WEDBUSH QUICK NOTE

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Christopher N. Marai, Ph.D. (415) 274-6861 Gregory R. Wade, Ph.D. (415) 274-6863 David M. Nierengarten, Ph.D. (415) 274-6862

PTC Therapeutics (PTCT - OUTPERFORM): Beyond Ataluren: SMA Trial Start Ahead of Schedule Triggers $7.5M Milestone - Buy on Weakness around EMA Decision - Reiterate OUTPERFORM

Price: $28.18 **12-Month Price Target: $55**

- **This morning, PTCT announced that its SMA compound, partnered with Roche and the SMA foundation, has entered clinical development.** The Phase I trial will assess the safety and tolerability of PTCT's oral SMA candidate in healthy volunteers. Initiation of the trial triggers a $7.5M milestone payment to PTC from Roche, recall the company previously received a $10M payment for selection of a SMA candidate. Under the agreement with Roche, PTC is eligible for up to double-digit royalties and up to $442.5M in remaining milestone payments for certain development and commercialization milestones.

- **We note that the initiation of clinical testing is ahead of schedule, which we believe is indicative of Roche's excitement around the compound and a potential future rapid clinical development path - data from the trial is expected in 2015.** We believe that as an orally bioavailable small molecule PTCT/Roche's (ROG:not covered) SMA candidate is differentiated from the ISIS/BioGen Idec (ISIS:not covered, BIIB:not covered) approach for SMA (Figure 1). We highlight that ease and safety of systemic delivery of PTCT's compound 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.

- **Importantly we note that Roche, in collaboration with PTCT, has run an SMA biomarker trial** (NCT01910168) **which we believe may further expedite the clinical development of this compound.** The biomarker trial enrolled 40 patients with 5q-autosomal recessive spinal muscular atrophy (SMA) type I, II or III as judged by their neurologist upon diagnosis. The trial will measure SMN1/SMN2 in blood by mRNA as well as SMN protein levels in blood and lymphocytes.

- **PTC/Roche's oral SMA compound demonstrated impressive systemic efficacy in SMA mouse models, prolonging survival and reducing phenotypic abnormalities.** In addition to demonstrating a dose-dependent increase in SMN protein, PTCT showed that early therapy (day-0) with their compound appears to result in optimal restoration of near-normal life span and phenotypic characteristics. Demonstration of systemic impact of their SMA candidate, imparted by excellent bioavailability, included prevention of tail necrosis, as well as reduction in paw edema in SMA Type III mice. The candidate has also been shown to confer protection from neuromuscular junction denervation.

- **We believe that as an orally bioavailable small molecule, PTCT/Roche's SMA candidate is differentiated from the ISIS (ISIS:Not Covered) and PMO approaches for SMA.** We highlight that ease and safety of systemic delivery of PTCT's compound 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. Trial designs and data highlighted on pages 3-9.

- **We anticipate that the EMA/CHMP will announce their opinion regarding the conditional approval of ataluren (*Translarna*) in nmDMD next week at the January 20-23 meetings (result January 24). Conditional approval of ataluren for nmDMD remains upside to our $55 price target. We recommend aggressively accumulating shares of PTCT on any weakness or higher volume around this event in anticipation of several other catalysts for PTCT in 2014.** Importantly, we believe that the company may launch ataluren on a *named-patient basis* in the EU should it not be conditionally approved (our base case) in late 2014.

- **We view PTCT with an EV of ~$500 and breadth of their clinical programs as undervalued relative to peers at similar stages of development.** 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, potential new indications for ataluren and additional SMA data updates.

- **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

Investment Thesis

PTC is a biotechnology company focused on the development of ataluren, a molecularly targeted, orally delivered treatment for rare and ultra-rare diseases including Duchenne muscular dystrophy and cystic fibrosis caused by non-sense mutations. Ataluren may also be effective in treating 2500 other rare diseases and certain cancers caused by non-sense mutations since its mechanism of action is broadly applicable to these molecular lesions. PTC is also developing a candidate for spinal muscle atrophy a fatal and rare disease that most severely impacts infants. We believe that ataluren will be shown to be safe and efficacious in on going Phase III trials in nmDMD as well as those set to begin (H1:14) in nmCF. In our opinion, the Street is overly discounting their lead Phase III program for ataluren in nnDMD, which showed mixed results, overall in a Phase II trial, but positive trends and nominal statistically significant benefit in the current subgroups being evaluated in the Phase III trial. We believe that, given lessons learned from prior pioneering trials of ataluren in DMD, PTC has enriched their Phase III trial for success and that it is highly likely to show positive results in mid:2015. Additionally, PTC may also receive conditional approval for ataluren for nmDMD in the EU by Q1:14, a scenario which would result in a 2014 launch and upside to our $55, 12-month price target. Final read-outs from a soon to be initiated (H1:14) trial for ataluren CF are anticipated by mid:16. PTC's SMA program, partnered with Roche, remains additional upside to our estimates. We believe that this program likely be accelerated through the clinic due to significant unmet medical need in this devastating disease and that breakthrough results in Phase I/II trials could form the basis for a registration filing as early as 2015.

Valuation Methodology

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 by Q1:14 remains upside to our price target and would yield a 12-month price target of $75/share. We project that approval and commercialization of ataluren could generate ~$550 million in annual worldwide revenues in 2017 (our valuation year) in nmDMD and nmCF and potential peak global sales of >$1.5 billion. Success of the PTC's pre-clinical SMA candidate remains upside to our estimates. Similarly we arrive at our $55 price target by applying a 15x multiple to PTC's fully taxed EPS in 2017 discounted back 20% annually.

Upcoming Milestones

~~Q4/Q1:14~~	~~CHMP SAG meeting regarding potential conditional approval for ataluren for nmDMD in the EU~~
Jan. 20-24	Potential conditional approval of ataluren for nmDMD in the EU
Q1:14	Initiation of a Phase III trial of ataluren in nmCF
Q1:14	MAA filing for conditional approval of ataluren for nmCF in the EU
~~**H1:14**~~	~~**Initiation of Phase I/II trials of SMN2 candidate for SMA**~~
Mid:14	Full enrollment in the confirmatory Phase III trial of ataluren in nmDMD
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 in the EU
YE:14	Potential conditional approval of ataluren for nmCF in the EU
H1:15	Completion of the confirmatory Phase III trial of ataluren in nmDMD
H2:15	FDA and MAA filing for full approval of ataluren for nmDMD
H2:15	Completion of the confirmatory Phase III trial of ataluren in nmCF
2015	Data from initial trials of PTCT's and Roche's SMA candidate
Late 2015	Potential accelerated approval of candidate for SMA
H1:16	FDA and MAA filing for full approval of ataluren for nmCF

PTC/Roche's Oral SMA Compound has 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}

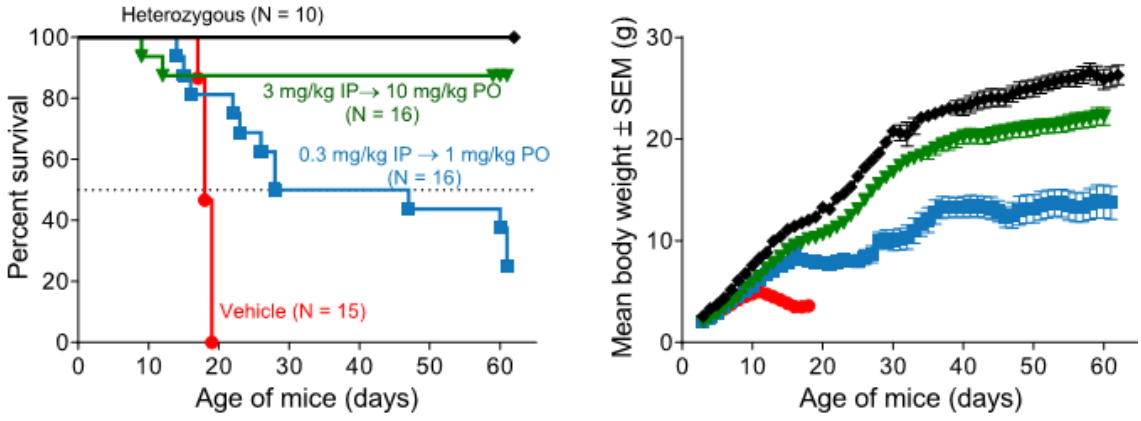
	ISIS/Biogen's SMN_{RX}	PTC's SMN2 Candidate
Route of Administration	Intrathecal	Oral
Current Phase of	I/II	I
Mechanism of Action	SMN2 gene alternative splicing	SMN2 gene alternative splicing
Drug Type	RNA antisense	Small Molecule

Source: Company data, Wedbush Securities, Inc.

PTC's Candidate Improves Survival and Weight Gain in Multiple Mouse Models

PTC's compound 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 6). The ability of an orally available compound to induce this level of phenotypic rescue in multiple mouse models appears promising.

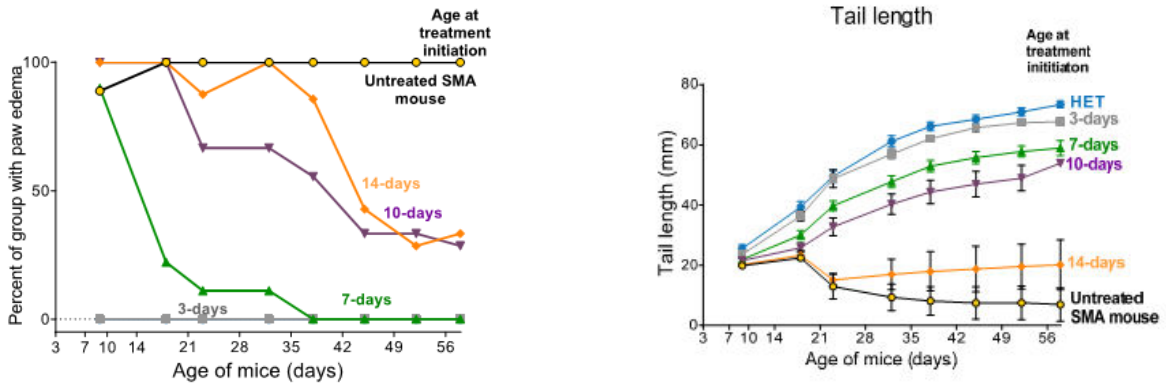
Figure 2: PTC's Candidate Improves Survival and Weight Gain in a Severe SMA Mouse Model



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.

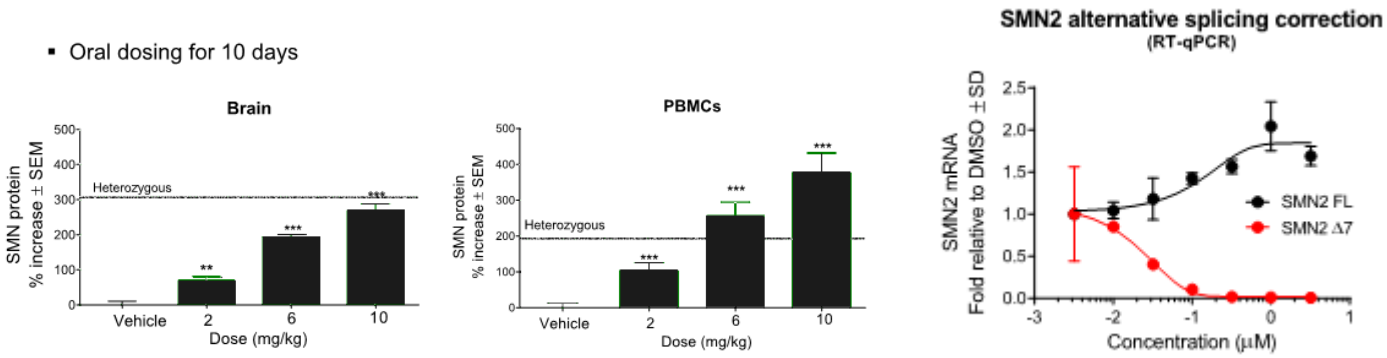
Figure 3: PTC's Compound Reduced Paw Edema and Tail Necrosis in Mild SMA Mouse Models



Source: Company data, Wedbush Securities, Inc.

This candidate has demonstrated the ability to cross the blood brain barrier and induce SMN full length protein in the brains of mouse models. We note that SMN1 heterozygotes are asymptomatic and considered carriers. We believe it is reasonable to propose that achieving levels below SMN1 heterozygote levels may be effective in treating the disease.

Figure 4: PTC's Candidate can Induce Full Length SMN Expression in the Brain and Blood (left) and Dose-Dependently Increases SMN Full Length Transcripts (right)

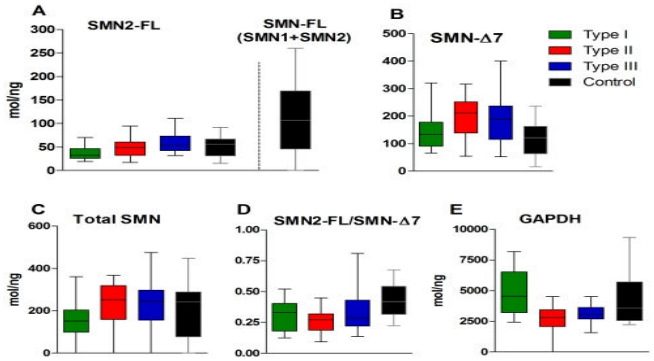


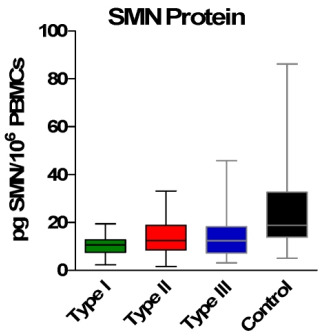
Source: Company data, Wedbush Securities, Inc.

SMN levels are a validated causative agent in SMA

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 5, 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 5: Full length SMN Transcript Levels (left) and SMN Protein Levels (right) Correlate with SMA Type.





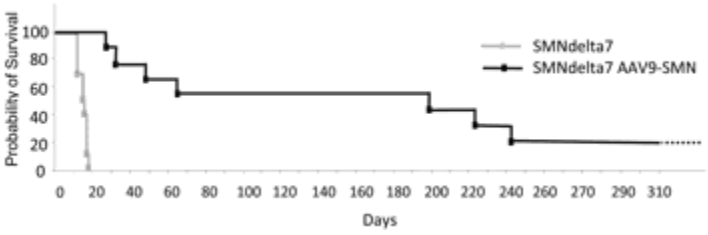
ANCOVA pair-wise comparisons controlling for age	Within SMA Type comparisons			SMA Type vs Control		
	I vs. II	I vs. III	II vs. III	Type I	Type II	Type III
SMN-FL	0.033	<0.001	0.020	<0.001	<0.001	<0.001
SMN protein	0.12	0.10	0.90	<0.001	0.002	0.004
SMN2 copy number*	0.032	<0.001	0.013	0.007	<0.001	<0.001

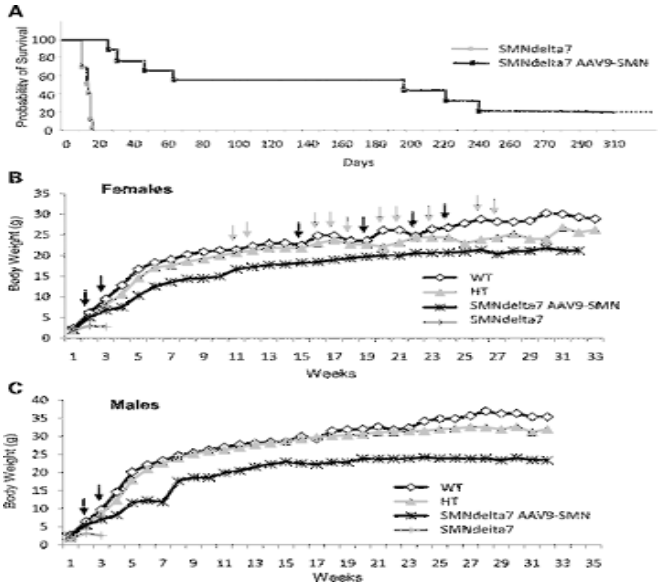
*Source: Crawford et al. PLoS One. 2012;7(4):e33572 *SMN2 copy number values were not log transformed as they are ordinal values.*

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 6 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 6: SMN Replacement by Viral Vectors Substantially Rescues SMA Mouse Models





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

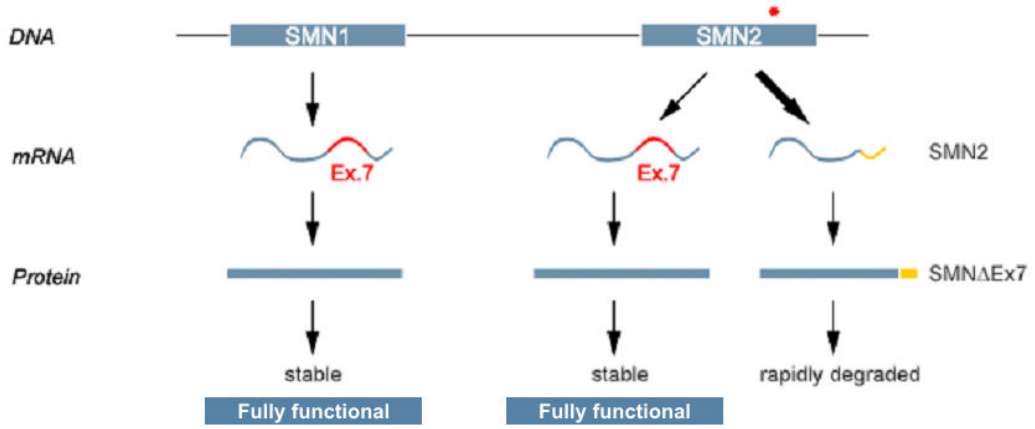
Taken together, the correlation in human studies and the causative relationship demonstrated in mouse models validates the SMN transcript level as a marker of SMA disease. Importantly, SMN is expressed by most cells in the body and transcript level is easily assessed by a blood test and does not require 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. PTC's candidate increases the proportion of full-length SMN protein produced from the SMN2 gene by modifying alternative splicing pathways.

Figure 7: SMN2 Alternative Splicing Leads to Non-functional SMN Protein



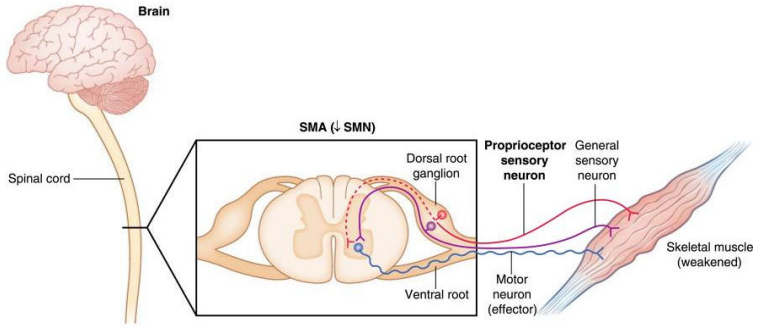
Source: Company data, Wedbush Securities, Inc.

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

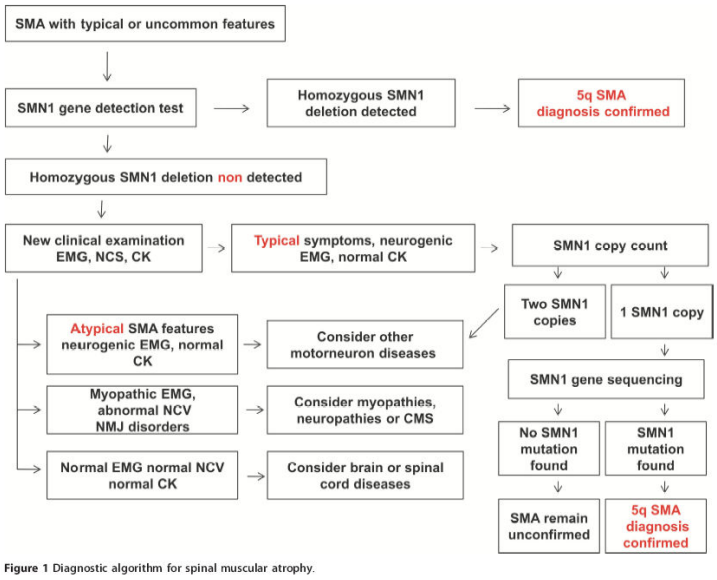


Figure 1 Diagnostic algorithm for spinal muscular atrophy.

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

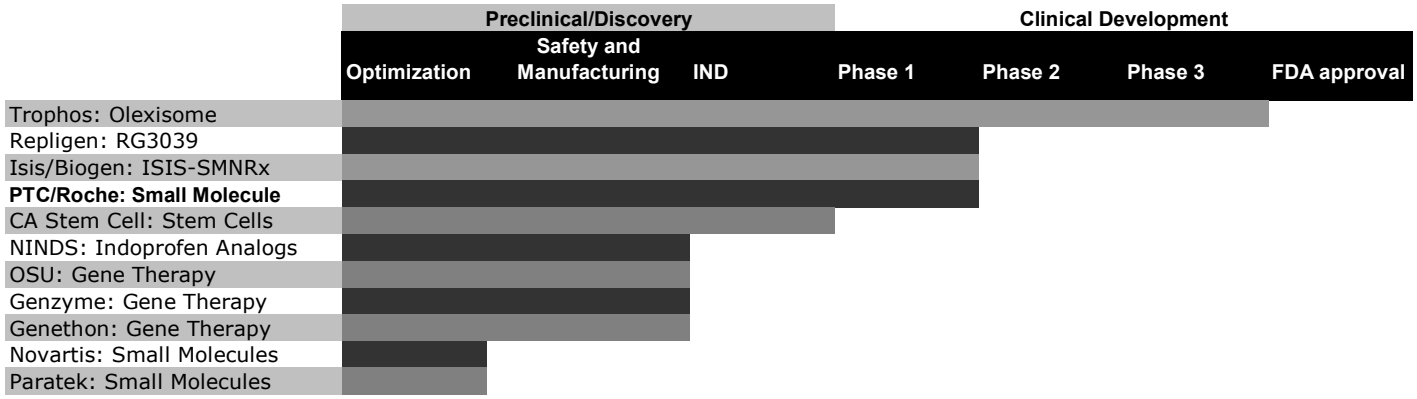
protection and improve skeletal muscle function. It is likely, similar to other genetic disorders that one therapeutic solution alone will not be sufficient and combined therapeutic strategies may be considered.

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.

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Company	Disclosure
PTC Therapeutics	1,3,4,5

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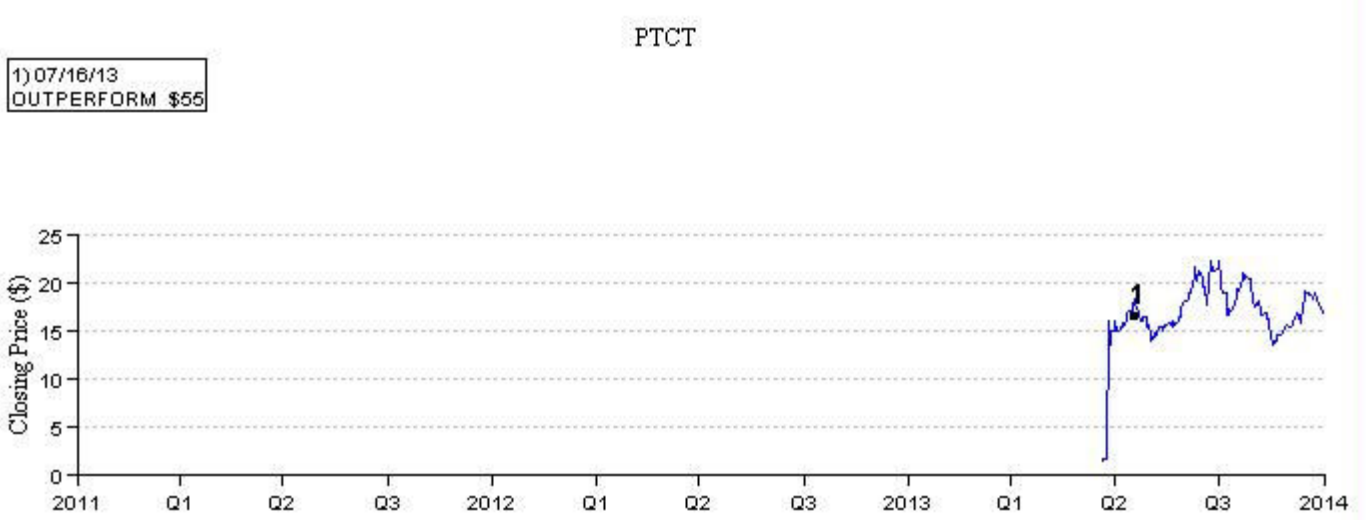

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EQUITY RESEARCH DEPARTMENT

(213) 688-4529

DIRECTOR OF RESEARCH
Mark D. Benson (213) 688-4435

MANAGER, RESEARCH OPERATIONS
Ellen Kang (213) 688-4529

RETAIL AND CONSUMER

Consumer Products
Rommel T. Dionisio (212) 938-9934
Kurt M. Frederick, CFA CPA (415) 274-6822
Alicia Reese (212) 938-9927

Footwear, Apparel and Accessories
Corinna Freedman (212) 668-9876

Healthy Lifestyles
Kurt M. Frederick, CFA CPA (415) 274-6822

Restaurants
Nick Setyan (213) 688-4519
Colin Radke (213) 688-6624

Specialty Retail: Hardlines
Joan L. Storms, CFA (213) 688-4537
John Garrett, CFA (213) 688-4523

Seth Basham, CFA (212) 938-9954

RETAIL/CONSUMER MARKET RESEARCH

Gabriella Santaniello (213) 688-4557

INDUSTRIAL GROWTH TECHNOLOGY

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Craig Irwin (212) 938-9926
Min Xu (212) 938-9925

Environmental Services / Building Products
Al Kaschalk (213) 688-4539
Taryn Kuida (213) 688-4505

Water and Renewable Energy Solutions
David Rose, CFA (213) 688-4319
James Kim (213) 688-4380

TECHNOLOGY, INTERNET, MEDIA & SOCIAL MEDIA

Communications and Application Software
Shyam Patil, CFA (213) 688-8062
Andy Cheng (213) 688-4548

Communications Equipment
Rohit Chopra (212) 668-9871
Sanjit Singh (212) 938-9922
Ryan Flanagan (212) 938-9942

Computer Services: Financial Technology
Gil B. Luria (213) 688-4501
Aaron Turner (213) 688-4429

Enterprise Software
Steve Koenig (415) 274-6801
Kevin Ikeda (213) 688-4423

Entertainment: Retail
Michael Pachter (213) 688-4474
Nick McKay (213) 688-4343
Nick Citrin (213) 688-4495

Entertainment: Software
Michael Pachter (213) 688-4474
Nick McKay (213) 688-4343
Nick Citrin (213) 688-4495

Internet: Media and Gaming
Michael Pachter (213) 688-4474
Nick McKay (213) 688-4343
Nick Citrin (213) 688-4495

Internet: Social Media, Advertising & Technology
Shyam Patil, CFA (213) 688-8062
Andy Cheng (213) 688-4548

Media
James Dix, CFA (213) 688-4315

Movies and Entertainment
Michael Pachter (213) 688-4474
Nick McKay (213) 688-4343
Nick Citrin (213) 688-4495

Semiconductors
Betsy Van Hees (415) 274-6869
Ryan Jue, CFA (415) 263-6669

LIFE SCIENCES AND HEALTH CARE

Biotechnology/Biopharmaceuticals/BioDefense
Gregory R. Wade, Ph.D. (415) 274-6863
David M. Nierengarten, Ph.D. (415) 274-6862
Christopher N. Marai, Ph.D. (415) 274-6861

Emerging Pharmaceuticals
Liana Moussatos, Ph.D. (415) 263-6626
Richard Lau, CFA (415) 274-6851

Healthcare Services - Managed Care
Sarah James (213) 688-4503

Medical Devices
Tao Levy (212) 938-9948

Medical Diagnostics and Life Sciences Tools
Zarak Khurshid (415) 274-6823

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Boston	(617) 832-3700	Boston	(617) 832-3700

CORPORATE HEADQUARTERS
1000 Wilshire Blvd., Los Angeles, CA 90017-2465
Tel: (213) 688-8000 www.wedbush.com