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PTC Therapeutics (PTCT - OUTPERFORM): SMA Data Update: ISIS SMA Candidate to Inform and Potentially Expedite Development of PTC/ROCHE's Oral RG7800 for SMA - Reiterate OUTPERFORM

Price: $29.99 **12-Month Price Target: $55**

- **Monday morning ISIS (ISIS: Not Covered) provided an update on interim results from their ongoing Phase Ib/IIa trial of SMNrx in children with SMA.** Preliminary results were compelling and support the notion that induction of SMN protein via the SMN2 gene may represent a disease modifying therapy for patients with SMA. The trial enrolled patients with Type II or III SMA aged 2-14 years to receive multiple doses of 3, 6, 9 or 12 mg (ongoing) SMNrx. Recall ISIS is developing SMNrx with partner Biogen (BIIB: Not Covered) across several trials summarized in Figures 1-2, Page 3-4. We anticipate an additional update from ISIS's SMA trial at AAN, April 26-May 3 (Philadelphia, PA).

- **ISIS reported a dose dependent increase of 1.5, 2.3 and 3.7 points in the Hammersmith Functional Motor Scale (highlighting improved function) in the 3, 6 and 9 mg cohort respectively measured 9 months after the first dose of SMNrx.** The expanded Hammersmith scale ISIS used in this trial (HFSME) is a 70 point scale that was designed for and validated in SMA type II and III patients (Glanzam 2011). Importantly, Dr. Thomas Crawford (John Hopkins), a SMA specialist noted on the call that a change of 3 points on the Hammersmith score is clinically meaningful to patients with SMA.

- **ISIS found a 160% (> 2 fold) increase in SMN protein in the CSF in children with SMA receiving 9 mg SMNrx (120%, > 2 fold, at 6 mg) 9 months after dosing in the "CS1" single ascending dose trial. In the multiple dose study, CS2, SMN protein levels increased 115% at the time of the 9 mg second dose.** Importantly it also appeared that Hammersmith scores were improving overtime, something that is unexpected given the natural history of the disease. They noted that in early animal models, a maximum of 3.5 fold increase was found and ISIS believes that a 2.5 fold increase is enough to be "transformative" in the lives of SMA patients.

- **We note that PTC's compound, RG7800, demonstrated a nearly 3 fold increase in SMN protein levels in the brain in early animal models, enough to nearly achieve healthy heterozygote protein levels (Figure 3, Page 4).** Although slightly lower than SMNrx in preclinical models, this is encouraging data for an oral candidate compared to ISIS' injection directly into the spinal column. We also note that duration and frequency of dosing possible with oral RG7800 may also lead to higher fold increases in SMN protein levels. We highlight that ease and safety of systemic delivery of RG7800 may be preferential over intrathecally delivered compounds and may better facilitate delivery very early in life a time during which animal models of SMA suggest maximum therapeutic benefit may be conferred.

- **Results from the Phase I trial of RG7800 may, in addition to safety and PK data, confirm mechanistic action through the measurement SMN transcript levels in healthy patients blood plasma.** Recall that RG7800 is designed to increase the levels of full length SMN2 transcripts and thus SMN protein levels compared to the alternatively spliced SMN2 delta7 transcript that produces dysfunctional protein. We anticipate that decreased levels of SMN delta7 transcript in blood plasma cells, and increased levels of SMN may be indicative of the drugs mechanistic activity in healthy volunteers.

- **We anticipate that the next catalysts for PTCT shares will include an update on full enrollment in the Phase III trial of ataluren in nmDMD and additional SMA data updates. Updated milestones page 2.** PTCT is also expected to announce a new indication for ataluren in 2014 as well as the start of enrollment in a Phase III trial for ataluren in cystic fibrosis (nmCF). New indications for ataluren's clinical development are to be selected in 2014 and may offer rapid routes to registration supported by excellent safety and tolerability from the current 600-patient strong clinical data set.

- **Given the breadth of programs, opportunity for ataluren in new indications we view PTCT with ~$260M in cash and an EV of $620 million as attractive on a valuation basis despite the recent run.**

- **We reiterate our OUTPERFORM rating and 12-month price target of $55/share.** Our $55 price target is derived by applying an 8X multiple to estimated 2017 revenues for ataluren in nmDMD and nmCF, discounted 25% and 35% annually, respectively. Conditional approval of ataluren in the EU and success of the SMA candidate remain upside to our price target.

Risks to the attainment of our price target include 1) failure of ataluren in the clinic in DMD or CF; 2) regulatory failure of ataluren; and 3) inability to fund the development or execute on the commercializing of ataluren globally

Upcoming Milestones

Q1:14	Initiation of a Phase III trial of ataluren in nmCF
Apr 26-May 3	AAN Meeting (Philadelphia, PA), potential ataluren updates.
Apr 26-May 3	AAN Meeting (Philadelphia, PA) ISIS/BIIB updates on SMNrx Phase I results
Q2:14	Feedback from CHMP SAG meeting regarding request for reconsideration of conditional approval for ataluren for nmDMD in the EU
Q2:14	Potential MAA filing for conditional approval of ataluren for nmCF in the EU
Q2:14	Potential new opinion following a re-examination of the negative opinion regarding conditional approval of ataluren for nmDMD in the EU
Q2:14	Full enrollment in the confirmatory Phase III trial of ataluren in nmDMD
2014	Nomination of one or two new indications for ataluren (likely Aniridia and/or MPS I)
2014	Open label trial updates for ataluren as nmDMD at a scientific conference (US study safety only, EU efficacy at 0, 6,12,18 months)
H2:14	Potential data from the Phase IIb open-label extension study of ataluren in 4 in the EU
YE:14/Q1:15	Potential start of proof-of-concept Phase II trials (potentially pivotal) for ataluren in one or two indications (ahead of ataluren in nmDMD)
YE:14	Potential conditional approval of ataluren for nmCF in the EU
YE:14/Q1:15	Potential top-line data and biomarker data from Roche/PTCT's Phase I healthy volunteers study of SMA candidate RG7800
H1:15	Top-line data from the confirmatory Phase III trial of ataluren in nmDMD
2015	Potential first commercial sale (named patient/compassionate use) sales of ataluren nmDMD in EU (or ROW) markets
H2:15	FDA and MAA filing for full approval of ataluren for nmDMD
H2:15	Top-line data from the confirmatory Phase III trial of ataluren in nmCF
2015	Potential initiation of pivotal Phase II/III trials RG7800 in patients with SMA
H1:16	FDA and MAA filing for full approval of ataluren for nmCF
2016	First commercial sales of ataluren following potential approval on Phase III data

PTC/Roche's RG7800 has Several Advantages vs. Competitors

SMN is a validated causative agent in spinal muscular atrophy, and other companies are developing therapies targeting the alternative splicing of SMN2. PTC's compound has significant advantages over ISIS/Biogens's antisense RNA that must be injected directly into the spinal column of an infant, increasing the risk of a serious brain infection. Importantly, SMN2 is expressed in most cell types including PBMC's and the molecular effect of the drug can be ascertained with a blood test. We highlight that this Phase I trial will be informed and enriched by the bio-marker analysis currently being conducted (NCT01910168) by PTC and Roche.

Figure 1: PTC's Candidate Compared to ISIS/BioGens SMN_{RX}

	ISIS/Biogen's SMN_{RX}	Repligen's RG3039	PTC's SMN2 RG7800
Route of Administration	Intrathecal	Oral	Oral
Current Phase	Phase I/II	Phase Ib	Phase I
Mechanism of Action	SMN2 gene alternative splicing	SMN2 Inducer	SMN2 gene alternative splicing
Drug Type	RNA antisense	Small Molecule	Small Molecule
Phase I Trial Design	28 Type II or III pts, aged 2-14 yrs	Not Disclosed	Not Disclosed
Key Safety Results	NO SAE's or DLT"s found up to 9 mg	"Interim phase I clinical results in healthy volunteers indicate no adverse effects at drug exposure that inhibits DcpS enzyme completely in circulating PBMC"	Ongoing
Key Efficacy Results	SMN Protein levels increased 160% at 9 mg	Not Disclosed	Ongoing

Source: Company data, Wedbush Securities, Inc.

ISIS SMNrx Trial and Data Overview

ISIS SMNrx has been tested in 3 primary clinical trials and two extension studies. The first Phase I, single ascending dose trial, *CS1*, enrolled SMA Type II or III patients to receive single doses of 1, 3, 6, and 9 mg. Key entrance criteria included documented SMN1 homozygous gene deletion, clinical signs attributable to SMA aged 2-14 years and no ventilation, gastric feeding tube or previous scoliosis surgery. The primary endpoint of the study was the number of patients with adverse events; the secondary endpoint was plasma PK. This trial found that no SAE's or DLT's up to 9 mg and later trials were amended to increase the dose to 12 mg. Importantly, this trial found increases of 160% in SMN protein level over baseline (260% of baseline) at the 9mg dose 9 months later. We highlight that PTC's preclinical data suggest that heterozygous SMN1 mutated mice have roughly 3x higher SMN protein than homozygous mice (Figure 3).

The dose expansion study, *CS2*, in children enrolled Type II and III patients with documented 5q SMA homozygous gene deletion or mutation, clinical signs attributable to SMA, aged 2-15 years, no ventilation, gastric tube or prior scoliosis surgery. The primary endpoint of the study is the number of participants with adverse events; secondary endpoints include plasma PK and CSF PK. Interim results from this trial indicate SMNrx is safe and well tolerated at 3 and 6 mg given on days 1, 29 and 85 and at 9 mg given on day 1 and 85. The 12 mg cohort is still enrolling. Importantly this trial found a positive trend towards improving clinical benefit as measured by the Expanded Hammersmith Functional Motor Scale (HFMSE), a validated scale in SMA type II and III.

The infant study, *CS3A*, enrolled infants with Type I SMA, < 210 days old with documented 5q SMA mutation or deletion, with onset of signs and symptoms consistent with SMA between 21 day and 180 days (6 months), body weight > 5 th percentile, gestational age 35-42 weeks and gestation body weight ≥ 2kg and no hypoxemia, untreated active infection, or implanted CSF chunt or CNS catheter. The primary endpoint of this study is the number of participants with adverse events; secondary endpoints are plasma PK and CSF PK. Patients received multiple doses of 2 and 6 mg SMNrx, later amended to include a 12 mg cohort. At ages 9.5-16 months, four of four patients in the 6 mg cohort were alive and not on ventilation, encouraging, given the typical natural history of the disease.

Figure 2: ISIS' SMNrx Clinical Trial Program

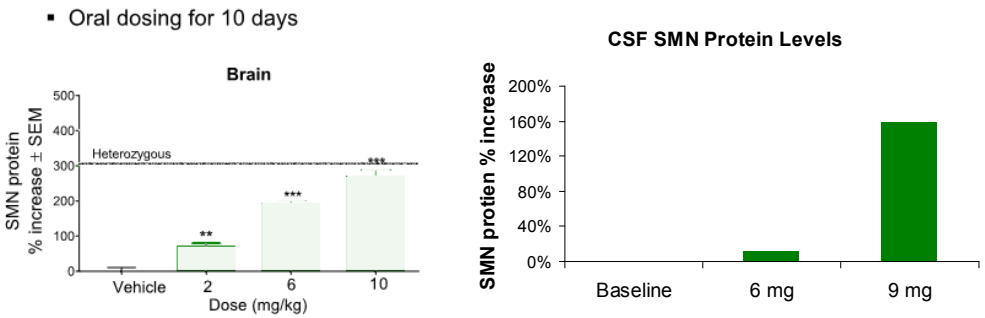
Name	CS3A	CS2	CS1	CS10	CS12
N	20	32	28	28	52
Design	Non-randomized, open-label	Non-randomized, open-label	Non-randomized, open-label	Single-arm	Single-arm
Type	Multiple-Dose Expansion in SMA Infants	Multiple dose expansion in SMA patients	Single ascending dose	Extension study of CS1	Extension of CS2 or CS10
Age	< 210 days	2-15 years	2-14 years	2-14 years at CS1 screening	2-15 years
Entrance Criteria	Genetic 5q SMA deletion or mutation	Genetic 5q SMA deletion or mutation	SMN1 homozygous gene deletion, SMA Type II or III	SMN1 homozygous gene deletion	SMN1 deletion or mutation
Status	Recruiting	Recruiting	Completed	Active	Enrolling
Dose	2, 6 and 12 mg	3 and 6 mg administered on days 1, 29 and 85. 9mg administered on day 1 and 85. 12 mg recruiting	1, 3, 6, and 9 mg	Monitoring only	12 mg
Results	4/4 infants in 6 mg alive and not on ventilation after 9.5-16 months. 9 of 10 children in 12 mg alive. Single death occurred after 2 doses and < 2 months of treatment	3, 6 and 9 mg achieved HFSME improvements of 1.5, 2.3 and 3.7 points 9 months following first dose	No SAE's or DLT's found, all AE's mild or moderate. SMN protein levels in the CSF increased 160% (9mg) and 120% (6mg) 9 months after first dose.	Ongoing	Ongoing
NCT	NCT01839656	NCT01703988	NCT01494701	NCT01780246	NCT02052791

Source: Company data, Wedbush Securities, Inc.

RG7800 and SMNrx in Animal Models May Help Predict In Human Performance

RG7800 caused a nearly 3 fold increase in SMN protein increase in the brain after oral dosing in homozygous SMA mouse models. This compares favorably with ISIS' 3.5 fold increase on SMNrx when injected directly into the brain. We highlight that ISIS is comfortable going forward into a Phase III pivotal trial with the 2.5 fold SMN protein increase at 9 mg dose they are currently seeing in clinical trials.

Figure 3: RG7800 in Animal Models (left) and SMNrx in Humans (right) Dose Dependently Induces SMN Protein Expression in the Brain

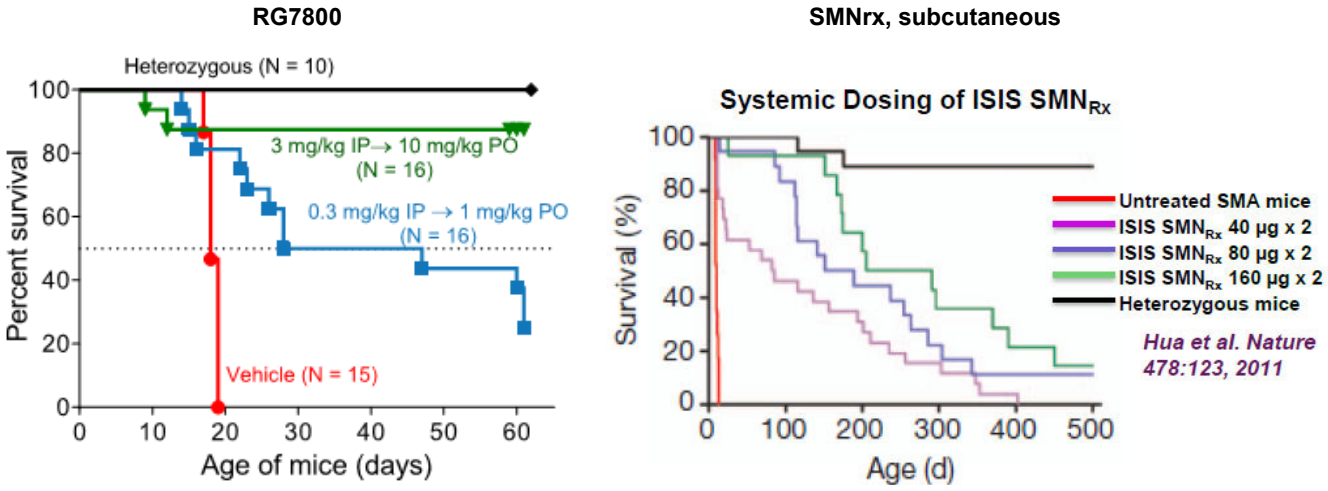


Source: Company data, Wedbush Securities, Inc.

RG7800 Improved Survival and Weight Gain in Multiple Mouse Models and Demonstrated Similar Results to ISIS's SMNrx

RG7800 improved survival and weight gain in a severe Type 1 SMA mouse model. This is a remarkable improvement, qualitatively comparable to the AAV induced SMN expression shown by Dominguez et al (Figure 7). This compares to SMNrx out to 60 days at least. We note that comparisons across trials should be made cautiously, especially in preclinical studies with different model animals and dosing regimes as in this comparison.

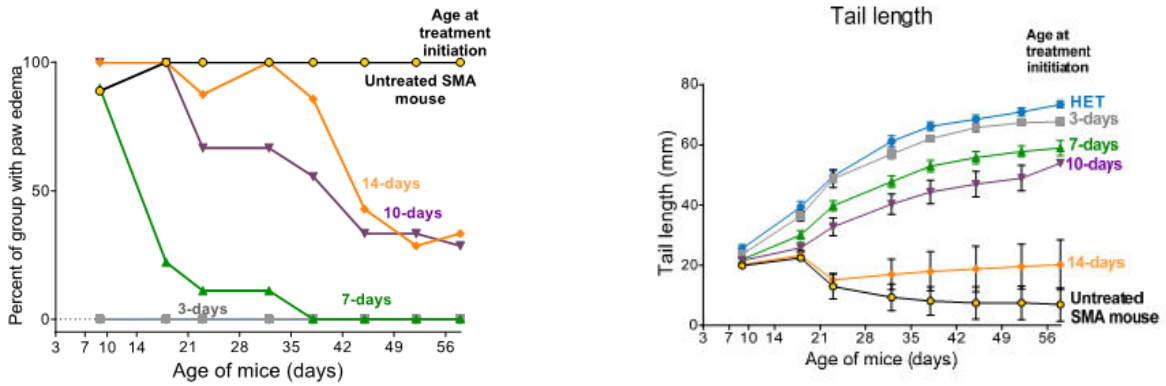
Figure 4: RG7800 (left) and SMNrx (right) Improves Survival and Weight Gain in a Severe SMA Mouse Model



Source: Company data, Wedbush Securities, Inc.

This compound has also been able to rescue milder models, including reducing tail necrosis and paw edema in an SMA III mouse model. Importantly, it is effective even when given several days after birth. One of the challenges of SMA is that the disease progresses rapidly from birth and is not always diagnosed until the disease has progressed. Note here that it is effective more than 10 days after birth in mice.

Figure 5: RG7800 Reduced Paw Edema and Tail Necrosis in Mild SMA Mouse Models

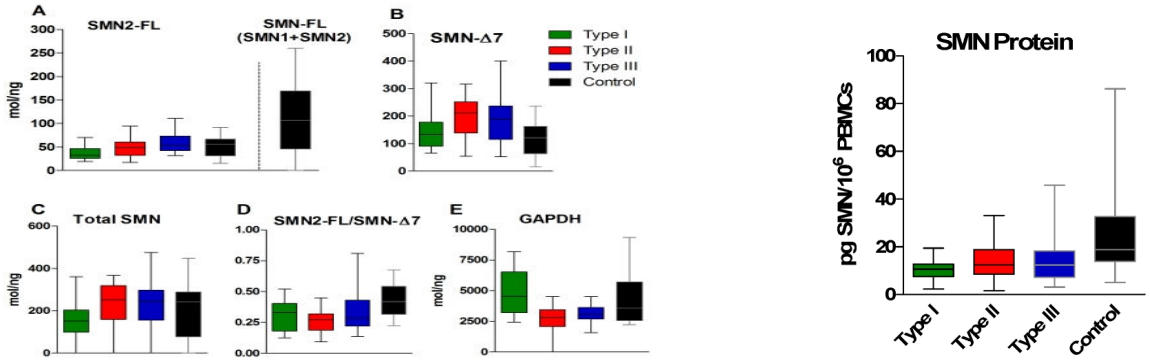


Source: Company data, Wedbush Securities, Inc.

SMN Levels Appear Predictive of SMA Severity

SMA transcript and protein levels correlate with SMA type, as defined by disease severity in multiple studies. Recall "Type I" defines those who never sat independently, "Type II" those who sat, but never walked, and "Type III" those who were able to achieve independent ambulation. Importantly, SMN transcript levels can be analyzed by a blood test and do not require invasive biopsies. Note that in Figure 6, SMN2-FL is translated into fully functional SMN protein. Transcript levels were all statistically significant at p<.05, protein levels were not, though a trend is visible.

Figure 6: Full length SMN Transcript Levels (left) and SMN Protein Levels (right) Correlate with SMA Type.



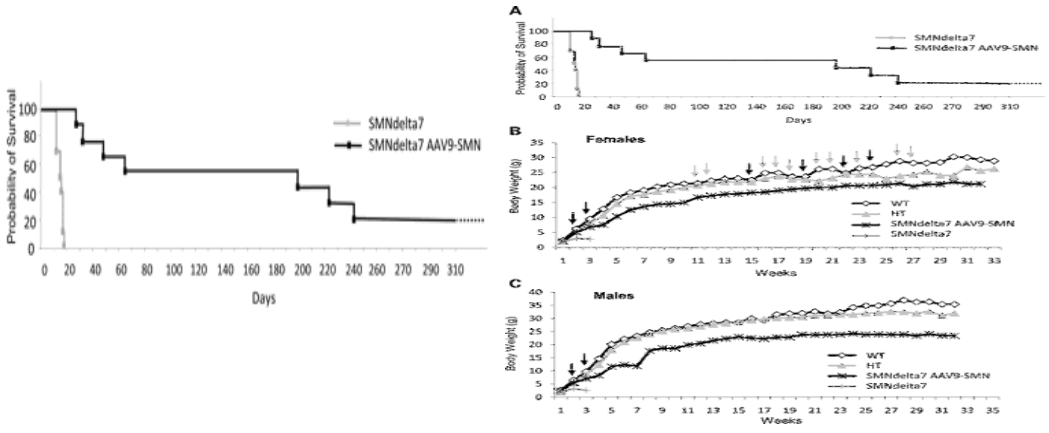
ANCOVA pair-wise comparisons controlling for age	Within SMA Type comparisons			SMA Type vs Control		
	I vs. II	I vs. III	II vs. III	Type I	Type II	Type III
SMN-FL	0.033	<0.001	0.020	<0.001	<0.001	<0.001
SMN protein	0.12	0.10	0.90	<0.001	0.002	0.004
SMN2 copy number*	0.032	<0.001	0.013	0.007	<0.001	<0.001

*Source: Crawford et al. PLoS One. 2012;7(4):e33572 *SMN2 copy number values were not log transformed as they are ordinal values.*

Replacement of SMN by Gene Therapy Rescues Mouse Models

Several attempts have been made to upregulate SMN protein. Viral vectors can produce dramatic and specific SMN upregulation. Multiple studies have shown that is the critical re-introduction of SMN transcript via viral vectors increases survival in several mouse models. Shown below in Figure 7 is an example of the dramatic rescue SMN production can have in mouse models. Note that even without high levels of SMN expression in the brain, mice gained weight and survived much like WT mice.

Figure 7: SMN Replacement by Viral Vectors Substantially Rescues SMA Mouse Models



Source: Dominguez et al Hum Mol Genet. 2011 Feb 15;20(4):681-93

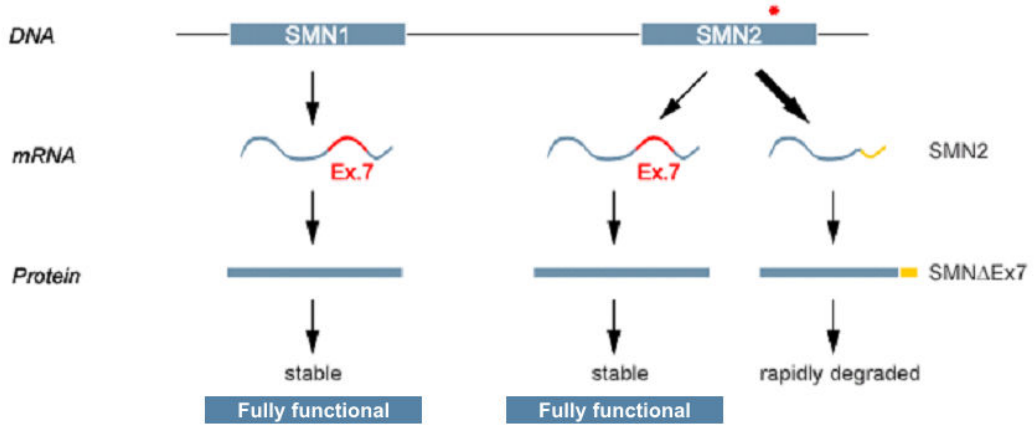
Taken together, the correlation in human studies and the causative relationship demonstrated in mouse models validates the SMN transcript level as a marker of SMA disease. Importantly, SMN is expressed by most cells in the body and transcript level is easily assessed by a blood test and does not require the muscle biopsy needed in some diseases, such as DMD.

Spinal Muscular Atrophy

Spinal Muscular Atrophy (SMA) is caused by low levels of the SMN protein, due to deletions or defects in the SMN1 gene. There is no treatment available. Spinal motor neurons are especially sensitive to SMN loss, for reasons that are not clear, and die rapidly in SMA. Degeneration of spinal motor neurons followed by denervation of skeletal and intercostals muscles leads to muscle weakness, paralysis and eventual respiratory failure.

In humans, a second copy of the gene, SMN2, encodes SMN with a transcriptionally silent mutation that causes exon 7 to be spliced out, resulting in non-functional protein. ~10% of SMN2 RNA transcripts are alternatively processed to full SMN protein. RG7800 increases the proportion of full-length SMN protein produced from the SMN2 gene by modifying alternative splicing pathways.

Figure 8: SMN2 Alternative Splicing Leads to Non-functional SMN Protein



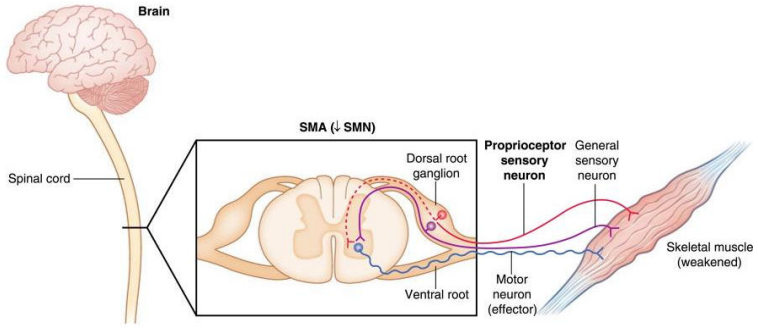
Source: Company data, Wedbush Securities, Inc.

Spinal Muscular Atrophy

Spinal muscular atrophy (SMA) is an autosomal recessive neurodegenerative condition with a wide clinical spectrum characterized by muscle wasting and weakness. The disease generally manifests early in life. Spinal muscular atrophy is caused by defects in the Survival Motor Neuron 1 (SMN1) gene that encodes the survival motor neuron (SMN) protein. The mechanism by which SMN1 deletion/defects causes SMA remains unknown; however, two hypotheses have been proposed. The first hypothesis is that deletion of SMN1 disrupts small nuclear ribonucleoproteins that are important for motor neuron circuits. The second hypothesis is that SMN1 deletion results in deficits of mRNA transport in neurons. A homozygous deletion in the SMN1 gene mapped to chromosome 5q13 was found to cause SMA in 98.6% of patients. In approximately 67% of type 1 SMA chromosomes, the gene for neuronal apoptosis inhibitory protein was found to be deleted. Disease severity may be associated with deletion of this protein; however, the mechanism leading to increased disease severity is still not well understood.

The SMN protein is critical to the health and survival of the nerve cells in the spinal cord responsible for muscle contraction (Figure 8). When SMN is not present α-motor neurons of the anterior horn of the spinal cord are selectively destroyed, manifestations typical of SMA, including proximal muscle and trunk weakness occur. A second gene, SMN2, is very similar to SMN1, except that the former produces less effective SMN protein because, unlike SMN1, SMN2 does not include a particular nucleotide sequence known as exon 7. It is believed that by including exon 7 in SMN2 translation, SMN protein produced by the SMN2 gene may replace defective SMN in patients with SMA. There is currently no marketed therapy approved to treat the underlying cause of spinal muscular atrophy. Currently available treatments for spinal muscular atrophy are only palliative.

Figure 8: The Physiological Impact of Lower SMN from SMA



Source: Swoboda KJ. Curr Neurol Neurosci Rep. 2012 Feb;12(1):42-53

SMA Prognosis is Dependent on SMN2 and SMN

Spinal muscular atrophy is the leading genetic cause of death in infants and toddlers. The mean ages of onset for types I, II, and III SMA are about 3.1, 8.7, and 21.1 months, respectively (Figure 9). Among SMA type 1 children, 45% survive to 1 year of age, 38% to year 2 and 29% to year three. Type I infants with symptom onset after 2 months of age have significantly increased survival compared to those with earlier onset, reinforcing the prognostic value of age at onset of symptoms, which has been consistently demonstrated across and within subtypes as a significant predictor of phenotypic severity.

Figure 9: Clinical Classification Criteria for Spinal Muscular Atrophy

SMA Type	Onset	Motor Milestones	Outcome	SMN2 copies
0	Neonatal	Severe weakness and arthrogryposis	Death in first weeks	1
I	Before 6 mos.	Cannot sit	Death in first 2 years	2
II	6-18 mos.	Cannot walk	Respiratory problems and scoliosis complicating survival	3
III	After 18 mos.	Eventually loses walking ability	Adult life but with respiratory problems and scoliosis	3 or 4
IV	Adult life	Usually can walk	No major complications	4

Source: Wedbush Securities, Inc.

In SMA, the observation that SMN2 mRNA level remains the most potent modifier of phenotype, with an inverse relationship between SMN2 mRNA and disease severity has been critical in driving therapeutic development. Nevertheless, phenotype correlation with SMN2 levels remains insufficient for use in clinical practice for prognosis, a point worth emphasizing given the increasingly widespread application of this assay to newly diagnosed patients. Current molecular techniques used to determine SMN2 mRNA levels typically do not detect intact copies, but rather target exon 7 only, providing potentially incomplete genotypic data. Although one must be cautious about using SMN2 dosage for prognosis, the identification of two SMN2 copies in a child with early infantile onset, particularly if respiratory muscle weakness is already apparent, is helpful to predict SMA type 0.

Current Treatment of SMA

There is no known cure for SMA, current treatment is symptomatic and focuses on nutritional, respiratory, cardiac and muscle skeletal-directed therapy. We highlight the diagnostic process for patients suspected to have SMA (Figure 10).

Figure 10: Diagnosis of SMA

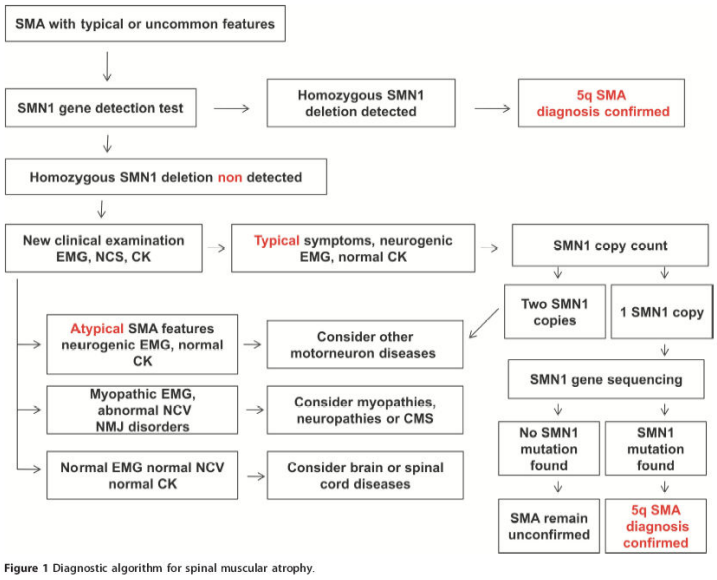


Figure 1 Diagnostic algorithm for spinal muscular atrophy.

Source: Bertini E. Orphanet J Rare Dis. 2011 Nov 2;6:71. doi: 10.1186/1750-1172-6-71.

Market Opportunity in SMA

According to the SMA Foundation, spinal muscular atrophy is the leading genetic cause of death in infants and toddlers. The SMA Foundation estimates that spinal muscular atrophy affects approximately 10,000 to 25,000 children and adults in the United States and that between one in 6,000 and one in 10,000 children are born with the disease. The α-motor neurons of the anterior horn of the spinal cord are selectively destroyed, which leads to manifestations typical of SMA, including proximal muscle and trunk weakness. In SMA 0, the most the most severe form manifests in the first months of life, usually with a quick and unexpected onset ("floppy baby syndrome"). Rapid motor neuron death results in inefficiency of organs - especially of the respiratory system. Pneumonia-induced respiratory failure is the most frequent cause of death. 60% of births are type I or 0 SMA. Babies diagnosed with SMA type 0 do not generally live past two years of age with death occurring as early as within weeks in the most severe cases. We believe that the opportunity in SMA for PTC is substantial even with low-double digit royalties from their partner Roche. We believe that their SMA candidate could be approved in 2016 and see peak US sales of $3.5 billion owing to the deadliness of the disease in children. We conservatively estimate these royalties to be worth $350,000 million to PTC annually with no expenses associated with them beyond low single-digit royalties to the SMA foundation for their initial financial support.

SMA Competitive Landscape

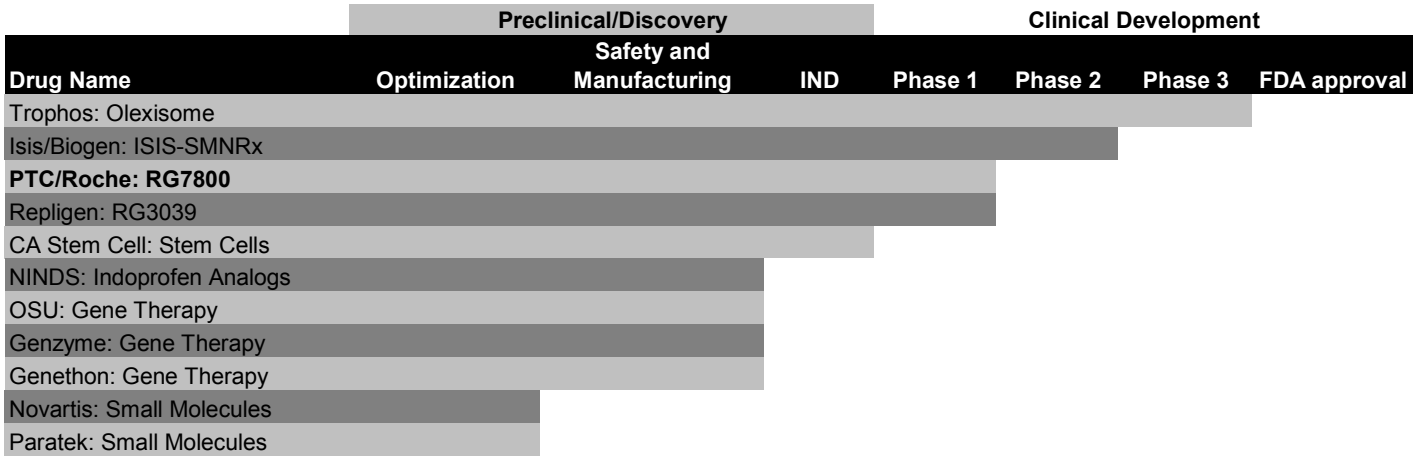
There are no approved treatments for the underlying cause of spinal muscular atrophy with currently available therapies for spinal muscular atrophy are only palliative. A substantial amount of research is focusing on new therapeutic possibilities in SMA. These efforts primarily fall under two main strategies: SMN dependent approaches focus on attempts to address the genetic defect via SMN2 stimulation by drugs or via SMN1 replacement by gene therapy. SMN independent approaches aims to provide motor neuron protection and improve skeletal muscle function. It is likely, similar to other genetic disorders that one therapeutic solution alone will not be sufficient and combined therapeutic strategies may be considered.

There are several SMA treatments in development, three of which are in the clinic, including Trophos' olesoxime, mitochondrial pore modulator in Phase 3 trials, Repligen's RG3039, a DcpS inhibitor in Phase I trials, an ISIS Pharma's/Biogen Idec's SMNRX an antisense therapy. Therapies in development for SMA are summarized in Figure 11.

Figure 11: SMA Therapies in Development

Mechanism	Developer	Drug type (name)	Phase
SMN splicing	Isis Pharmaceuticals & Biogen Idec	Antisense oligonucleotide (ISIS-SMNRx)	1b/2a and 2
	Roche & PTC Therapeutics	**Small molecule**	**Phase 1**
	Novartis	Small molecule	Preclinical
	Paratek Pharmaceuticals	Small molecule	Preclinical
SMN translation	Repligen	Small molecule (RG3039)	1b
SMN stabilization	NINDS	Small molecule (ALB-111)	Preclinical
SMN gene therapy	Genzyme	Viral vector	Preclinical
	Généthon & INSERM	Viral vector	Preclinical
	Nationwide Children's Hospital	Viral vector	Preclinical
Neuroprotection	Trophos	Small molecule (olesoxime)	3

*Olesoxime, a mitochondrial pore modulator, is designed to protect neurons but does not affect SMN levels.



Source: Company data, Wedbush Securities, Inc.

Analyst Certification
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Wedbush Equity Research Disclosures as of February 24, 2014

Company	Disclosure
PTC Therapeutics	1,3,4,5,7

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5. WS provided investment banking services within the last 12 months.
6. WS is acting as financial advisor.
7. WS expects to receive compensation for investment banking services within the next 3 months.
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10. The research analyst, a member of the research analyst's household, any associate of the research analyst, or any individual directly involved in the preparation of this report has a long position in the common stocks.
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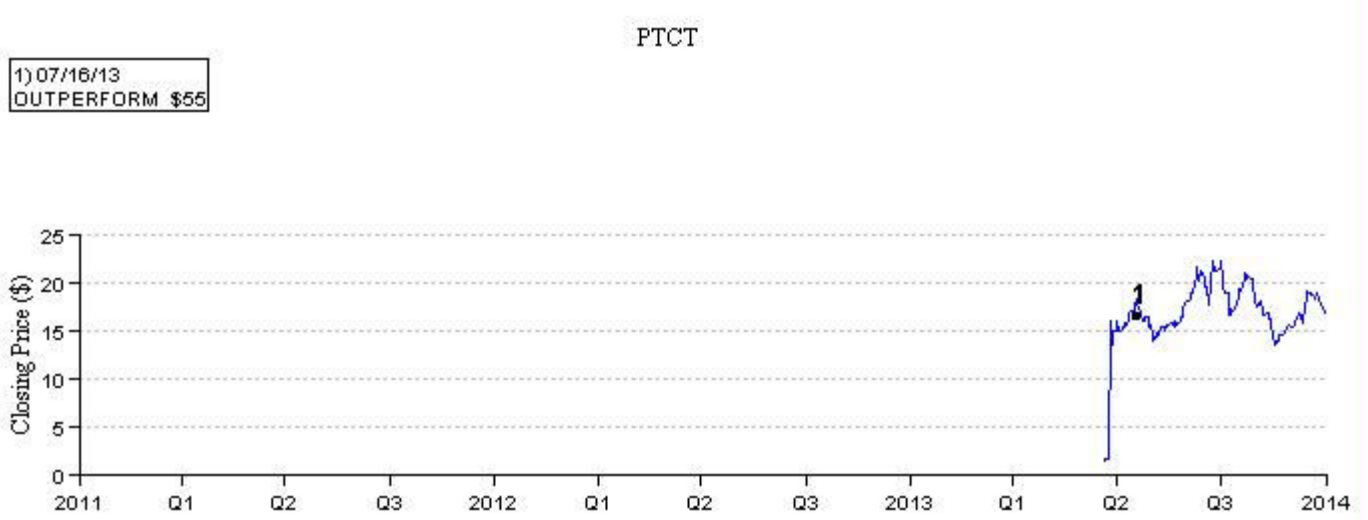
Price Charts
Wedbush disclosure price charts are updated within the first fifteen days of each new calendar quarter per FINRA regulations. Price charts for companies initiated upon in the current quarter, and rating and target price changes occurring in the current quarter, will not be displayed until the following quarter. Additional information on recommended securities is available on request.



* WS changed its rating system from (Strong Buy/Buy/Hold/Sell) to (Outperform/ Neutral/Underperform) on July 14, 2009.
Please access the attached hyperlink for WS' Coverage Universe: http://www.wedbush.com/services/cmg/equities-division/research/equity-research Applicable disclosure information is also available upon request by contacting Ellen Kang in the Research Department at (213) 688-4529, by email to ellen.kang@wedbush.com, or the Business Conduct Department at (213) 688-8090. You may also submit a written request to the following: Business Conduct Department, 1000 Wilshire Blvd., Los Angeles, CA 90017.

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