Nov '13, but we believe this is a lower probability and it is not assumed in our model. Given a high probability of success for ataluren in DMD, good optionality in CF, plus a free call option on EU approval in DMD in 4Q13, we're initiating coverage with an Overweight rating.

- **Key value driver is ataluren in nmDMD.** Ataluren failed in a prior phase 2b study due to an inverse dose response and enrollment criteria that led to wide variability in the six-minute walk distance (6MWD). Indeed, a post-hoc subgroup analysis with more restrictive enrollment criteria resulted in a positive outcome (6MWD: 49.9m relative to placebo; p = 0.0096). Importantly, applying this enrollment criterion in the ongoing phase 3 trial and a deeper understanding of the dose response maximize the probability of success, in our view. Additionally, physician feedback has been positive with a bar of a 30m benefit on 6MWD, which we believe is achievable given the phase 2b analysis.
- **Ataluren in nmCF provides meaningful optionality.** Ataluren failed in a prior phase 2/3 study driven by background use of inhaled antibiotics, which interfered with ataluren activity. A pre-specified subgroup analysis indicated a significant 6.7% mean relative improvement in FEV1 (lung function) compared with placebo in patients not receiving inhaled antibiotics. These patients will be excluded from the second phase 3 study expected to begin in 1H14, improving the chances for success, in our view.
- **Overweight rating; $20 Dec 2014 PT.** Our NPV analysis conservatively includes WW sales of ataluren in DMD only (70% probability of success and a 10% discount rate). Taken together with net cash, we derive a YE14 $20/shr PT.

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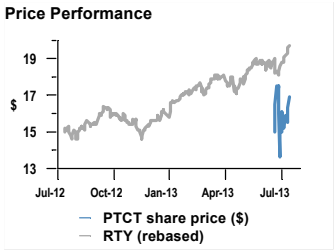
Price Performance



	YTD	1m	3m	12m
Abs	12.9%	12.9%	12.9%	12.9%
Rel	-9.1%	6.3%	3.0%	-18.4%

PTC Therapeutics (PTCT;PTCT US)

FYE Dec	2011A	2012A	2013E	2014E	2015E
EPS Reported ($)					
Q1 (Mar)	-	-	(1.51)A	-	-
Q2 (Jun)	-	-	(0.77)	-	-
Q3 (Sep)	-	-	(0.66)	-	-
Q4 (Dec)	-	-	(0.77)	-	-
FY	5.39	7.75	(3.54)	(2.75)	(3.17)

Source: Company data, Bloomberg, J.P. Morgan estimates.

Company Data	
Price ($)	16.94
Date Of Price	12 Jul 13
52-week Range ($)	17.92-13.04
Market Cap ($ mn)	460.40
Fiscal Year End	Dec
Shares O/S (mn)	27
Price Target ($)	20.00
Price Target End Date	30-Dec-14

See page 30 for analyst certification and important disclosures.

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Table of Contents

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Investment Thesis

PTC Therapeutics (PTCT)

Overweight

Ataluren is the key driver

PTC is focused on the development and commercialization of small molecules for orphan and ultra orphan diseases that target post-transcriptional control processes. Indeed, PTC has developed an integrated technology platform to identify small molecules that modulate downstream processing of mRNA and protein synthesis, which can address a broad range of known targets and diseases. Ataluren is the most advanced compound with the remaining pipeline in the discovery or preclinical development stages. Ataluren is currently in phase 3 development for Duchenne muscular dystrophy (DMD) and cystic fibrosis (CF), two "hot" orphan markets. Of note, other companies are also developing therapies for DMD and CF, but ataluren has no direct competitors given it specifically targets patients with nonsense mutations (aka premature stop codons). While we believe that ataluren has potential in both indications we conservatively include revenues only for DMD with peak WW sales of $700M. That said, the addition of revenues for CF would easily double WW ataluren revenues to $1.4B.

Probability of success in DMD is maximized, in our view

In a phase 2b trial, ataluren failed to meet the primary endpoint of 6MWD. However, further analysis revealed that the failure was due to an inverse dose response in the high-dose arm and wide variability in changes in 6MWD as patients not experiencing a decline in 6MWD were included in the study. Indeed, a post-hoc subgroup analysis excluding these patients resulted in a significant improvement in 6MWD of 49.9m for ataluren compared with placebo ($p = 0.0096$). These findings formed the basis for the enrollment criteria used in the ongoing phase 3 trial ensuring that only patients in the decline phase of the disease (i.e., 6MWD decreasing) are selected for the study. We believe this approach maximizes the probability of success in phase 3. Indeed, physicians agree ataluren is active in DMD, and they are excited by the potential for a new treatment option. Additionally, physicians noted any delay in deterioration in physical function is meaningful, but 30m is a reasonable bar for efficacy on the 6MWD test. Based on the phase 2b analysis, we believe that ataluren can achieve this level of improvement. Data from the ongoing phase 3 trial are expected in mid 2015 with an expected launch in 2H16. Of note, ataluren could receive conditional approval in Europe for nmDMD in November, driving significant upside, but we believe this is unlikely given EMA feedback

CF opportunity provides optionality

Ataluren failed to meet the primary endpoint in a prior phase 2/3 trial in CF. However, a pre-specified subgroup analysis revealed this was driven by a subgroup of patients that received inhaled antibiotics. Specifically, patients not receiving inhaled antibiotics in the phase 3 study had a statistically significant 6.7% relative FEV1 difference for ataluren versus placebo ($p=0.013$). Of note, PTC is currently in discussions with the FDA and EMEA regarding the design of a second phase 3. Importantly, PTC plans to exclude patients on inhaled antibiotics, which in our view will provided the best opportunity for success. This second phase 3 trial is expected to begin in 1H14.

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Risks to Rating and Price Target

Clinical risk

Predicting the outcome of late-stage clinical trials is very difficult. Of note, ataluren is currently in a phase 3 trial for DMD, while a phase 3 trial in CF is expected to begin in 1H14. These phase 3 trials may be unsuccessful. As a reminder, in a phase 2b and phase 3 study, ataluren failed to meet the primary endpoint within the pre-specified level of significance in DMD and CF, respectively. As such, ataluren's ability to demonstrate a meaningful benefit in DMD and CF is critical to PTCT shares and a key source of clinical risk.

Regulatory risk

Assuming ataluren is successful in phase 3 trials in DMD and CF, the next step would be regulatory approval. Even if ataluren demonstrates a clinical benefit, there is no assurance that US or EU regulators will approve the drug. As such, ataluren could face difficulties obtaining approval from the FDA or EMA.

Commercial risk

PTC currently has no marketed products. For ataluren, the company intends to market the drug on its own with a specialized sales force in the US and EU for both DMD and CF. Considering the company has no prior experience, this could be challenging. As such, even if ataluren has a compelling profile, it could fail to gain meaningful market share.

Financial risk

Following completion of its initial public offering, PTC has ~$168M in cash on hand. Of note, PTC currently has no source of revenues and ataluren is not expected to launch until 2016. In the meantime, PTC continues investing in its pipeline, most notably phase 3 development of ataluren, and will continue to burn cash. As such, PTC may choose to raise additional capital, which could dilute current shareholders.

Legal risk

PTC relies on patents to protect its business. For ataluren, US and EU patents expire from 2024 to 2027. An inability to defend these patents could substantially limit the commercial opportunities.

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Company Description

PTC is a biopharmaceutical company focused on the development and commercialization of small molecules for orphan and ultra orphan diseases. PTC applies proprietary technologies and an extensive understanding of post-transcriptional control processes to discover and develop drugs. The company currently has no marketed products. The most advanced candidate is ataluren for the treatment of genetic disorders resulting from nonsense mutations, which is currently in phase 3 testing. The remainder of the pipeline is in the discovery or preclinical stages of development.

Background and Pipeline

PTC was founded in 1998 with a focus on the discovery and development of small molecules that target post-transcriptional control processes. Transcription is the cellular processes of converting DNA into mRNA, which is then processed and translated into proteins. Indeed, PTC has developed several integrated technology platforms to identify small molecules that modulate downstream processing of mRNA into proteins. Importantly, this technology can address a broad range of known targets as well as those that have not previously been drugable. Through this approach PTC can develop drugs for a wide variety of diseases such as genetic disorders, cancer, musculoskeletal disorders, inflammation, metabolic disorders, cardiovascular conditions and neurological disorders.

The proprietary platform consists of three technologies, including GEMS, alternative splicing and nonsense suppression. GEMS identifies molecules that act through the untranslated regions (UTRs) of the mRNA, which play a role in regulating protein production. Alternative splicing is a technology that enables the identification of small molecule modifiers of pre-mRNA splicing, which can correct for the altered regulation of alternative splicing that can be the direct cause of many human diseases. Using this technology PTC has identified small molecules that correct splicing of the SMN2 RNA that is believed to play a role in spinal muscular atrophy. Nonsense suppression is a technology to identify molecules that promote read-through of premature stop codons to create full-length, functional proteins. In the absence of read-through, a nonsense mutation results in a short nonfunctional protein. Ataluren is an example of a nonsense suppressor developed by PTC.

PTC currently has no marketed products. The pipeline consists of one drug in late-stage clinical testing with the remainder all in discovery or preclinical development (Table 1). The most advanced is ataluren for the treatment of genetic disorders due to nonsense mutations. Ataluren is currently in phase 3 development for nmDMD and nmCF. Additionally, SMN2 is currently in preclinical testing for the treatment of spinal muscular atrophy utilizing the PTC's alternative splicing technology. Of note, this program is partnered with Roche and the SMA Foundation on this program. Other preclinical programs include PTC596 (chemo-resistant cancers), PTC725 (HCV), Utrophin (DMD) and Serca2a (DMD). Additionally, PTC also has antibacterial (MDR Gram negative bacteria) and virology (HIV latency and Dengue) programs in early development.

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Table 1: PTC Pipeline Overview

Product	Indication	Partner	Preclinical	PhI	PhII	PhIII	Marketed
Ataluren	nmDMD						
Ataluren	nmCF						
SMN2	Spinal Muscular Atrophy	Roche and SMA Foundation					
PTC596 (BMI1)	Chemo-resistant cancers						
PTC725 (NS4b)	HCV						
Utrophin	DMD						
Serca2a	DMD						
Antibacterial	MDR Gram negative bacteria						
Virology	HIV latency, Dengue						

Source: Company reports.

Catalyst and Milestones

Key upcoming catalysts for PTC are related to ataluren. While conditional approval of ataluren in nmDMD could be granted by the EMA in 2H13, we believe this is unlikely given feedback from regulators to date (details below). As such, the most important catalyst is phase 3 data for ataluren in nmDMD is expected in mid-2015. Regulatory filings in the US and EU are expected late 2015/early 2016 with approval in 2H16. For SMN2, Roche and PTC will select a development candidate in 2013 and begin clinical testing in 2014

Table 2: PTC Catalysts

Est Timing	Drug	Indication	Event	Significance
2H13	Ataluren	nmDMD	Potential conditional approval in Europe	Low
1H14	Ataluren	nmCF	Initiate second phase 3 trial	Low
Mid-2014	Ataluren	nmDMD	Complete enrollment in phase 3 trial	Low
2H14	Ataluren	nmCF	Potential conditional approval in Europe	Low
2014	SMN2	SMA	Initiate phase 1 trial	Low
Mid-2015	Ataluren	nmDMD	Phase 3 data	High
Late 2015/Early 2016	Ataluren	nmDMD	NDA and MAA filing	Med
Mid-2016	Ataluren	nmCF	Phase 3 data	High
2H16	Ataluren	nmDMD	US and EU approval	High

Source: Company reports and J.P. Morgan estimates.

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Ataluren for DMD

Ataluren is a small molecule being developed for the treatment of nmDMD. Ataluren is currently in a phase 3 trial with data expected in mid-2015. Of note, in a phase 2b trial ataluren failed to meet the primary endpoint of 6MWD. However, further analysis revealed this was driven by wide variability in 6MWD as patients not experiencing a decline in 6MWD were included in the study. Indeed, a post-hoc subgroup analysis excluding these patients resulted in significant improvement in 6MWD of 49.9m for ataluren relative to placebo ($p = 0.0096$). These findings were used to design the enrollment criteria for the ongoing phase 3 trial and maximize the probability of success, in our view. We anticipate a launch of ataluren in nmDMD in 2H16 with peak sales of $700M.

DMD: A Brief Overview

DMD is a genetic disorder that results in progressive muscle deterioration and weakness (skeletal, smooth and cardiac muscles). The disease is caused by a lack of the protein dystrophin, which plays an important role in muscle function. Specifically, dystrophin functions to support muscle fibers and prevent muscle fiber injury. The dystrophin gene is located on the X chromosome and is inherited in a recessive pattern.[1] This means that only one copy of the mutant gene in males is required to have the disease. As such, DMD is more common in males.

Symptoms of DMD typically appear in children before the age of 6. The absence of dystrophin leads to muscle weakness, which usually first appears in the hips, pelvis, thighs and shoulders. Later the weakness occurs in the arms, legs and trunk with children typically requiring a wheelchair between the ages of 7 and 12. The muscle deterioration continues in the early teen years and begins to impact cardiac muscle. Additionally, during this period deterioration of the diaphragm and other muscles used for breathing begins. Of note, the most common causes of death in DMD patients is cardiac dysfunction and respiratory failure.[2]

Currently there are no therapies available for DMD that treat the underlying cause of the disease. However, advances in supportive care, particularly cardiology and pulmonology have resulted in modest increases in life expectancy (median survival 22 years). For example, ACE inhibitors and beta blockers have been shown to improve muscle strength.[3] Additionally, corticosteroids have been used to slow the progression of the disease.[4] Ventilators are also used to treat respiratory insufficiency.

Competitive Landscape

Several companies are developing therapies that address the underlying cause of DMD, namely by dystrophin production (Table 3). PTC's ataluren specifically addresses DMD caused by a nonsense mutation (13% of DMD patients). However, other companies are developing therapies for different subsets of DMD patients. Indeed, Prosensa/GSK's (covered by JPM analyst James Gordon) drisapersen targets

[1] Monaco et. al. Human Genetics 1987; 75: 221-227
[2] Nigro et. al. International Journal of Cardiology 1990; 26: 271-277
[3] Politano et. al. Acta Myologica 2012; 31: 24-30
[4] Angelini et. al. Acta Myologica 2012; 31: 9-15

exon 51 skipping (13% of DMD patients) and is similarly in phase 3 testing. Sarepta's eteplirsen also targets exon 51 skipping and is currently preparing for phase 2/3 studies. Additionally, Prosensa's PRO-044 targets exon 44 skipping (6% of DMD patients) is currently in a phase 1/2a trial. As such, PTC has no direct competitors in nmDMD.

Table 3: DMD Competitive Landscape

Company	Product	Target	% DMD Population	Stage of development	Results
PTC	Ataluren	Nonsense mutation	13%	Phase 3	Phase 2b (n=174): Mean change in 6MWD vs placebo of 31.3m (p=0.0561) at 48 wks
Prosensa / GSK	Drisapersen	Exon 51 skipping	13%	Phase 3	Phase 2 (DEMAND 2; n=36): Mean change in 6MWD vs. placebo of 35.8m (p=0.051) at 48 wks
Sarepta	Eteplirsen	Exon 51 skipping	13%	Phase 2b	Phase 2b (n=8): Mean change in 6MWD vs baseline of 67.3m (p≤0.001) at 48 wks
Prosensa	PRO-044	Exon 44 skipping	6%	Phase 1/2a	Phase 1/2a: Data expected mid-2013

Source: Company reports.

Ataluren: Mechanism of Action

Ataluren targets genetic diseases that are caused by nonsense mutations. This mutation interferes with the process of translation, where mRNA is used by the ribosome to guide the production of a protein. Under disease conditions, the nonsense mutations results in a premature stoppage of the translation process, resulting in a truncated protein that is not functional.

In nmDMD and nmCF, the nonsense mutation results in the absence of dystrophin and cystic fibrosis transmembrane conductance regulator (CFTR) protein, respectively. Ataluren interacts with the ribosome allowing it to read through the premature stop codon, resulting in a full-length functional protein.[5, 6] Additionally, ataluren does not promote read-through at a normal stop codon due to a group of proteins known as the termination surveillance complex. This complex is located downstream of normal stop codons, but not downstream of premature stop codons. As such, ataluren results in full-length functional dystrophin and CFTR proteins.

A recent publication by McElroy et. al. has created some controversy related to ataluren's ability to read through stop codons.[7] Indeed, the publication concluded that there is no evidence of ataluren activity. However, there are several issues with the analysis, in our view. The analysis specifically focused only on reporter assays. In contrast, PTC's analysis began with reporter assays, but then expanded to nonsense mutation disease models (cultured cells and mice) and patients. PTC's findings were also confirmed independently in both reporter assays and nonsense disease models (animal and cell based). Of note, PTC was not consulted in the preparation of ataluren, constructs, cell lines or protocols used in the McElroy et. al. analysis. Additionally, McElroy et. al. did not repeat any experiments demonstrating ataluren's activity or take into account independently published research supporting

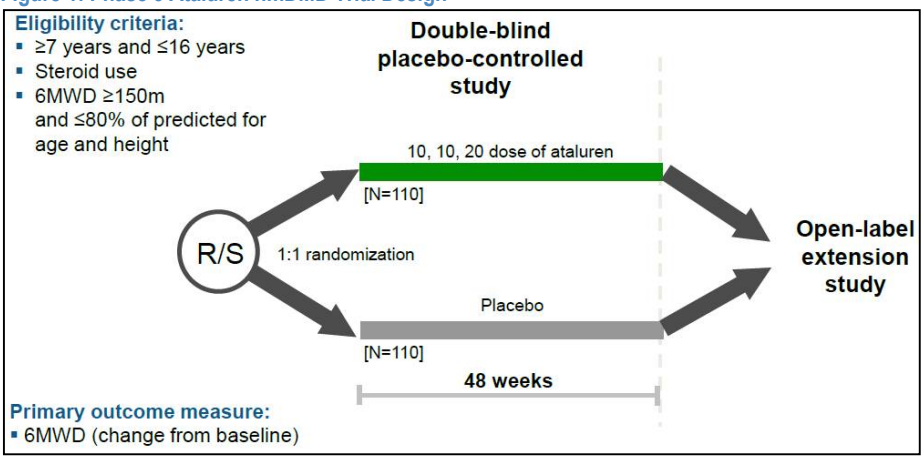
[5] Barton et. al. Neurology 2005; 64 (suppl 1): A176 (#060)
[6] Du et. al. Pediatric Pulmonology 2004; 38 (suppl 27): #195
[7] McElroy et. al. PLOS Biology 2013; 11 (6):e 1001593

ataluren's activity.[8, 9, 10, 11, 12 13] As such, we remain confident in ataluren's mechanism of action.

Phase 3 Program Under Way

A phase 3 trial of ataluren in nmDMD was initiated in April 2013 (Figure 1). The study is targeting enrollment of 220 patients. Patients will be randomized 1:1 to receive either 40 mg/kg of ataluren per day or placebo. Of note, ataluren dosing will consist of 10mg/kg in the morning, 10 mg/kg mid day and 20 mg/kg in the evening (10, 10, 20), which is the same does used in the phase 2b trial. This study is expected to complete enrollment in mid-2014 with data expected in mid-2015.

Figure 1: Phase 3 Ataluren nmDMD Trial Design



Source: Company reports.

Based on findings from the phase 2b trial, a very specific patient population is being enrolled in phase 3 to maximize the probability of success. Key inclusion criteria consist of patients between the ages of 7 and 16 that have used corticosteroids for a minimum of 6 months prior to starting the study. Additionally, at baseline patients must have the ability to walk at least 150m, but no more that 80% of predicted (compared with healthy boys matched for age and height) on 6MWD.

The primary endpoint of the study is the mean change from baseline in 6MWD over 48 weeks. Of note, 6MWD has been well established as an endpoint in DMD.[14] Secondary endpoints include physical function, patient or parent reported activities of daily living and disease symptoms, quality of life, safety, ataluren blood levels and compliance. Timed function tests include the time to climb four stairs, descend four stairs and run/walk for 10m. Additionally, The North Star Ambulatory Assessment (NSAA), specifically designed for DMD, will also be used for the first time to asses

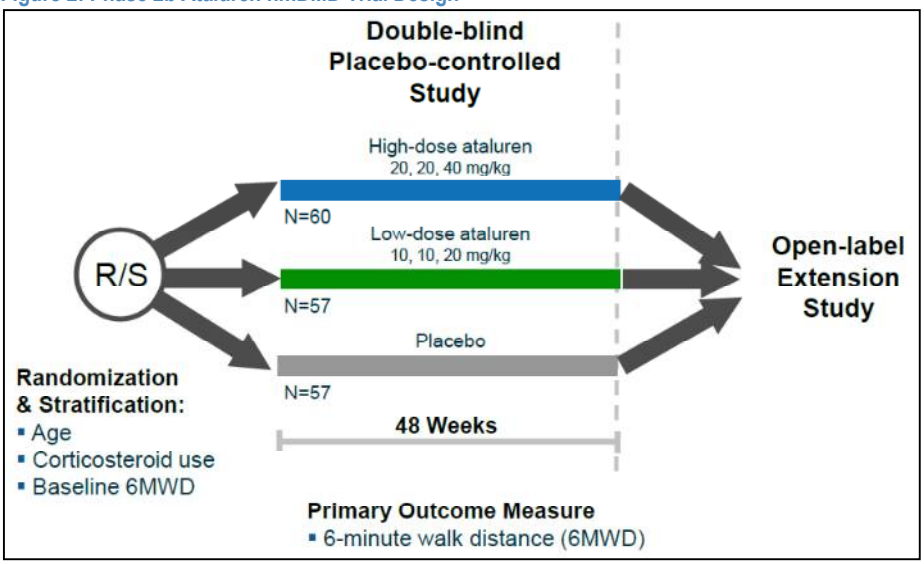
[8] Gonzalez et. al. Orphanet. J. Rare Dis. 2012; 7:58
[9] Buck et. al. Biochem. Biophys. Res. Comm 2012; 427: 753-757
[10] Goldman et. al. EMBO Mol. Med. 2012; 4: 1186-1199
[11] Drake et. al. Am. J. Respir. Cell Mol. Biol. Online: April 16, 2013
[12] Zhou et. al. J. Invest. Dermatol. Online: May 23, 2013
[13] Du et. al. Molec. Therapy Online: June 18, 2013
[14] McDonald et. al. Muscle & Nerve 2010; 42: 500-510

physical function. Of note, the patient reported outcome endpoint was designed by PTC and will also be used for the first time.

Review of Phase 2b Data

A phase 2b trial (Study 007) of ataluren in nmDMD enrolled 174 patients from 37 sites in 11 countries. Patients received ataluren (low dose 40mg/kg/day or high dose 80 mg/kg/day) or placebo for 48 weeks (Figure 2). Of note, the low-dose regimen consisted of 10 mg/kg in the morning, 10 mg/kg midday and 20 mg/kg in the evening (10, 10, 20), while the high-dose regimen consisted of 20mg/kg in the morning, 20 mg/kg midday and 40 mg/kg in the evening (20, 20, 40). The primary endpoint of the study was the mean change from baseline in 6MWD over 48 weeks.

Figure 2: Phase 2b Ataluren nmDMD Trial Design



Source: Company reports.

On an ITT Basis, the DMD Study Didn't Achieve Its Endpoint

On a pre-specified intent-to-treat (ITT) basis, the study failed to demonstrate a significant difference relative to placebo in 6MWD. Specifically, low-dose ataluren resulted in a mean decrease from baseline to 48 weeks of -12.9 meters (SD 72m) in 6MWD compared with -42.6 meters (SD 90m) for placebo. This represents a 29.7 meter difference for ataluren low dose relative to placebo (p=0.149). In contrast, high-dose ataluren did not demonstrate a difference in 6MWD relative to placebo.

Following completion of the study, and an initial analysis of the data, issues were identified, including omissions from the statistical analysis plan and inclusion of 6MWD for two patients with lower limb injuries. After correcting for these issues, low-dose ataluren resulted in the same mean decrease from baseline to 48 weeks in 6MWD of -12.9 meters (SD 72m) compared with a greater -44.1 meters (SD 88m) for placebo (Figure 3). This represents a 31.3 meter difference of low-dose ataluren relative to placebo (p=0.0561). As such, even after correcting for these issues, statistical significance was still not met; however, it was nearly achieved.

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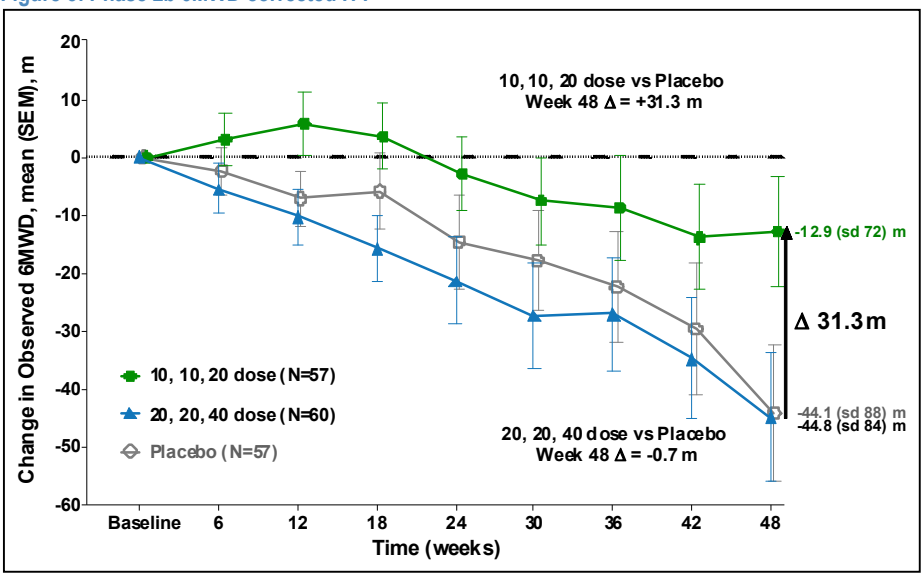
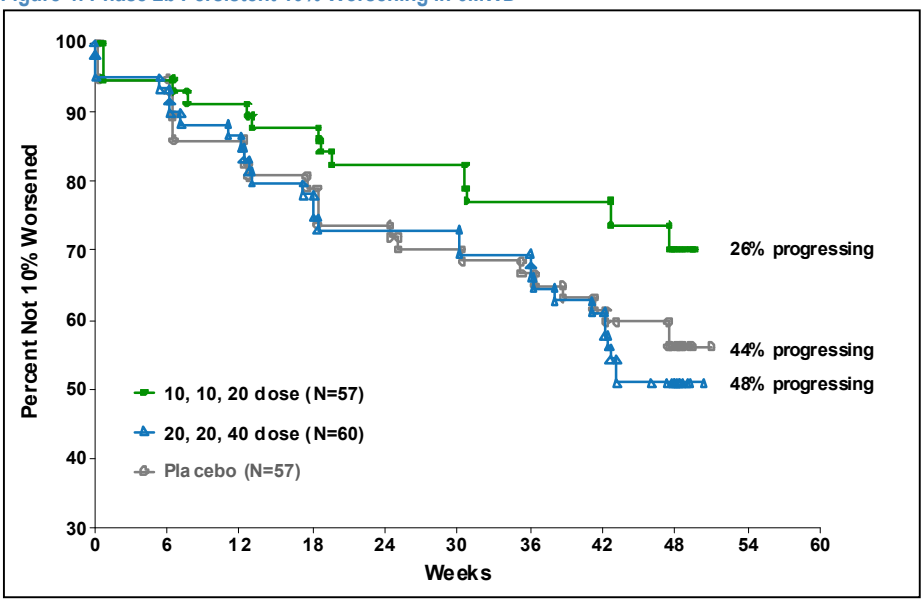
Figure 3: Phase 2b 6MWD corrected ITT



Source: Company reports.

An analysis of the proportion of patients with at least 10% persistent worsening in 6MWD from baseline over 48 weeks was also conducted (Figure 4). Of note, a 10% change in walking ability in one year generally leads to a substantial decline in a patient's clinical status over the next year. At 48 weeks, 26% (p=0.039) of low-dose and 44% (p=0.606) of high-dose ataluren patients progressed compared with 48% for placebo. This analysis suggests a meaningful delay in the decline in ambulation for patients in the low-dose arm.

Figure 4: Phase 2b Persistent 10% Worsening in 6MWD



Source: Company reports.

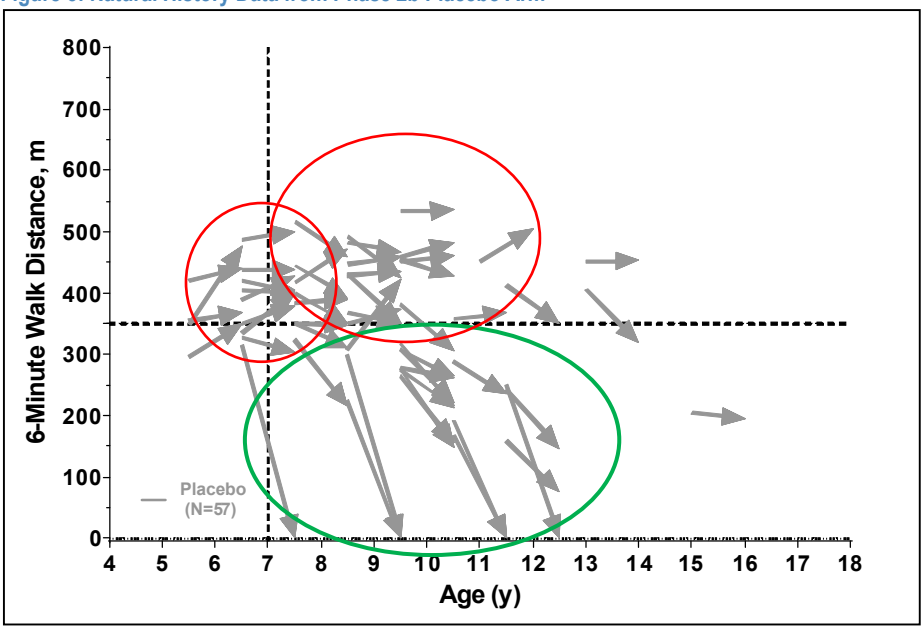
What Went Wrong in the Original Analysis?

While the target treatment effect of 30 meters for the low-dose ataluren arm was met, it was not statistically significant. This was due to greater-than-expected variability in 6MWD due to the heterogeneous nmDMD population selected for the phase 2b study. Indeed, PTC designed the phase 2b study assuming the population would have a standard deviation (SD) of 50 meters on 6MWD over 48 wks. This turned out to be inadequate as the actual SD was 72-88 meters. The high variability was due to substantial differences in disease progression across those patients enrolled in the study (wide age range of <18 years old). Of note, younger patients (<7 years old) and those with higher baseline 6MWD (>350m) were less likely to experience meaningful declines in 6MWD over 48 weeks and, as such, were not ideal candidates for the study.

Subgroup Analyses Tell a Different Story

Natural history data from the placebo arm of the phase 2b study provided some interesting insights about the patients most likely to progress. Indeed, stratifying patients in the placebo arm of the phase 2b study based on age and 6MWD revealed that patients older than 7 years with a baseline 6MWD of <350 meters were most likely to be in the decline phase of the disease (Figure 5). As such, these patients would maximize the treatment effect of ataluren. Indeed, these findings were used to define enrollment criteria for the phase 3 trial.

Figure 5: Natural History Data from Phase 2b Placebo Arm



Source: Company reports.

A subgroup analysis of the phase 2b trial, applying the same enrollment criteria defined for phase 3, resulted in a significant benefit for ataluren relative to placebo. Indeed, the difference between low-dose ataluren and placebo in the mean change from baseline in 6MWD through 48 weeks was 49.9 meters (nominal p = 0.0096; Figure 6). Importantly, this subgroup had less variability in 6MWD as compared with the entire ITT population supporting a significant result. We believe this analysis de-risks the phase 3 trial and maximizes the probability for success.

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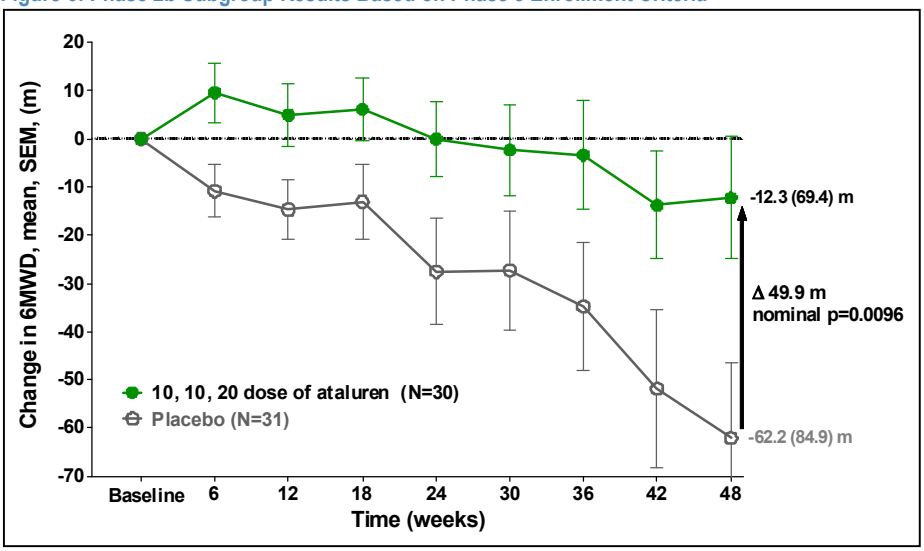
Figure 6: Phase 2b Subgroup Results Based on Phase 3 Enrollment Criteria

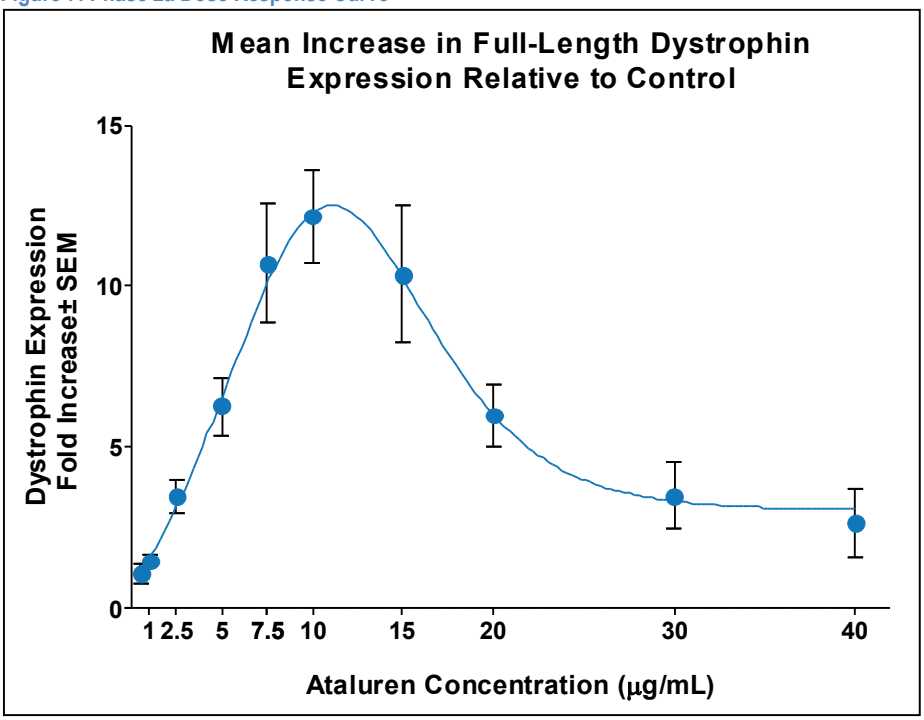


Source: Company reports.

Bell-Shaped Dose Response Curve

In the phase 2b trial, assessing dystrophin levels was unsuccessful as the majority of the muscle biopsies was compromised. However, even if this were not the case, current methods for quantifying dystrophin levels are unreliable. Of note, variations in dystrophin levels can occur within the same muscle as well as between muscles making reliable sampling very difficult. Keeping this caveat in mind, in a phase 2a trial a dose response in dystrophin levels was observed (Figure 7). Additionally, data from preclinical studies confirmed this bell-shaped dose response curve.

Figure 7: Phase 2a Dose Response Curve



Source: Company reports.

Of note, the phase 2b study included a higher dose (20, 20, 40), at the FDA's request, in addition to the 10, 10, 20 dose previously tested, to see if the response would improve at a higher dose. However, the study demonstrated an inverse dose response with the high dose essentially showing no difference relative to placebo. PTC believes that at the higher doses ataluren may also interact with a second binding site on the ribosome disrupting its ability to read through the premature stop codon.

Safety

In the phase 2b study, ataluren was generally well tolerated (Table 4 and Table 5). Of note, the most common AEs include vomiting, headache, diarrhea, nasopharyngitis, pyrexia, cough and upper abdominal pain. Importantly, these AEs were similar across treatment arms and are typical of illnesses among children. Additionally, SAEs were infrequent and none was considered related to ataluren (appendicitis, dehydration, lower limb fracture and supraventricular tachycardia). Additionally no patients discontinued due to AEs.

Table 4: Most Common AEs

Adverse Event	Placebo N=57	Low Dose N=57	High Dose N=60
Vomiting	39%	56%	45%
Headache	25%	39%	25%
Diarrhea	25%	19%	28%
Nasopharyngitis	23%	23%	17%
Pyrexia	21%	25%	12%
Cough	19%	16%	22%
Abdominal pain upper	16%	16%	22%
URT infection	18%	16%	18%
Fall	12%	19%	10%
Nausea	12%	14%	17%
Abdominal pain	7%	12%	17%
Procedural pain	12%	10%	13%
Pain in extremity	11%	12%	13%
Back pain	9%	16%	10%
Influenza	14%	11%	12%

Source: Company reports.

Table 5: AEs Severity and Discontinuations

Characteristic	Placebo N=57	Low-dose N=57	High-dose N=60
Adverse events (AEs) by worst severity*			
Grade 1 (mild)	37%	28%	33%
Grade 2 (moderate)	46%	54%	45%
Grade 3 (severe)	16%	14%	17%
Grade 4 (life-threatening)	0%	0%	0%
AEs by relatedness			
Unrelated	25%	14%	18%
Unlikely	28%	30%	22%
Possible	35%	44%	48%
Probable	11%	9%	7%
Discontinuations due to AE	0%	0%	0%
Serious AEs**	9%	7%	5%

Notes: *Grading by Common Terminology Criteria for Adverse Events (CTCAE); **Serious AEs were those requiring hospitalization.
Source: Company reports.

Regulatory History / Expectations

In March 2011, the final components of an NDA filing for ataluren in nmDMD were submitted to the FDA. However, the FDA refused to file the NDA on the basis that the phase 2b study did not achieve statistical significance in the pre-specified analysis. PTC filed a formal dispute resolution, but the FDA ultimately reaffirmed its original decision in January 2012. As such, PTC anticipates refilling the NDA for nmDMD in late 2015/ early 2016 with data from the ongoing phase 3 trial. As described above, we believe the phase 3 trial has a high probability of success. We expect the FDA's focus to be on ataluren demonstrating a statistically significant benefit in 6MWD compared with placebo. Indeed, the phase 3 study is conservatively powered for a 30m benefit, while a larger benefit (49.9m) based on the phase 2b subgroup analysis is likely.

In October 2012, PTC filed an MAA with the EMEA for conditional approval of ataluren in nmDMD. The filing was accepted for review in November 2012 and an initial response in March 2013 (120-day questions) indicated several major objections that would need to be addressed to support conditional approval (Table 6). PTC anticipates submitting a response in July 2013 with a final EMA decision on conditional approval expected in November 2013. However, given the EMEA's objections we believe conditional approval is unlikely based on the phase 2b data. As such, we similarly expect a refilling in Europe based on data from the phase 3 trial in late 2015/ early 2016. We expect the EMEA focus to be on the magnitude of the benefit for ataluren compared with placebo in 6MWD. We anticipate a similar benefit of 49.9m to that observed in the phase 2b subpopulation being studies in phase 3, which we believe will be adequate for approval.

Table 6: EMEA Major Objections

EMEA Objections	PTC Response
Evidence of efficacy is insufficient in terms of robustness of the data given the number of post-hoc changes was too large.	The post-hoc modifications were warranted and scientifically appropriate.
The separation from placebo in 6MWD was observed only at 42 and 48 wks, which suggest the duration was too short or the results were a chance finding.	A greater effect on 6MWD was seen over time in the trial as indicated by the 10% worsening analysis.
Outcomes of the primary endpoint were not supported by the results from the secondary endpoints, which were inconsistent and difficult to interpret, and there was a similar proportion of patients who lost ambulation in all three arms.	The results of the primary endpoint were supported by trends in secondary endpoints, and while a similar number of patients in all three arms lost ambulation, this was not a trial endpoint.
Question whether a 30m difference in 6MWD is clinically relevant.	Believe 30m represents a clinical relevant distant in DMD. Natural history studies have demonstrated that a 10% decrease in walking ability over 48 wks predicts a loss of ambulation in the following year.
Given weak evidence of efficacy, the risk-benefit is negative for purposes of conditional approval.	Any slowing of disease progression is meaningful, and the safety profile supports a positive risk-benefit.
The choice of the 10, 10, 20 dose of ataluren is not well substantiated given the lack of dose response studies, insufficient data supporting a bell-shaped dose response curve and uncertainties about the effective dose.	A bell-shaped dose response curve is supported by multiple studies and analyses including non-clinical data, published studies, clinical concentration analysis and related modeling efforts that show plasma concentrations in the effective range.

Source: Company reports.

Physicians' Perspective

Physicians we spoke to seem to be excited about the potential for new treatments in DMD. Indeed, enthusiasm for ataluren appears high given currently limited treatment options such as steroids, which are associated with significant side effects. Based on existing data, consensus among physicians was that ataluren is active in DMD. While some expressed concerns about the lack of a response at the higher dose this was not a deal breaker particularly if a significant improvement in 6MWD is confirmed in phase 3. Physicians agreed that any improvement in function is meaningful to patients and 30m seems to be a reasonable bar. Many highlighted the clean safety profile as a key positive. Indeed, some physicians indicated that even if the benefit were lower than 30m, they would still consider using ataluren given the safety profile.

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Ataluren Market Opportunity in DMD

Our model currently includes the opportunity for ataluren in nmDMD (Table 7). We assume a prevalence of 75K DMD patients WW with an estimated 15K in the US, 19K in the EU and the remaining 40K in the ROW. Of these patients, we assume 13% is caused by a nonsense mutation and would be eligible for treatment with ataluren. This results in an addressable patient population of 2K in the US and 2.5K in Europe. We further assume an annual price of $200K, consistent with that of other therapies for rare diseases. In the US and EU we conservatively assume a peak penetration of 50% with a much smaller 5% peak penetration in ROW. We assume a launch of ataluren in 2H16. We project 2016-18 WW sales of $25M, $214M and 308M, respectively. We project WW peak ataluren nmDMD of approximately $700M.

Table 7: Ataluren DMD Market Model

US	2013	2014	2015	2016	2017	2018	2019	2020
Duchenne Muscular Dystrophy (DMD) patients	15400	15708	16022	16343	16669	17003	17343	17690
DMD nonsense patients	2002	2042	2083	2125	2167	2210	2255	2300
% nonsense	13%	13%	13%	13%	13%	13%	13%	13%
Ataluren treated patients	0	0	0	42	542	774	902	1150
% penetration	0%	0%	0%	2%	25%	35%	40%	50%
Price				$200,000	$206,000	$212,180	$218,545	$225,102
US Ataluren Revenues ($M)				**$8.5**	**$111.6**	**$164.1**	**$197.1**	**$258.8**
% growth					*1213%*	*47%*	*20%*	*31%*

EU	2013	2014	2015	2016	2017	2018	2019	2020
Duchenne Muscular Dystrophy (DMD) patients	19000	19380	19768	20163	20566	20978	21397	21825
DMD nonsense patients	2470	2519	2570	2621	2674	2727	2782	2837
% nonsense	13%	13%	13%	13%	13%	13%	13%	13%
Ataluren treated patients	0	0	0	26	401	545	834	1135
% penetration	0%	0%	0%	1%	15%	20%	30%	40%
Price				$200,000	$200,000	$200,000	$200,000	$200,000
EU Ataluren Revenues ($M)				**$5.2**	**$80.2**	**$109.1**	**$166.9**	**$227.0**
% growth					*1430%*	*36%*	*53%*	*36%*

ROW	2013	2014	2015	2016	2017	2018	2019	2020
Duchenne Muscular Dystrophy (DMD) patients	40000	40800	41616	42448	43297	44163	45046	45947
DMD nonsense patients	5200	5304	5410	5518	5629	5741	5856	5973
% nonsense	13%	13%	13%	13%	13%	13%	13%	13%
Ataluren treated patients	0	0	0	55	113	172	234	299
% penetration	0%	0%	0%	1%	2%	3%	4%	5%
Price				$200,000	$200,000	$200,000	$200,000	$200,000
ROW Ataluren Revenues ($M)				**$11.0**	**$22.5**	**$34.4**	**$46.8**	**$59.7**
% growth					*104%*	*53%*	*36%*	*28%*

Total Ataluren WW Revenues ($M)				**$25**	**$214**	**$308**	**$411**	**$546**
% growth					*765%*	*44%*	*34%*	*33%*

Source: J.P. Morgan estimates.

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Ataluren for CF

Ataluren is also being developed for the treatment of nmCF with a second phase 3 trial expected to begin in 1H14. Of note, ataluren failed to meet the primary endpoint in a prior phase 3 trial in nmCF. That said, a pre-specified subgroup analysis revealed that patients not receiving inhaled antibiotics had a statistically significant 6.7% relative FEV1 difference between ataluren and placebo (p=0.013). As such, the second phase 3 trial excludes these patients, maximizing the probability of success. While we currently do not include revenues for nmCF in our model, we believe ataluren has potential in this indication. That said, we project peak nmCF sales of $700M.

CF: A Brief Overview

CF is caused by genetic mutations in the CFTR gene that encodes for the CFTR protein. Under normal conditions, the CFTR protein functions as an ion channel that transports chloride across cell membranes. An inability to regulate chloride results in the buildup of thick sticky mucus that impacts many organs, such as the lungs, pancreas and liver. Mucus in the lungs can lead to airway obstruction and promote bacterial infections. Mucus in the pancreas prevents essential digestive enzymes from reaching the intestines resulting in malnutrition from difficulties breaking down and absorbing food.

Symptoms of CF include salty skin, persistent coughing, wheezing, shortness of breath, frequent lung infections and poor weight gain. Over time, progressive loss of lung function occurs and can eventually lead to respiratory failure and death. The median life expectancy for a person with CF is 37 years. Therapies largely include mucolytics, such as Pulmozyme, which help clear mucus from the lungs. Inhaled antibiotics, such as tobramycin and aztreonam, are also used to keep bacterial infections under control. However, none of these therapies treats the underlying disease. To date, the only approved therapy that impacts the underlying disease is Vertex's Kalydeco.

CF can be broken down into 5 different classes based on mutations (Table 8). Class I is due to a nonsense mutation that inserts a premature stop codon resulting in a truncated nonfunctional CFTR protein. As such, since there is an absence of CFTR, Class I mutations result in the most severe form of CF. Ataluren specifically targets Class I mutations. Class II mutations are the result of misfolding of a full-length CFTR protein, which prevents it from reaching in the cell surface. This is the most common mutation and can be treated by correctors. Correctors, such as Vertex's VX-809 and VX-661, work by getting more CFTR channels to the cell surface. Class III, IV and V mutations result in milder forms of CF disease as CFTR protein successfully reaches the cell surface, but either functions improperly or is present at low levels. Vertex’s Kalydeco is a potentiatior that improves proper functioning of existing CFTR channels and is currently approved in patients with the G551D mutation. As such, since no other agents are targeting Class I mutations ataluren has no direct competitors in CF.

Table 8: Classes of CF Mutations

Mutation Class	CFTR Protien	Percent of CF Population	Drug Class	Drug	Company
I	No synthesis of CFTR due to nonsense mutations	10%	Nonsense suppresor	Ataluren	PTC
II	Processing blocking due to CFTR misfolding preventing it from reaching the cell surface (Ex. F508del)	70%	Corrector	VX-809 and VX-661	Vertex
III	Gating issues resulting in a CFTR that does not function properly (Ex. G551D)	3-5%	Potentiatior	Kalydeco	Vertex
IV	Conductance issue as opening in CFTR is faulty	2%	Potentiatior		
V	Reduced synthesis resulting in lower levels of CFTR	2%	Potentiatior		

Source: J.P. Morgan estimates, Company data.

Design of Upcoming Phase 3 Trial

A second phase 3 trial of ataluren is expected to begin enrollment in 1H14. PTC is currently in discussions with the FDA and EMA regarding trial design and anticipates conducting only one study that will meet the requirements of both agencies. PTC anticipates the study will enroll 210 CF patients due to nonsense mutations. The primary endpoint is expected to be the relative change in percent predicted FEV1, while secondary endpoints will include pulmonary exacerbations and other pulmonary function measures (lung capacity and expiratory flow).

Importantly, patients on inhaled aminoglycoside antibiotics, such as tobramycin, will be excluded from the study as well as those with any changes in treatment 4 weeks prior to the study, have been recently treated with IV antibiotics or have major complications of the disease. Patients will be randomized 1:1 to ataluren 10mg/kg in the morning, 10 mg/kg mid day and 20 mg/kg in the evening (10, 10, 20) or placebo for a duration of 48 weeks, similar to that of the prior phase 3 study. We believe these enrollment criteria will help maximize the probability of success in the second phase 3 study. Data from this second phase 3 trial are expected in 1H16.

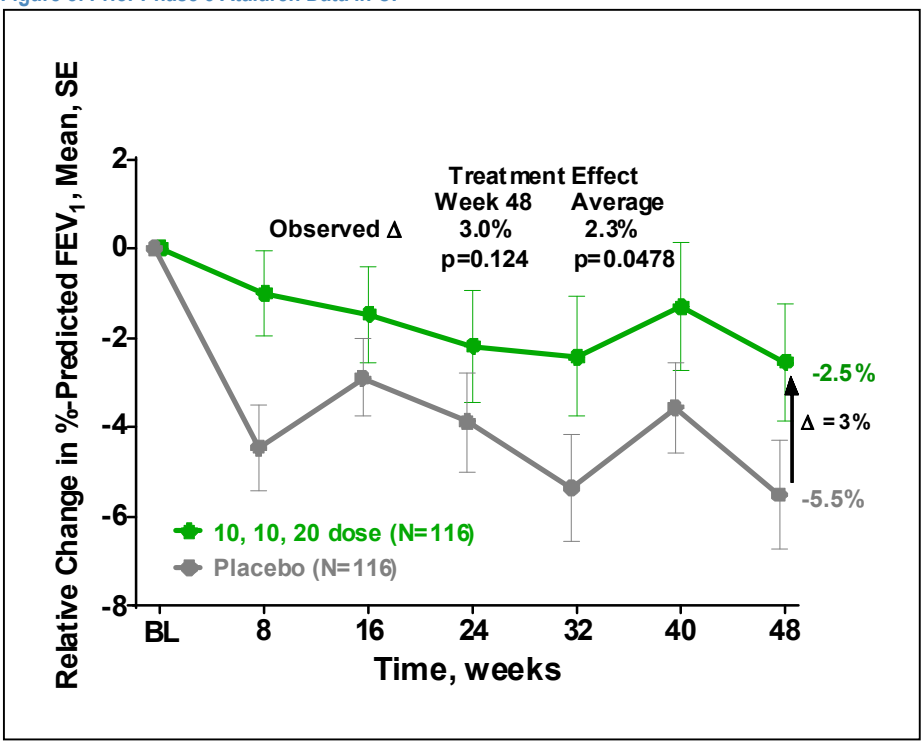
Review of Completed Phase 3 Data

A phase 3 trial of ataluren enrolled 238 CF patients with a nonsense mutation. Patients were randomized 1:1 to ataluren 10mg/kg in the morning, 10 mg/kg mid day and 20 mg/kg in the evening (10, 10, 20) or placebo for a duration of 48 weeks. The primary endpoint was the mean relative change in percent predicted FEV1 from baseline, while secondary endpoints included pulmonary exacerbations.

Primary Endpoint Not Met

The study failed to meet the primary endpoints with a 3.0% difference in relative FEV1 for ataluren (-2.5%) compared with placebo (-5.5%) that was not statistically significant (p=0.124; Figure 8). On an absolute basis a 1.8% difference in FEV1 for ataluren (-1.3%) compared with placebo (-3.1%) was observed and was also not statistically significant (p=0.136).

Figure 8: Prior Phase 3 Ataluren Data in CF

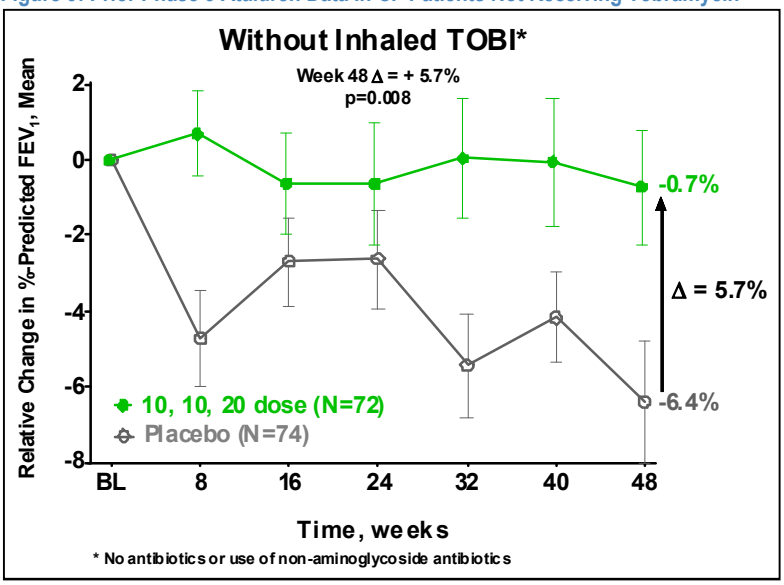


Source: Company reports.

Pre-specified Subgroup Analyses Provide Insights

Pre-specified stratification factors included age, baseline FEV1 and use of inhaled antibiotics. Of note, in patients that did not receive inhaled antibiotics a statistically significant 6.7% relative FEV1 difference between ataluren and placebo was observed (p=0.013). PTC believes that inhaled antibiotics such as tobramycin, which also bind to the ribosome, interfered with ataluren's mechanism of action. This hypothesis was confirmed in a cell-based assay that demonstrated ataluren's ability to read through premature stop codons was decreased when administered in combination with tobramycin or gentamicin. However, this was not the case when ataluren was used in combination with non-aminioglycosides (colistin or aztreonam). Indeed, in the completed phase 3 study, patients that did not receive tobramycin had a statistically significant 5.7% relative difference in FEV1 for ataluren compared with placebo (p=0.008; Figure 9). As such, the use of tobramycin in the phase 3 study seems to be the key driver behind the study not meeting the primary endpoint.

Figure 9: Prior Phase 3 Ataluren Data in CF Patients Not Receiving Tobramycin



Source: Company reports.

Regarding pulmonary exacerbations, a similar trend was observed as the ITT population was not significant, but it was significant in those patients not receiving inhaled antibiotics. Specifically, in the ITT population there was a 23% reduction in the rate of pulmonary exacerbations for ataluren versus placebo (p=0.099). In contrast, patients not receiving tobramycin had 41% fewer exacerbation compared with placebo (p=0.005). As such, this result further confirms tobramycin is likely the culprit in the completed failed phase 3 study.

Market Opportunity for Ataluren in CF

While we believe ataluren has potential in nmCF, we currently do not include revenues in our model. An estimated 70K patients worldwide have CF with approximately 30K in the US and 40K in the ROW. Of these patients 10% would be addressable, resulting in 3K patients in the US and 4K patients in the ROW. Further assuming a $200K price consistent with that assumed in nmDMD and a conservative peak penetration of 50%, we derive a peak WW opportunity of $700M for ataluren in nmCF.

Financial Outlook

Income Statement

We project 2013-16 total revenues of $25M, $10M, $7M and $27M, respectively (Table 10). We anticipate a launch of ataluren in nmDMD in 2H16 in both the US and Europe with 2016-18 WW sales of $25M, $214M and $308M, respectively. Of note, we exclude revenues for ataluren in all other indications including nmCF.

We expect R&D expenses to increase with ataluren in late-stage development including an ongoing phase 3 in nmDMD and a phase 3 in nmCF expected to begin in 1H14. We also expect increases in SG&A to support the expected ataluren

nmDMD launch beginning in 2H16. We project 2013-18 GAAP EPS of $(3.72), $(2.75), $(3.17), $(2.97), $1.99 and $3.40, respectively.

Balance Sheet and Cash Flows

At the end of March 2013, PTC had $50M in cash and equivalents. Following the IPO completed June, we estimate PTC currently has ~$168M in cash and equivalents (Table 11 and Table 12). We conservatively assume additional equity could be raised in 2014.

Valuation

Sum of the Parts

Our December 2014 price target of $20 for PTCT is based on our sum-of-the-parts NPV analysis including ataluren in nmDMD only (Table 9). We project ataluren nmDMD sales to 2024, consistent with IP protection, assume no terminal value and a 10% discount rate. We further assume a 70% probability of success for ataluren in Phase 3. We believe this appropriately reflects the risks of the phase 3 DMD program given the outcome of the prior phase 2b trial. We derive a value of $19/share for ataluren. This taken in combination with net cash of $1/share supports our December 2014 PT of $20.

Table 9: PTC Sum-of-the-Parts Valuation

	Total Value	Per share
Ataluren	$504.3	$19
Cash	$40.0	
Debt	$3.8	
Net Cash	$36.2	$1
Value	**$540.5**	**$20**

Source: J.P. Morgan estimates.

Management

Stuart W. Peltz, Ph.D.
CEO and Director

Since inception in 1998, Dr. Peltz has served as the CEO and as a member of the board. Prior, he was a professor in the Department of Molecular Genetics and Microbiology at the Robert Wood Johnson Medical School, Rutgers University. During that period, Dr; Peltz received a number of scientific awards and published more than 80 papers on post-transcriptional control processes. He received his Ph.D. from the McArdle Laboratory for Cancer Research at the University of Wisconsin.

Claudia Hirawat
President

Since April 2012 Ms. Hirawat has served as President and from April 2006 to April 2012 as SVP of Corporate Development and has held several other positions since

joining PTC in 2000. Prior to joining PTC, she served over 1995-2000 as a VP at LedbetterStevens, a management consulting firm focused on the biopharmaceutical industry.

Shane Kovacs
CFO

Since June 2013, Mr. Kovacs has served as CFO. From March 2014 to May 2013 he held positions of increasing responsibility at Credit Suisse, most recently as a Managing Director. Prior to that, he served as an associate at National Bank Financial from July 2002 to March 2004. He received a B.Eng and a B.S. in Chemical Engineering and Life Sciences from Queen's University and an MBA from the University of Western Ontario, and he also holds the CFA designation.

Mark E. Boulding
Executive VP and Chief Legal Officer

Since March 2012, Mr. Boulding has served as EVP and Chief Legal Officer, while holding SVP and General Counsel positions from April 2002 to February 2012. Before joining PTC, he held positions at MedicalLogic/Medscape, Inc. as General Counsel, EVP and Secretary (May 2000 to April 2002) and General Counsel of Medscape, inc. (June 1999 to May 2000). He was also a partner in two Washington, D.C., based law firms and he received a JD from the University of Michigan and a BA from Yale.

Mark A. Rothera
Chief Commercial Officer

Since April 2013, Mr Rothera has served as Chief Commercial Officer. Prior to joining PTC, from April 2012 to January 2013, he was Global President of Aegerion Pharmaceuticals, Inc. He also served as VP and General Manager of commercial operation for Shire Human Genetic Therapies, Inc. in Europe, the Middle East and Africa. He has also held several positions in various global strategic, operational marketing and sales roles in French and UK operations of Glaxo Wellcome. He received an MA in Natural Science from Cambridge University and an MBA from the European Institute for Business Administration.

Neil Almstead, Ph.D.
Senior VP of Research and CMC

Since July 2008, Dr. Almstead has served as SVP of Research and CMC and previously as SVP Chemistry and CMC (January 2007 to June 2008). Prior, he was a Project Manager at Procter & Gamble Company. He also co-authored over 75 papers and patents related to the design and synthesis of lead-candidate compounds for genetic disorders, oncology and inflammatory disease. He received a BS from Clarkson University and a Ph.D. in Organic Chemistry from the University of Illinois at Urbana-Champaign.

Jay Barth, M.D.
VP of Clinical Development

Since January 2011, Dr. Barth has served as VP of Clinical Development and previously as ED of Clinical Development (February 2009 to December 2010). Prior, he was ED of Clinical Research at Merck (July 2007 to October 2008) and VP of Clinical Research and Medical Affairs at Altana Pharma Company. (July 2007 to October 2008). He received a BA form Columbia University and an MD from the University of Pennsylvania School of Medicine.

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Financial Statements

Table 10: PTC Income Statement

($ in millions except per share data)	2011A	2012A	2013E	2014E	2015E	2016E	2017E	2018E
Revenues								
WW Ataluren (DMD only)	0.0	0.0	0.0	0.0	0.0	24.8	214.3	307.7
Collaboration revenue	99.0	28.8	21.1	8.0	5.0	2.5	2.5	2.5
Grant revenue	6.5	5.2	4.1	2.0	2.0	0.0	0.0	0.0
Total Revenues	**105.4**	**33.9**	**25.1**	**10.0**	**7.0**	**27.3**	**216.8**	**310.2**
Operating Expenses								
Cost of sales	0.0	0.0	0.0	0.0	0.0	2.5	21.2	31.4
Research and development	58.7	46.1	51.2	56.3	61.9	68.1	74.9	82.4
Sales, general and administrative	16.2	14.6	28.5	29.9	35.9	50.2	60.2	69.3
Total Operating expenses	**74.8**	**60.8**	**79.6**	**86.2**	**97.8**	**120.8**	**156.4**	**183.1**
Operating Income	30.6	-26.8	-54.5	-76.2	-90.8	-93.5	60.5	127.1
Interest expense, net	-2.4	-1.2	-2.0	1.0	1.0	2.0	2.0	3.0
Other income, net	0.5	1.8	0.4	0.5	0.5	0.5	0.5	0.5
Total Other Income	-2.0	0.6	-1.6	1.5	1.5	2.5	2.5	3.5
Pretax Income	28.6	-26.2	-56.1	-74.7	-89.3	-91.0	63.0	130.6
Income tax (benefit)	-2.3	0.0	0.0	0.0	0.0	0.0	0.0	19.6
Net Income	**30.9**	**-26.2**	**-56.1**	**-74.7**	**-89.3**	**-91.0**	**63.0**	**111.0**
Deemed dividend	0.0	0.0	-18.2	0.0	0.0	0.0	0.0	0.0
Gain on exchange of convertible preferred stock in connection with recapitalization	0.0	160.0	3.4	0.0	0.0	0.0	0.0	0.0
Less beneficial conversion charge	0.0	-0.4	0.0	0.0	0.0	0.0	0.0	0.0
Net income allocable to common stockholders	30.9	133.3	-71.0	-74.7	-89.3	-91.0	63.0	111.0
Diluted GAAP EPS	5.39	7.75	-3.72	-2.75	-3.17	-2.97	1.99	3.40
Fully diluted shares outstanding	5.7	17.2	20.1	27.2	28.2	30.7	31.7	32.7

Source: Company reports and J.P. Morgan estimates.

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Table 11: PTC Balance Sheet

($ in millions)	2011A	2012A	2013E	2014E	2015E	2016E
Assets						
Cash and cash equivalents	28.4	2.7	133.0	139.4	49.9	63.2
Prepaid expenses and other current assets	3.4	0.9	0.9	0.9	0.9	0.9
Grant and collaboration receivables, net	1.2	1.0	1.0	1.0	1.0	1.0
Total Current Assets	33.1	4.6	134.9	141.3	51.7	65.1
	-	-	-	-	-	-
Fixed assets, net	10.8	8.3	8.6	8.9	9.2	9.5
Deposits and other assets	0.3	0.2	0.2	0.2	0.2	0.2
Total Long Term Assets	11.1	8.5	8.8	9.1	9.4	9.7
	-	-	-	-	-	-
Total Assets	44.1	13.1	143.6	150.4	61.1	74.7
	-	-	-	-	-	-
Liabilities and Equity	-	-	-	-	-	-
Accounts payable and accrued expenses	13.0	7.0	7.0	7.0	7.0	7.0
Current portion of long-term debt	7.1	4.4	4.4	4.4	4.4	4.4
Deferred revenue	23.0	16.7	16.7	16.7	16.7	16.7
Total Current Liabilities	43.1	28.2	28.2	28.2	28.2	28.2
Deferred revenue, less current portion	16.4	0.7	0.7	0.7	0.7	0.7
Long-term debt, less current portion	4.5	0.4	0.4	0.4	0.4	0.4
Other long-term liabilities	4.2	2.5	2.5	2.5	2.5	2.5
Total Long Term Liabilities	25.2	3.7	3.7	3.7	3.7	3.7
Total Liabilities	68.4	31.9	31.9	31.9	31.9	31.9
Total Shareholders' Equity	(24.2)	(18.8)	111.8	118.5	29.2	42.8
Total Liabilities and Equity	44.1	13.1	143.6	150.4	61.1	74.7

Source: Company reports and J.P. Morgan estimates.

Table 12: PTC Cash Flows

($ in millions)	2011A	2012A	2013E	2014E	2015E	2016E
Net Income	30.9	(26.2)	(71.0)	(74.7)	(89.3)	(91.0)
Depreciation	2.9	2.7	2.7	2.7	2.7	2.7
Change in fair value of warrant liability	(0.5)	(1.8)	-	-	-	-
Non-cash interest expense	0.4	0.2	-	-	-	-
Stock-based compensation expense	2.8	2.3	-	-	-	-
Changes in operating assets and liabilities:	-	-	-	-	-	-
Prepaid expenses and other current assets	(1.4)	2.5	-	-	-	-
Grant and collaboration receivables	2.1	0.2	-	-	-	-
Deposits and other assets	0.2	0.1	-	-	-	-
Accounts payable and accrued expenses	(3.2)	(6.0)	-	-	-	-
Other long-term liabilities	(0.1)	0.1	-	-	-	-
Deferred revenue	(55.0)	(22.0)	-	-	-	-
Net change in Working Capital	(57.3)	(25.1)	-	-	-	-
Net Cash From Operations	(20.8)	(47.9)	(68.3)	(72.0)	(86.6)	(88.3)
Purchases of fixed assets	(0.2)	(0.2)	(3.0)	(3.0)	(3.0)	(3.0)
Purchases of investments	(2.0)	-	-	-	-	-
Maturities of investments	29.9	-	-	-	-	-
Net Cash from Investing	27.7	(0.2)	(3.0)	(3.0)	(3.0)	(3.0)
	-	-	-	-	-	-
Payments on long-term debt	(7.2)	(6.9)	-	-	-	-
Net proceeds from sale of Series One preferred stock	-	29.4	-	-	-	-
Proceeds from issuance of common stock	0.0	-	114.3	81.4	-	104.6
Net Cash from Financing	(7.2)	22.4	114.3	81.4	-	104.6
Net Increase (Decrease) in Cash	(0.2)	(25.7)	43.0	6.4	(89.6)	13.3
Cash and cash equivalents at beginning of period	28.7	28.4	90.0	133.0	139.4	49.9
Cash and cash equivalents at end of period	28.4	2.7	133.0	139.4	49.9	63.2

Source: Company reports and J.P. Morgan estimates.

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PTC Therapeutics: Summary of Financials

Income Statement - Annual	FY12A	FY13E	FY14E	FY15E
Revenues	34	25	10	7
Cost of products sold	0	0	0	0
Gross profit	-	-	-	-
SG&A	(15)	(28)	(30)	(36)
R&D	(46)	(51)	(56)	(62)
Operating income	(27)	(54)	(76)	(91)
EBITDA	(27)	(54)	(76)	(91)
Net interest (income) / expense	(1)	(2)	1	1
Other income / (expense)	2	0	1	1
Income taxes	0	0	0	0
Net income - GAAP	(26)	(56)	(75)	(89)
Net income - recurring	133	(71)	(75)	(89)
Diluted shares outstanding	17	20	27	28
EPS - excluding non-recurring	(1.52)	(2.80)	(2.75)	(3.17)
EPS - recurring	7.75	(3.54)	(2.75)	(3.17)
Balance Sheet and Cash Flow Data	**FY12A**	**FY13E**	**FY14E**	**FY15E**
Cash and cash equivalents	3	133	139	50
Accounts receivable	-	-	-	-
Inventories	-	-	-	-
Other current assets	2	2	2	2
Current assets	5	135	141	52
PP&E	8	9	9	9
Total assets	13	144	150	61
Total debt	5	5	5	5
Total liabilities	32	32	32	32
Shareholders' equity	(19)	112	118	29
Net income (including charges)	(26)	(71)	(75)	(89)
D&A	3	3	3	3
Change in working capital	(25)	0	0	0
Other	1	0	0	0
Cash flow from operations	(48)	(68)	(72)	(87)
Capex	(0)	(3)	(3)	(3)
Free cash flow	(47)	(69)	(74)	(89)
Cash flow from investing activities	(0)	(3)	(3)	(3)
Cash flow from financing activities	22	114	81	0
Dividends	-	-	-	-
Dividend yield	-	-	-	-

Income Statement - Quarterly	1Q13A	2Q13E	3Q13E	4Q13E
Revenues	7A	6	6	6
Cost of products sold	0A	0	0	0
Gross profit	-	-	-	-
SG&A	(4)A	(6)	(8)	(10)
R&D	(11)A	(13)	(13)	(14)
Operating income	(9)A	(13)	(15)	(18)
EBITDA	(9)A	(13)	(15)	(18)
Net interest (income) / expense	(1)A	(1)	(1)	(1)
Other income / (expense)	0A	0	0	0
Income taxes	0A	0	0	0
Net income - GAAP	(9)A	(13)	(16)	(18)
Net income - recurring	(24)A	(13)	(16)	(18)
Diluted shares outstanding	16A	17	24	24
EPS - excluding non-recurring	(0.57)A	(0.77)	(0.66)	(0.77)
EPS - recurring	(1.51)A	(0.77)	(0.66)	(0.77)
Ratio Analysis	**FY12A**	**FY13E**	**FY14E**	**FY15E**
Sales growth	(67.8%)	(25.9%)	(60.2%)	(30.0%)
EBIT growth	(187.7%)	103.2%	39.8%	19.2%
EPS growth - recurring	43.7%	(145.6%)	(22.4%)	15.3%
Gross margin	-	-	-	-
EBIT margin	(79.0%)	(216.7%)	(761.6%)	(1296.6%)
EBITDA margin	(79.0%)	(216.7%)	(761.6%)	(1296.6%)
Tax rate	0.0%	0.0%	0.0%	0.0%
Net margin	392.8%	(282.3%)	(746.6%)	(1275.2%)
Net Debt / EBITDA	(8.0%)	235.2%	176.7%	49.6%
Net Debt / Capital (book)	(12.9%)	783.2%	837.8%	285.4%
Return on assets (ROA)	466.1%	(90.6%)	(50.8%)	(84.4%)
Return on equity (ROE)	(619.6%)	(152.7%)	(64.9%)	(120.9%)
Enterprise value / sales	-	-	-	-
Enterprise value / EBITDA	-	-	-	-
Free cash flow yield	(16.1%)	(20.4%)	(16.1%)	(18.6%)

Source: Company reports and J.P. Morgan estimates.
Note: $ in millions (except per-share data).Fiscal year ends Dec

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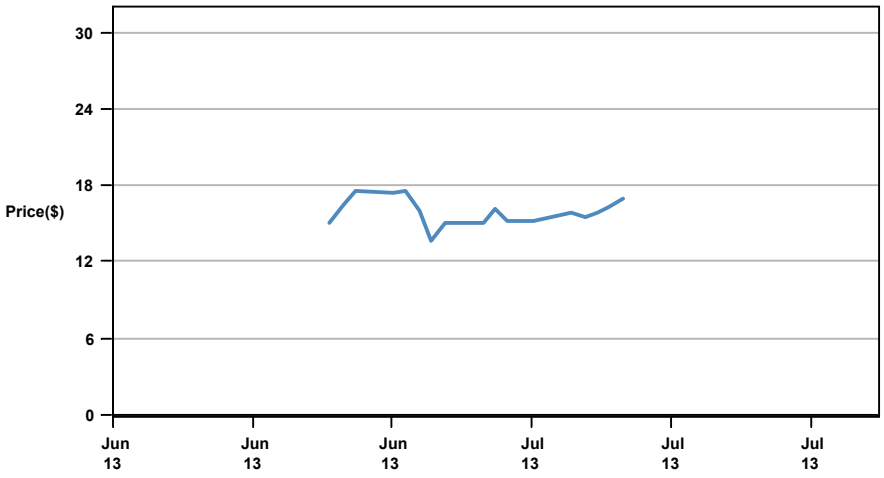

Source: Bloomberg and J.P. Morgan; price data adjusted for stock splits and dividends.

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