JMP

Atara Biotherapeutics, Inc. (ATRA)

Initiating Coverage at Market Outperform; Atara Fields Two Innovative Platforms for Tackling Serious Diseases

MARKET DATA

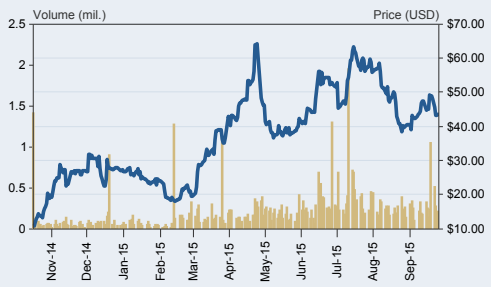
Price	$43.48
52-Week Range:	$9.66 - $65.56
Shares Out. (M):	25.6
Market Cap ($M):	$1,113.1
Average Daily Vol. (000):	224.0
Cash (M):	$349
Cash/Share:	$5.11
Enterprise Value (M):	$1,072
Float (M):	20.0
LT Debt (M):	$0

Source: Thomson Reuters and JMP Securities LLC

FY DEC		2014A	2015E	2016E
Revenue ($M)	1Q	$0.0	$0.0A	--
	2Q	$0.0	$0.0A	--
	3Q	$0.0	$0.0	--
	4Q	$0.0	$0.0	--
	FY	**$0.0**	**$0.0**	**$0.0**
EPS	1Q	--	($0.42)A	--
	2Q	--	($0.62)A	--
	3Q	--	($0.40)	--
	4Q	$0.00	($0.44)	--
	FY	**($1.43)**	**($1.64)**	**($2.13)**
	P/E	NM	NM	NM

Source: Company reports and JMP Securities LLC

STOCK PRICE PERFORMANCE



MARKET OUTPERFORM | Price: $43.48 | Target Price: $61.00

INVESTMENT HIGHLIGHTS

We are initiating coverage on Atara Biotherapeutics (ATRA) with a Market Outperform rating and $61 price target based on a synthesis of our DCF, SOTP, and comparable companies valuation methodologies. Atara is developing two highly differentiated platforms with the potential to tap into vast market segments. Atara's first platform consists of a portfolio of molecular therapeutics, in-licensed from Amgen (AMGN, NC), targeting the biology of transforming growth factor-beta (TGF-β) family members. The lead molecule from this portfolio, PINTA-745, has applications in wasting diseases, such as cancer cachexia and protein energy wasting in dialysis patients. Atara has also recently licensed the rights to a platform of "off the shelf" cytotoxic T-cell (CTL) therapeutics from Memorial Sloan Kettering Cancer Center (MSKCC). These CTLs provide a pathway to rapid approval in rare indications such as Epstein-Barr Virus (EBV) and cytomegalovirus (CMV) mediated lymphomas in transplant patients. Furthermore, they hold the promise of broad anti-cancer activity against tumors expressing viral antigens, such as glioblastoma, and against rare tumor-associated antigens, such as Wilms Tumor 1 (WT1).

ATRA management brings large-cap biotech experience to bear on the company's clinical development candidates. Atara's management has a deep understanding of the biology of TGF-β, with experience accumulated at Amgen and Celgene (CELG, MO, $147 PT). Atara was formed as a spinout from Amgen and Amgen continues to hold a ~5% stake in Atara. The TGF-β portfolio that was licensed from Amgen is supported by a wealth of preclinical studies and a Phase I trial validating the potential of the lead molecule, PINTA-745, in wasting diseases such as cancer cachexia and protein energy wasting in dialysis patients. Atara is currently testing PINTA-745 in a Phase II trial in dialysis patients and has also initiated a Phase I trial with its second candidate, STM-434, for treating cancers driven by TGF-β growth factors. A third candidate, ATA-842, is nearly ready for an IND submission for treating cancer cachexia. The T-cell portfolio provides Atara access to libraries of allogeneic CTLs that have already been extensively validated for treating viral infections in Phase I/II trials. Additional Phase I/II data from trials run by MSKCC will continue to read-out in the final quarter of 2015 and in 2016, while ATRA will move quickly into registration-directed trials with EBV-CTL and CMV-CTL.

Near-term value drivers can launch the company to commercial success as data build in larger markets. While not yet approved by the FDA, EBV-specific CTLs are already included in the National Comprehensive Cancer Network (NCCN) guidelines as a second-line therapy for treating post-transplant lymphoproliferative disorders (PTLD). As a consequence, we see a very high likelihood that Atara will be able to secure FDA approval in this orphan indication following a small, short pivotal trial. We model

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first revenues from this program as soon as 2018, with worldwide sales of ~$200MM by 2025. Atara intends to pursue other larger oncology markets in which viral antigens are expressed, such as glioblastoma (GBM) and Hodgkin's lymphoma. We model Atara achieving revenues of ~$500MM by 2033 assuming the CMV-CTLs are approved for treating GBM.

Setting PEW on a rapid clinical path. Similar to the CTL clinical strategy, we anticipate Atara will seek the fastest route to approval for PINTA-745 to treat protein energy wasting (PEW) in dialysis patients. Using endpoints such as increased muscle mass and physical function tests, we estimate Atara could receive approval for PINTA-745 as early as 2018, allowing the company to establish a presence in the dialysis market. We believe larger outcomes studies, demonstrating improved morbidity and mortality, could allow ATRA to capture an even larger segment of the dialysis population, with peak combined sales for the two indications reaching $3B worldwide. The use of ATA-842 for treating other wasting syndromes, such as cancer cachexia, represents further upside not included in our model.

Atara represents a unique opportunity to invest in a de-risked, late-stage pipeline company. In our opinion, Atara represents a strong buying opportunity with several near-term catalysts and a more diversified portfolio than other CTL-therapy based companies that command a larger market cap, such as Juno Therapeutics (JUNO, NC) and Kite Pharma (KITE, NC). Researchers at MSKCC continue to generate clinical data from the EBV-CTL and CMV-CTL programs, with further trial data anticipated prior to the end of the year. Also, by year-end, Atara expects to release top-line Phase II data from dialysis patients with PEW treated for 12 weeks with PINTA-745. If the results look positive, it will set the stage for registration-directed trials to initiate in 2016. Finally, early 2016 should also bring the first data from the STM-434 program for treating solid tumors, with a focus on ovarian cancers.

INVESTMENT THESIS

Atara Biotherapeutics formed in 2012 as an LLC with the goal of licensing out individual members of the AMGN TGF-β portfolio to interested biopharma companies. However, given the opportunity for value creation via the development of these assets, Atara's management subsequently decided to raise funds and develop the programs internally. Atara also diversified in June 2015 when it exercised an option to license three cytotoxic T-cell programs from MSKCC. Atara's pipeline thus traces its origins to two institutions with world-class pedigrees. In our opinion, ATRA's lead therapeutics are very well validated and de-risked for a clinical-stage biotherapeutics company, particularly one with such a brief corporate history.

The Amgen TGF-β portfolio came with a substantial amount of preclinical work invested and one compound, AMG-745, had even been tested in a Phase I trial where it demonstrated signs of efficacy and an acceptable safety profile. TGF-β family members regulate a wide variety of biological processes, ranging from cell growth to inflammation to tissue patterning. The different members of the portfolio thus exhibit potential applicability in a wide range of disease states. In our view, PINTA-745 represents a particularly attractive asset. PINTA-745 binds to myostatin, a negative regulator of muscle growth. It also exhibits anti-inflammatory characteristics in preclinical models. These dual characteristics make PINTA-745 an attractive therapeutic for treating protein energy wasting (PEW) in dialysis patients. PEW is characterized by inflammation, poor appetite, malnutrition and the gradual loss of muscle mass. Studies have shown that dialysis patients with PEW have much greater morbidity

and mortality. Due to its anti-inflammatory properties and ability to promote muscle growth, PINTA-745 appears to be a logical candidate to test in PEW. In fact, its characteristics seemed so promising that Atara was approached by DaVita Healthcare, one of the largest dialysis providers in the U.S., to collaborate on the PEW program. In our opinion, this collaboration represents not only a validation of PINTA-745, but also gives Atara a competitive advantage in the dialysis space, because of its contacts and its access to DaVita's substantial data resources.

We note that while PINTA-745 represents the largest component of our model, accounting for ~$26 of the $71 price target in our SOTP analysis, STM-434 has significant market potential as well. A number of cancers including ovarian, breast, and prostate are driven by activin signaling. Activin is a TGF-β family member that is bound and sequestered by STM-434. STM-434 inhibited the growth of ovarian cancer cells in preclinical models and is currently in a Phase I trial for solid tumors. We note that STM-434 actually has two mechanisms of action that may promote cancer patient survival. The first is by inhibiting tumor growth, and the second is by inhibiting the morbidity and mortality associated with cancer cachexia. STM-434 accounts for ~$10 of the stock price target in our SOTP analysis, assuming approvals in subsets of ovarian cancer and in breast cancer patients who have progressed after multiple lines of prior therapy. Finally, ATA-842 is another anti-myostatin therapeutic which Atara is currently preparing for IND submission to treat cancer cachexia. This program represents additional upside not currently incorporated in our model.

Atara's cytotoxic T-cell therapy program also comes very well validated, in our view. As mentioned previously, the EBV-CTLs have already been added to the National Comprehensive Cancer Network (NCCN) guidelines for treating EBV mediated post-transplant lymphoproliferative disorders. As a reminder, the NCCN is the recognized authority for cancer treatment protocols for the U,S, and in many other parts of the world. While the CTL space is highly competitive, Atara's advantage lies in the extensive data set, treatment algorithms, and allogeneic T-cell library that MSKCC has developed. Atara's program is also an "off-the-shelf" allogeneic T-cell product. This means that the product can be generated at a substantially lower cost than other personalized immunotherapies, such as CAR-T cells, and can be made rapidly available to the patient, a critical factor in diseases such as EBV mediated PTLD, where the patient may succumb within weeks. While many companies are still working out the logistics of how to effectively treat patients with alloreactive T-cells, MSKCC has already built a bank of cell lines from 300 donors that can effectively treat >95% of potential recipients in the U.S. We think that the ability to build on MSKCC's platform will allow Atara to provide a safe, predictable, competitively priced product that will enable rapid market uptake.

FIGURE 1. Upcoming Milestones

Timing	Drug	Milestones
2H15	EBV-CTL	Data at scientific meetings, venue not disclosed
4Q15	PINTA-745	Ph II -topline data on muscle mass and safety in ESRD PEW
1H16	STM-434	Ph I -dose escalation data in solid tumors
2H16	STM-434	Ph I -initiation of dose escalation in ovarian cancer
1H16	PINTA-745	Ph II -full data including 8 week follow up ESRD PEW
1H16	PINTA-745	Ph I -dose escalation data in solid tumors
2H16	STM-434	Ph I -initiation of dose escalation in ovarian cancer

Source: ATRA presentations

VALUATION

We arrive at our $61 price target based on the synthesis of our discounted cash flow (DCF), sum-of-the-parts analysis, and a relative valuation against a set of comparable biotechnology platform companies (Figure 2).

FIGURE 2. Price Target Synthesis

Synthesis of Valuation Approaches		
Approach	Weight	Valuation
DCF Analysis	33%	$48.00
Comparables	33%	$76.00
SOTP	33%	$62.00
Price Target		**$61.00**

Source: JMP Securities LLC and Company Reports

Our DCF valuation projects worldwide sales of PINTA-745 for PEW, STM-434 in recurrent ovarian and breast cancers, EBV-CTL and CMV-CTL in PTLD and GBM, and WT1-CTL in AML out to 2033 while subtracting COGS, projected operating expense, and tax. ATRA is also obligated to pay up to $86MM in developmental and commercial milestones and two tiered, mid-single-digit royalties to Amgen and an additional $33MM in milestones and low double-digit percentage royalties to MSKCC. Net cash flows to the company are discounted back to 2015 year end, by an integrated, risk-adjusted, discount rate of 30% that reflects the promising activity exhibited by Atara's most advanced clinical candidates, in Phase I and Phase II clinical trials. A terminal value for the company, calculated by applying a -20% long-term growth rate, was similarly discounted to present day. Present value of free cash flows, together with the terminal value, were added to arrive at a residual value for the company, to which estimated cash and long-term debt were added and subtracted, respectively. We thereby arrive at an equity valuation of $1,400MM. Dividing this by our estimated 2015 year-end outstanding share count, we derive a per share valuation of ~$48. Our DCF assumptions are detailed further in Figure 3.

FIGURE 3. Discounted Cash Flow Valuation

Atara Biotherapeutics (ATRA) Atara Net Revenues	2014A	1Q15A	2Q15E	3Q15E	4Q15E	2015E	2016E	2017E	2018E	2019E	2020E	2021E	2022E	2023E	2024E	2025E	2026-2033
Sales																	
Total Product Sales and Royalties	*0.00*	0.00	0.00	0.00	0.00	0.00	0.00	0.00	18.45	201.25	581.30	1,095.58	1,806.32	2,778.75	3,739.26	4,649.99	
Milestone Revenues	*0.00*	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	
Additional Revenues	*0.00*	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	
Total Revenues	**$ -**					**$ -**	**$ -**	**$ -**	**$ 18**	**$ 201**	**$ 581**	**$ 1,096**	**$ 1,806**	**$ 2,779**	**$ 3,739**	**$ 4,650**	
Royalties and Milestones Paid						0.50	2.50	1.00	4.13	16.62	48.53	84.05	128.37	184.28	242.23	293.28	
Cost of goods sold	*0.00*	0.00	0.00	0.00	0.00	0.00	0.00	0.00	2.77	30.19	87.19	164.34	270.95	416.81	560.89	637.41	
COGS as % of revenue									15%	15%	15%	15%	15%	15%	15%	14%	
Gross Profit	*0.00*	0.00	0.00	0.00	0.00	(0.50)	(2.50)	(1.00)	11.56	154.45	445.57	847.19	1,407.00	2,177.65	2,936.13	3,719.31	
Total R&D	*14.38*	5.77	7.01	8.25	9.49	30.51	77.64	150.45	204.72	220.70	202.39	184.98	196.58	205.84	222.92	244.01	
R&D as a % of revenue										110%	35%	17%	11%	7%	6%	5%	
Total SG&A	*12.71*	3.54	3.60	3.66	3.72	14.52	16.33	18.13	24.94	65.89	90.19	103.73	150.62	185.84	205.94	220.62	
SG&A as a % of revenue									135%	33%	16%	9%	8%	7%	6%	5%	
Total operating expenses	*27.09*	9.31	10.61	11.91	13.20	45.03	93.96	168.58	229.66	286.59	292.58	288.71	347.20	391.68	428.86	464.63	
Operating income (EBIT)	*(27.09)*	(9.31)	(10.61)	(11.91)	(13.20)	(45.53)	(96.46)	(169.58)	(218.10)	(132.13)	153.00	558.49	1,059.80	1,785.97	2,507.27	3,254.68	
Operating margin (%)										-66%	26%	51%	59%	64%	67%	70%	
Taxes	*0.00*	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	83.77	264.95	535.79	877.55	1,139.14	
Tax rate	*0%*	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	15%	25%	30%	35%	35%	
After tax operating income	*(27.09)*	(9.31)	(10.61)	(11.91)	(13.20)	(45.53)	(96.46)	(169.58)	(218.10)	(132.13)	153.00	474.71	794.85	1,250.18	1,629.73	2,115.54	
Growth rate	*#DIV/0!*	-66%	14%	12%	11%	245%	112%	76%	29%	-39%	-216%	210%	67%	57%	30%	30%	
Discount year	*0.00*					0.00	0.50	1.50	2.50	3.50	4.50	5.50	6.50	7.50	8.50	9.50	
Discount factor	*1.00*					1.0	1.1	1.5	1.9	2.5	3.3	4.2	5.5	7.2	9.3	12.1	
PV	(27.1)					(45.5)	(84.6)	(114.4)	(113.2)	(52.7)	47.0	112.1	144.4	174.7	175.2	175.0	610.7
													Terminal Value		46.6		

Residual value of cash flow	1,075.3
+ Cash and Cash equivalents	343
Company value	1,418
- Long-term debt on 12/31/15	0
Value of equity	$1,418
Fully diluted shares outstanding on 12/31/15	29.8
Price/share	**$48.0**
Discount Rate	30.0%
Terminal growth rate	-20%

Source: JMP Securities LLC and Company Reports

We also derived a valuation based on our sum-of-the-parts methodology, incorporating worldwide sales from the ten indications where we anticipate Atara will gain approval. NPV contributions from PINTA-745, STM-434, and the CTL program are detailed in Figures 4, 5, 6 and 7. Utilizing a SOTP analysis, we arrive at a $62 per share value.

FIGURE 4. Sum-of-the-Parts Analysis

Sum of Parts Atara NPV	WW	US	Ex-US
PINTA-745 in dialysis ESRD	**$ 22.09**	$ 11.52	$ 10.57
STM-434	**$ 7.95**	$ 5.35	$ 2.60
CTL	**$ 20.55**	$ 11.21	$ 9.34
Cash and Equivs on Hand	**$ 11.51**		
Total NPV	**$ 62.11**	**$ 16.87**	**$ 13.18**

Source: JMP Securities LLC and Company Reports

FIGURE 5. SOTP Detailed Analysis of PINTA-745

PINTA-745 in dialysis ESRD		2015	2016	2017	2018	2019	2020	2021	2022	2023	2024	2025	2026
US Sales ($MM)		$ -	$ -	$ -	$ 12	$ 150	$ 255	$ 367	$ 512	$ 715	$ 889	$ 953	$ 997
Contribution Margin			*0%*	*-400%*	*-600%*	*-10%*	*25%*	*60%*	*57%*	*49%*	*45%*	*45%*	*45%*
Operating Margin			**0.0**	**0.0**	**(71.9)**	**(15.0)**	**63.8**	**220.1**	**292.1**	**350.5**	**399.8**	**429.1**	**448.6**
Terminal Value													**1,661.3**
Discount Period			0.5	1.5	2.5	3.5	4.5	5.5	6.5	7.5	8.5	9.5	10.5
PV			**0.0**	**0.0**	**(37.3)**	**(6.0)**	**19.6**	**52.0**	**53.1**	**49.0**	**43.0**	**35.5**	**134.2**
Discount Rate	30%												
Terminal Growth	3%												
NPV	**$ 343.06**												
# Shares outstanding (mm)	29.8												
Incremental price per share	**$ 11.52**												
EU Sales ($MM)			$ -	$ -	$ -	$ 4	$ 47	$ 82	$ 144	$ 220	$ 281	$ 336	$ 366
Contribution Margin			*0%*	*-400%*	*-600%*	*-10%*	*25%*	*60%*	*57%*	*49%*	*45%*	*45%*	*45%*
Operating Margin			**0.0**	**0.0**	**0.0**	**(0.4)**	**11.7**	**49.3**	**82.3**	**107.7**	**126.5**	**151.0**	**164.8**
Terminal Value													**610.3**
Discount Period			0.5	1.5	2.5	3.5	4.5	5.5	6.5	7.5	8.5	9.5	10.5
PV			**0.0**	**0.0**	**0.0**	**(0.2)**	**3.6**	**11.7**	**15.0**	**15.1**	**13.6**	**12.5**	**49.3**
Discount Rate	30%												
Terminal Growth	3%												
NPV	**$ 120.51**												
# Shares outstanding (mm)	29.8												
Incremental price per share	**$ 4.05**												
ROW Sales ($MM)			$ -	$ -	$ -	$ -	$ 9	$ 38	$ 118	$ 278	$ 427	$ 714	$ 815
Contribution Margin			*0%*	*-400%*	*-600%*	*-10%*	*25%*	*60%*	*57%*	*49%*	*45%*	*45%*	*45%*
Royalty to Acceleron			**0.0**	**0.0**	**0.0**	**0.0**	**2.3**	**22.8**	**67.3**	**136.3**	**192.0**	**321.3**	**366.9**
Terminal Value													**1,358.7**
Discount Period			0.5	1.5	2.5	3.5	4.5	5.5	6.5	7.5	8.5	9.5	10.5
PV			**0.0**	**0.0**	**0.0**	**0.0**	**0.7**	**5.4**	**12.2**	**19.0**	**20.6**	**26.6**	**109.8**
Discount Rate	30%												
Terminal Growth	3%												
NPV	**$ 194.36**												
# Shares outstanding (mm)	29.8												
Incremental price per share	**$ 6.53**												

Source: JMP Securities LLC and Company Reports

FIGURE 6. SOTP Detailed Analysis of STM-434

STM-434		2014	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024	2025	2026
US Sales ($MM)			$ -	$ -	$ -	$ -	$ 2	$ 8	$ 20	$ 65	$ 236	$ 442	$ 652	$ 740
Contribution Margin				*0%*	*-400%*	*-600%*	*-10%*	*25%*	*60%*	*57%*	*49%*	*45%*	*45%*	*45%*
Operating Margin				**0.0**	**0.0**	**0.0**	**(0.2)**	**2.1**	**11.8**	**36.8**	**115.7**	**198.8**	**293.3**	**332.8**
Terminal Value														**1,040.0**
Discount Period				0.5	1.5	2.5	3.5	4.5	5.5	6.5	7.5	8.5	9.5	10.5
PV				**0.0**	**0.0**	**0.0**	**(0.1)**	**0.6**	**2.8**	**6.7**	**16.2**	**21.4**	**24.3**	**87.3**
Discount Rate	35%													
Terminal Growth	3%													
NPV	**$ 159.19**													
# Shares outstanding (mm)	29.8													
Incremental price per share	**$ 5.35**													
EU Sales ($MM)				$ -	$ -	$ -	$ -	$ 3	$ 14	$ 28	$ 63	$ 145	$ 244	$ 343
Contribution Margin				*0%*	*-400%*	*-600%*	*-10%*	*25%*	*60%*	*57%*	*49%*	*45%*	*45%*	*45%*
Operating Margin				**0.0**	**0.0**	**0.0**	**0.0**	**0.8**	**8.3**	**15.8**	**30.7**	**65.3**	**110.0**	**154.1**
Terminal Value														**481.7**
Discount Period				0.5	1.5	2.5	3.5	4.5	5.5	6.5	7.5	8.5	9.5	10.5
PV				**0.0**	**0.0**	**0.0**	**0.0**	**0.2**	**2.0**	**2.9**	**4.3**	**7.0**	**9.1**	**40.5**
Discount Rate	35%													
Terminal Growth	3%													
NPV	**$ 65.93**													
# Shares outstanding (mm)	29.8													
Incremental price per share	**$ 2.21**													
Japan Sales ($MM)				$ -	$ -	$ -	$ -	$ -	$ 1	$ 7	$ 14	$ 24	$ 40	$ 60
Contribution Margin				*0%*	*-400%*	*-600%*	*-10%*	*25%*	*60%*	*57%*	*49%*	*45%*	*45%*	*45%*
Royalty to Acceleron				**0.0**	**0.0**	**0.0**	**0.0**	**0.0**	**0.9**	**3.9**	**6.9**	**10.7**	**18.2**	**26.9**
Terminal Value														**84.2**
Discount Period				0.5	1.5	2.5	3.5	4.5	5.5	6.5	7.5	8.5	9.5	10.5
PV				**0.0**	**0.0**	**0.0**	**0.0**	**0.0**	**0.2**	**0.7**	**1.0**	**1.2**	**1.5**	**7.1**
Discount Rate	35%													
Terminal Growth	3%													
NPV	**$ 11.61**													
# Shares outstanding (mm)	29.8													
Incremental price per share	**$ 0.39**													

Source: JMP Securities LLC and Company Reports

FIGURE 7. Detailed Analysis of CTL Program

CTL		2014	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024	2025	2025
US Sales ($MM)			$ -	$ -	$ -	$ 6	$ 40	$ 220	$ 364	$ 522	$ 715	$ 836	$ 893	$ 936
Contribution Margin				*0%*	*-400%*	*-600%*	*-10%*	*25%*	*60%*	*57%*	*49%*	*45%*	*45%*	*45%*
Operating Margin				**0.0**	**0.0**	**(38.8)**	**(4.0)**	**55.0**	**218.3**	**297.7**	**350.3**	**376.0**	**401.9**	**421.0**
Terminal Value														**1,315.7**
Discount Period				0.5	1.5	2.5	3.5	4.5	5.5	6.5	7.5	8.5	9.5	10.5
PV				**0.0**	**0.0**	**(20.1)**	**(1.6)**	**16.9**	**51.6**	**54.1**	**49.0**	**40.4**	**33.2**	**110.5**
Discount Rate	35%													
Terminal Growth	3%													
NPV	**$ 333.94**													
# Shares outstanding (mm)	29.8													
Incremental price per share	**$ 11.21**													
ROW Sales ($MM)			$0.0	$ -	$ -	$ -	$ 6	$ 38	$ 210	$ 410	$ 538	$ 697	$ 817	$ 866
Contribution Margin				*0%*	*-400%*	*-600%*	*-10%*	*25%*	*60%*	*57%*	*49%*	*45%*	*45%*	*45%*
Operating Margin				**0.0**	**0.0**	**0.0**	**(0.6)**	**9.6**	**125.8**	**233.8**	**263.5**	**313.4**	**367.7**	**389.8**
Terminal Value														**1,218.2**
Discount Period				0.5	1.5	2.5	3.5	4.5	5.5	6.5	7.5	8.5	9.5	10.5
PV				**0.0**	**0.0**	**0.0**	**(0.2)**	**3.0**	**29.7**	**42.5**	**36.8**	**33.7**	**30.4**	**102.3**
Discount Rate	35%													
Terminal Growth	3%													
NPV	**$ 278.16**													
# Shares outstanding (mm)	29.8													
Incremental price per share	**$ 9.34**													

Source: JMP Securities LLC and Company Reports

Finally, by taking the mean market cap valuation from a peer group of TGF-β and CTL therapeutic platform based on biotechnology companies, we derive a comparable valuation for ATRA of ~76 (Figure 8).

FIGURE 8. Comparable Company Valuation

Comparable Companies						
Comparable	Ticker	Price	Market Cap	Cash	Debt	EV
Aduro BioTech Inc	ADRO	$21.46	$1,336	$119	$0	$1,217
Agios Pharmaceuticals Inc	AGIO	$88.89	$3,328	$342	$0	$2,986
Akebia Therapeutics Inc	AKBA	$11.18	$323	$109	$0	$214
Alnylam Pharmaceuticals Inc	ALNY	$93.30	$7,895	$697	$0	$7,198
Bellicum Pharmaceuticals Inc	BLCM	$17.20	$456	$192	$0	$264
bluebird bio Inc	BLUE	$113.37	$4,111	$474	$0	$3,638
Celldex Therapeutics Inc	CLDX	$13.30	$1,311	$201	$0	$1,110
Cellectis	CLLS	$31.68	$932	$136	$0	$796
FibroGen Inc	FGEN	$27.95	$1,693	$180	$114	$1,627
Juno Therapeutics Inc	JUNO	$38.41	$3,862	$436	$0	$3,427
Kite Pharma Inc	KITE	$61.59	$2,694	$367	$0	$2,327
Lion Biotechnologies Inc	LBIO	$7.00	$330	$45	$0	$285
Nantkwest Inc	NK	$17.23	$1,398	$59	$0	$1,339
Puma Biotechnology Inc	PBYI	$88.27	$2,852	$141	$0	$2,711
Threshold Pharmaceuticals Inc	THLD	$4.79	$342	$59	$0	$283
TRACON Pharmaceuticals Inc	TCON	$12.77	$155	$35	$9	$128
Acceleron Pharma Inc	XLRN	$30.93	$1,023	$176	$0	$846
Intrexon Corp	XON	$45.38	$5,201	$116	$13	$5,098
Ziopharm Oncology Inc	ZIOP	$12.96	$1,696	$43	$0	$1,653
Average			$2,155			$1,955
Atara Biotherapeutics Inc	ATRA	$43.05	$1,227	$161	$0	$1,067

Comparable Valuation	$76.00

Source: JMP Securities LLC, Thomson Reuters

INVESTMENT RISKS

Clinical. Drug development is an inherently risky business, requiring significant investment of both time and capital. The company's clinical-stage candidates (PINTA-745, STM-434, EBV-CTL and CMV-CTL) could fail to achieve positive efficacy results, or might exhibit safety signals that preclude further clinical development. Such scenarios could decrease ATRA's innate value and adversely impact our valuation.

Regulatory and commercial. The ability of Atara to market its drugs depends upon those drugs obtaining approval from the FDA and foreign regulatory agencies. Failure to achieve approval or delays in the timelines to approval could negatively impact the company's share price.

Competitive. The development of cachexia and cancer therapeutics is intensely competitive and is dominated by biotechnology and pharmaceutical companies with expertise and resources that may be greater than those of Atara. There are direct competitors working on the same mechanism of action (Acceleron Pharma (XLRN, MO, PT $58), Pfizer (PFE, NC), Bristol-Myers Squibb (BMS, NC), Eli Lilly (LLY, NC) and Regeneron Pharmaceuticals (REGN, NC)), as well as companies working on distinct therapeutic modalities that could impact overlapping patient populations (such as Kite Pharma, Juno Therapeutics, and Lion Biotechnologies (LBIO, NC)).

Financial. Atara is currently well-funded, with $348.9MM in cash and cash equivalents; however, the company may require additional equity financing, in the form of a secondary offering, to complete the development of its drug candidates. The terms of any potential partnership deals remain unknown at this time, exposing existing shareholders to an uncertain level of dilution risk.

Legal. Atara was formed as a spin out from Amgen (AMGN, NC) and the products licensed from Amgen have strong IP claims through 2032. A number of academic institutions have conducted research on allogeneic cellular therapy platforms. Atara's intellectual property in this space will be based on the proprietary algorithms developed by Memorial Sloan Kettering for administering the T-cells from the allogeneic cell bank and trade secrets and know how.

COMPANY OVERVIEW

Atara Biotherapeutics was founded as a spinoff from Amgen, bringing along six early-stage therapeutics with the potential to modulate transforming growth factor-beta (TGF-β) biology. The TGF-β family participates in a broad array of biological processes, including tissue growth, immune cell function, and cancer biology. Atara's most advanced program is PINTA-745, which is currently in a Phase II trial for treating muscle loss associated with protein energy wasting (PEW) in dialysis patients. PINTA-745 has already demonstrated signs of efficacy in a Phase I trial for treating muscle wasting in prostate cancer patients undergoing androgen deprivation therapy. With top-line results anticipated by the end of 2015, Atara could move to a Phase III trial as soon as 2016.

Atara's second program targeting TGF-β family members is STM-434, which is currently being tested for anti-tumor effects in a Phase I trial. Atara recently diversified its portfolio by licensing the rights to several allogeneic cell therapy based platforms from the Memorial Sloan Kettering Cancer Center (MSKCC). These adoptive cell transfer therapies already have substantial clinical evidence demonstrating their effectiveness for treating Epstein Barr virus and cytomegalovirus infections in immunocompromised patients, but also exhibit broad potential as cancer therapeutics.

TARGETING THE TRANSFORMING GROWTH FACTOR-BETA PATHWAY

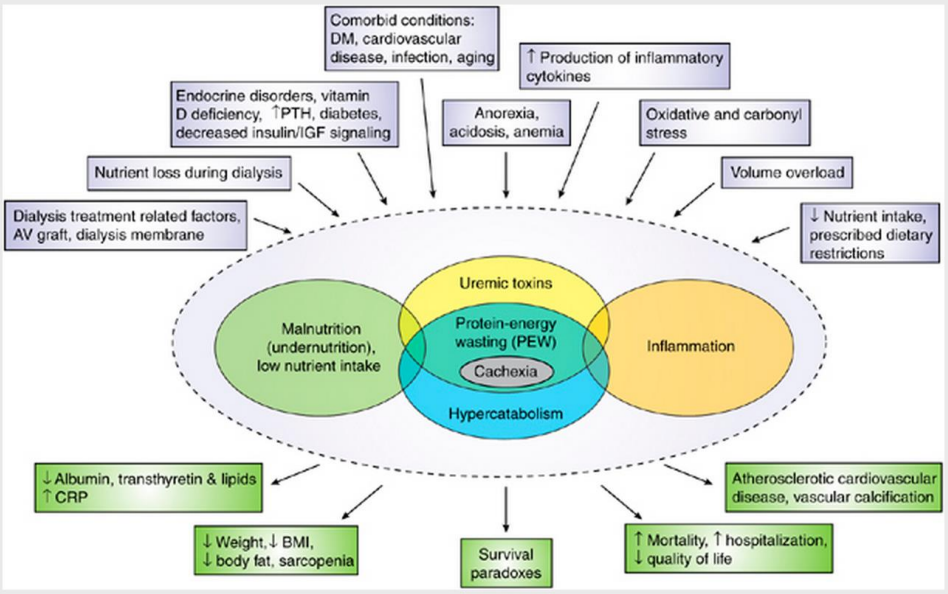
PINTA-745 For Treating Protein Energy Wasting (PEW)

Background

PINTA-745 is a peptide-antibody fusion (peptibody) with a human Fc domain at the N-terminus and a myostatin-neutralizing bioactive peptide at the C terminus. Myostatin is a member of the TGF-β family of molecules that negatively regulates muscle growth. PINTA-745 was initially developed by Amgen (AMGN, NC) as AMG-745, before being licensed to Atara. AMGN conducted a Phase I trial to see if AMG-745 could promote muscle growth and lean body mass in prostate cancer patients receiving androgen deprivation therapy. This population is particularly susceptible to muscle wasting and increased body fat due to the role of androgens, such as testosterone, in promoting muscle growth. The study demonstrated that PINTA-745 is capable of inducing a statistically significant increase in muscle mass after just one month of treatment even in a hormonal context that is not conducive to muscle growth. Atara is now testing PINTA-745 in a Phase II trial to see if it can reverse the morbidity and mortality associated with protein energy wasting (PEW) in patients with end-stage renal disease (ESRD).

Syndromes of wasting, inflammation, and malnutrition are associated with a number of chronic conditions including ESRD, cancer, cardiovascular disease, infection and malnutrition. Cachexia constitutes the most extreme form, characterized by marked loss of weight, muscle atrophy, and fatigue. Protein energy wasting (PEW) was the term adopted by the International Society of Renal Nutrition and Metabolism (ISRNM) to describe the perturbations in nutrition, metabolism, and inflammation experienced by patients with chronic kidney disease (CKD) or acute kidney injury (AKI) (Fouque, Kidney Int., 2007). Multiple mechanisms contribute to the development of PEW, including toxic uremia, endocrine disorders, inflammation, anorexia due to loss of appetite, loss of proteins and nutrients during dialysis, and hypercatabolism (Figure 9). Reliable criteria for diagnosing PEW include reductions in body mass, muscle mass, dietary intake and low serum levels of albumin, transthyretin, or cholesterol.

FIGURE 9. Protein Energy Wasting in End-Stage Renal Disease



Schematic representation of the causes and manifestations of the protein–energy wasting syndrome in kidney disease.

Source: Fouque, Kidney Int., 2007

While PEW is a complex disease, there are a number of pieces of data suggesting that muscle loss is intimately associated with the pathology. Frailty, as measured by weight loss, weakness, slow gait speed, exhaustion, and low physical activity, correlates with increased morbidity and mortality in dialysis patients (Kim, J. Am. Soc. Nephrol., 2013; McAdams-DeMarco, J. Am. Geriat. Soc., 2013). Numerous studies have attempted to counter the effects of frailty through the introduction of exercise programs, either during or between dialysis cycles. These programs have demonstrated statistically significant improvements in cardiopulmonary fitness, endurance, muscle strength, physical function and patient quality of life (Jung, CMJ, 2011). However, an acknowledged limitation of these studies is that they tend to include the youngest, healthiest, most active patients. Therefore a large unmet need remains for therapies that can benefit the majority of dialysis patients who start from a poor physical conditioning baseline and often have comorbidities that can limit fitness training.

If the Phase II trial of PINTA-745 in dialysis patients meets its primary endpoint of increasing muscle mass and demonstrates promising trends in the secondary endpoints assessing physical function, Atara anticipates seeking FDA approval based on these same endpoints. While this would provide the fastest route to approval, we believe the greater market potential for PINTA-745 lies in the possibility that it might reduce morbidity and mortality in PEW patients. As discussed below, there are three lines of evidence that suggest PINTA-745 could achieve this goal. First, stemming the loss of muscle mass may in and of itself reduce morbidity and mortality; second, in preclinical models, PINTA-745 suppressed levels of inflammatory cytokines believed to contribute to the etiology of PEW; third, by increasing muscle mass, PINTA-745 may improve glucose regulation and insulin responsiveness in the sizable diabetic dialysis population.

In contrast to what is observed in the general population, ESRD patients with higher body mass indices (BMIs) have better survival outcomes. This phenomenon is referred to as the obesity paradox. However, research suggests that BMI, per se, may be less important than the underlying loss of protein stores. Albumin is the most abundant protein in blood serum, while skeletal muscle is the largest organ in the body, accounting for ~40% of body mass. Creatinine is a chemical waste product generated by muscle metabolism, and circulating plasma levels can serve as an indirect measure of muscle mass. Studies of ESRD patients on dialysis have demonstrated that lower levels of albumin and/or creatinine correlate with worse mental and physical health scores and increased morbidity and mortality (Lacson, Hemodial. Int., 2010; Feroze, ASN, 2011; Park, Mayo Clin. Proc., 2013). Reduced creatinine levels have been found to correlate more closely with adverse outcomes than weight loss, consistent with the hypothesis that obesity is largely protective to the extent that it correlates with underlying muscle mass (Kalantar-Zadeh, Am. J. Epidemiol., 2012).

Together, these data suggest that loss of muscle mass contributes to the etiology of PEW and conversely that therapeutic intervention to reverse the course of muscle loss might improve patient outcomes. Moreover, ESRD patients have an average of four-fold higher circulating myostatin levels than healthy controls (Garcia, Anesthesia and Analgesia, 2010), indicating that myostatin may play a direct role in the loss of muscle as observed in PEW. PINTA-745 thus seems like a logical candidate to test in ESRD patients.

PEW in CKD, and especially ESRD patients, is characterized by the presence of both pro- and anti-inflammatory cytokines that are often several fold higher than in healthy individuals. Chronic low grade inflammation appears to contribute to numerous adverse conditions in ESRD patients including increased infection rates, cardiovascular disease and the development of muscle wasting (Carrero et al., Clin. Nutrition, 2008). Pro-inflammatory cytokines such as interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF-α) both suppress appetite and stimulate the breakdown of muscle protein (Susuki, JJCO, 2015). There is intense interest in therapeutic agents that can ameliorate the complications associated with PEW by treating the underlying immune dysregulation. TGF-β family members play critical roles in modulating the immune system. In mouse models of CKD, TNF-α upregulates myostatin levels, which, in turn, induces the expression of IL-6 (Zhang, FASEB, 2013). Findings from these studies indicate that a mouse version of PINTA-745 not only increases muscle mass in treated animals, but also suppresses inflammatory cytokines such as TNF-α and IL-6 (Zhang, FASEB, 2011). Coupled with its effect on muscle growth, these data make PINTA-745 a particularly compelling candidate for treating PEW in ESRD patients.

Diabetes is the most common cause of kidney failure, accounting for nearly 40% of all new cases (U.S. Renal Data System, 2011). Skeletal muscle is the major site of glucose uptake in humans, and skeletal muscle insulin resistance is considered to be the initiating or primary defect leading to the development of type 2 diabetes. There is a substantial body of evidence indicating that increasing muscle mass through exercise can help control glucose levels, resulting in less insulin production, which, in turn, reduces insulin resistance. Muscle wasting due to PEW thus aggravates the underlying conditions leading to type II diabetes. PINTA-745 could potentially benefit all patients with type II diabetes; however, the case is particularly compelling in dialysis patients, where the risk of hypoglycemia is higher than in non-dialysis patients. This has been documented in the context of variable dietary intake, such as when patients fail to eat prior to dialysis (Berns, Up To Date, 2015). Given the interplay between PEW and diabetes, it seems reasonable that treating dialysis patients with PINTA-745 for PEW could result in a decrease in diabetes-associated morbidity and mortality.

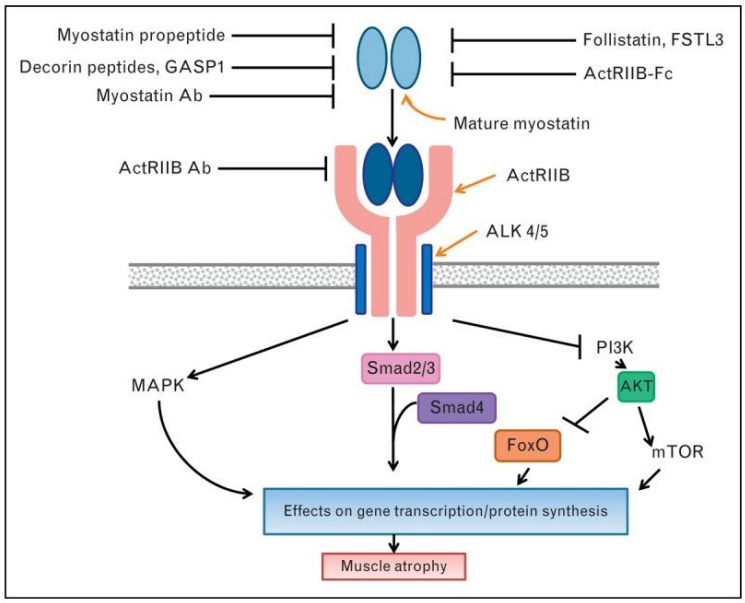
Treatment options

There are no drugs currently approved specifically for the treatment of muscle wasting or PEW. Therapeutic modalities including appetite stimulants, corticosteroids, anabolic steroids, and human growth hormone have demonstrated limited utility and can cause significant systemic side effects. Studies indicate that modulating diet and exercise can improve clinical outcomes; however, compliance remains an issue in a population that is frail and prone to anorexia. We spoke with KOLs who expressed a keen interest in therapeutics that could counter the muscle loss effects. Such a therapeutic could potentially work synergistically with a physical therapy program by improving the patients' ability to successfully execute an exercise program.

PINTA-745 mechanism of action

Myostatin is a protein both secreted and detected by muscle cells (referred to as autocrine signaling) to inhibit their own growth. Myostatin binds with high affinity to the activin receptor IIB (ActRIIB) and with low affinity to the activin receptor IIA (ActRIIA). Ligating these receptors results in the recruitment of the activin receptor-like kinases 4 and 5 (ALK-4,5) whose signaling inhibits the phosphatidylinositol-3 kinase (PI3K) and Wnt4/β-catenin pathways while triggering mitogen activated protein kinases (MAPK) and the SMAD transcription factors. Downstream events triggered by this signaling cascade include upregulation and activation of the ubiquitin proteolytic system (resulting in increased protein degradation), generalized suppression of protein synthesis, and downmodulation of myogenesis promoting genes, such as myogenin and Pax7 (Figure 10, 2013_COSPC).

FIGURE 10. Myostatin Signaling Pathway



Source: Smith, Current Opinion Supportive, and Palliative Care, 2013

Animals and humans with inactivating mutations or deletions of the myostatin gene exhibit increased skeletal muscle growth. Conversely, adult mice genetically engineered to overexpress myostatin experience muscle wasting. Myostatin is produced and secreted as a latent complex with a non-covalently bound propeptide. Proteolytic cleavage of the propeptide is required for myostatin activation. Overexpression of the propeptide in murine models results in sequestration of free myostatin, and consequently, stimulates the growth of increased muscle mass. This peptide served as the basis for the development of the AMG-745 peptibody.

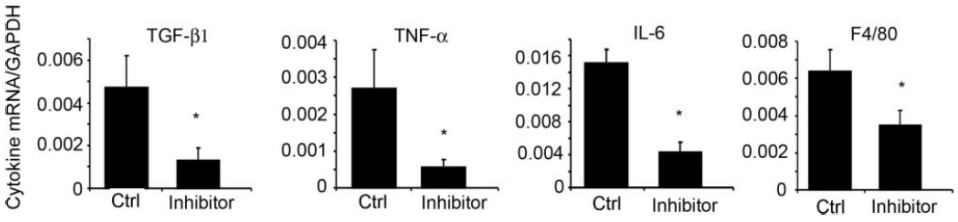
In a murine model of chronic kidney disease, a mouse version of PINTA-745 suppressed muscle atrophy (Figure 11) and suppressed inflammatory cytokines characteristic of PEW in the muscles of mice after just seven days of treatment (Figure 12).

FIGURE 11. A Mouse Form of PINTA-745 Maintains Muscle Mass in an Induced Model of Kidney Disease

	Muscle weight (mg)			
Treatment	tibiae anterior	Gastrocnemius	EDL	Soleus
Inhibitor	61 ± 1.57	174.20 ± 5.39	13.75 ± 0.78	11.08 ± 0.77
PBS	56 ± 2.37	157.80 ± 2.88	11.67 ± 0.17	9.33 ± 1.01
P value	0.037*	0.027*	0.038*	0.016*
Values are means ± se. *P < 0.05. EDL: extensor digitorum longus				

Source: Zhang, FASEB, 2011

FIGURE 12. A Mouse Form of PINTA-745 Suppresses Inflammatory Cytokines in the Muscles of Treated Mice With Kidney Disease



Source: Zhang, FASEB, 2011

Evaluation of clinical data

The Phase I study for PINTA-745 (AMG-745) was conducted in patients receiving androgen deprivation therapy for prostate cancer. Androgens are steroidal hormones that play a central role in promoting skeletal muscle growth. Thus, while many kinds of cancer induce cachexia, prostate cancer patients undergoing androgen deprivation therapy are particularly subject to skeletal muscle wasting and represent a stringent population within which to test PINTA-745.

In this trial 54 patients received subcutaneous doses of 0.3mg/kg, 1mg/kg, or 3mg/kg once weekly for four weeks. Eight patients, randomized 3:1 PINTA-745 to placebo, were enrolled in the 0.3mg/kg dose cohort or in the 1mg/kg dose cohort. Thirty-eight patients, randomized 1:1, enrolled in the 3mg/kg dose cohort. Physical exams, hematological labs, and urinalysis were conducted on all patients prior to treatment, at day 29, and one month after treatment. Patients in the 3mg/kg cohort were also screened by dual X-ray absorptiometry (DXA), to determine fat body mass, and lower extremity computed tomography (CT) scans, to measure muscle cross-sectional area (leg muscle). For these patients lower extremity strength was assessed based on maximum weight lifted using a knee extension machine, standing balance, timed walk test, and five repetitions of chair stand.

Patient populations appeared well balanced between the cohorts. The average age was 73.3 years (cohort means ranged from 71.8 to 73.5 years). The whole body lean mass of 53.4 kg (cohort means ranged from 52.1 kg to 56.6 kg) and baseline muscle cross-sectional area of 1173 cm2 (cohort means ranged from 1147.6 cm2 to 1240.2 cm2) also looked comparable.

The trial demonstrated statistically significant differences between patients who received PINTA-745 or placebo for several parameters. At day 29, PINTA-745 treated patients had a 1.5 ± 0.5% increase in lean body mass, whereas placebo-treated patients exhibited a loss of 0.7 ± 0.5% ($p \leq 0.008$). This difference was sustained at day 58, by which time treated patients had achieved a 1.9 ± 0.5% gain while placebo patients remained lower at 0.2 ± 0.5% ($p \leq 0.023$). Lower extremity muscle size narrowly missed statistical significance at 29 days, with treated patients exhibiting 1.2 ± 0.7% growth vs. a 0.7 ± 0.7% loss for placebo patients ($p \leq 0.065$). However, consistent with the pharmacokinetic data, which indicated that significant amounts of PINTA-745 remained in circulation at day 28, treated patients continued to demonstrate increases in lower extremity muscle size. At day 58, patients injected with PINTA-745 had achieved a 2.7 ± 0.7% increase in size while placebo-treated patients exhibited a 0.1 ± 0.7% loss ($p \leq 0.007$; Figure 13). Patients did not demonstrate improvements in strength tests, which may reflect the relatively short nature of the study.

FIGURE 13. PINTA-745 Increased Lean Body Mass and Muscle Size in Prostate Cancer Patients

Percentage Change From Baseline for Lean Body Mass, Whole-Body Fat, and Lower-Extremity Muscle Size

End point: % change from baseline to day 29¤	Lean Body Mass*	Whole-Body Fat§,‡	Lower-extremity Muscle Size˚
AMG 745	1.5% (0.5%)	-1.7% (0.7%)	1.2% (0.7%)
Placebo	-0.7% (0.5%)	0.8% (0.7%)	-0.7% (0.7%)
Between-group difference	2.2% (0.8%)	-2.5% (1.0%)	1.8% (1.0%)
P value	0.008	0.021	0.065
End point: % change from baseline to follow-up day 58¤			
AMG 745	1.9% (0.5%)	-1.5% (1.1%)	2.7% (0.7%)
Placebo	0.2% (0.5%)	0.5% (1.1%)	-0.1% (0.7%)
Between-group difference	1.7% (0.7%)	-2.0% (1.5%)	2.8% (1.0%)
P value	0.023	0.183	0.007

* As assessed by DXA scan. § Prespecified exploratory analysis. ‡ As assessed by CT scan. ˚ Values are least squares mean (SE), except P values.¤ Lean body mass (minus the head), as assessed by DXA scan.

Source: Padhi, JCEM, 2014

The adverse event profile looked generally acceptable. All adverse events were categorized as mild to moderate and non-serious, except for one incidence of syncope. This patient had a prior history of first-degree atrioventricular block with syncopal episodes and the adverse event was not considered to be related to treatment. There was one patient in the 3mg/kg cohort who discontinued following moderate erythema of the abdomen. There were four instances of fatigue (13%), four instances of diarrhea (13%) and three instances of contusion (10%) in the 3mg/kg cohort (Figure 14). We note that there have been no signs of fatigue or diarrhea in other clinical trials with therapeutics targeting myostatin, including ACE-031 from Acceleron Therapeutics (XLRN, MO, PT $58) or MYO-029 from Wyeth Pharmaceuticals (now Pfizer, PFE, NC).

FIGURE 14. PINTA-745 (AMG-745) Adverse Events

Incidence of Treatment-Emergent Adverse Events Reported by Three or More Subjects						
	Placebo	AMG 745				
	All Placebo Subjects,	0.3 mg/kg sc 4-Week Dosing,	1.0 mg/kg sc 4-Week Dosing,	3.0 mg/kg sc 4-Week Dosing,	All AMG 745 Subjects,	Total All Subjects,
Adverse event	(n = 23)	n, % (n = 6)	n, % (n = 6)	n, % (n = 19)	n, % (n = 31)	n, % (n = 54)
Fatigue	1 (4)	0 (0)	1 (17)	3 (16)	4 (13)	5 (9)
Injection site bruising	1 (4)	1 (17)	0 (0)	1 (5)	2 (6)	3 (6)
Diarrhea	2 (9)	0 (0)	0 (0)	4 (21)	4 (13)	6 (11)
Contusion	0 (0)	0 (0)	0 (0)	3 (16)	3 (10)	3 (6)

Source: Padhi, JCEM, 2014

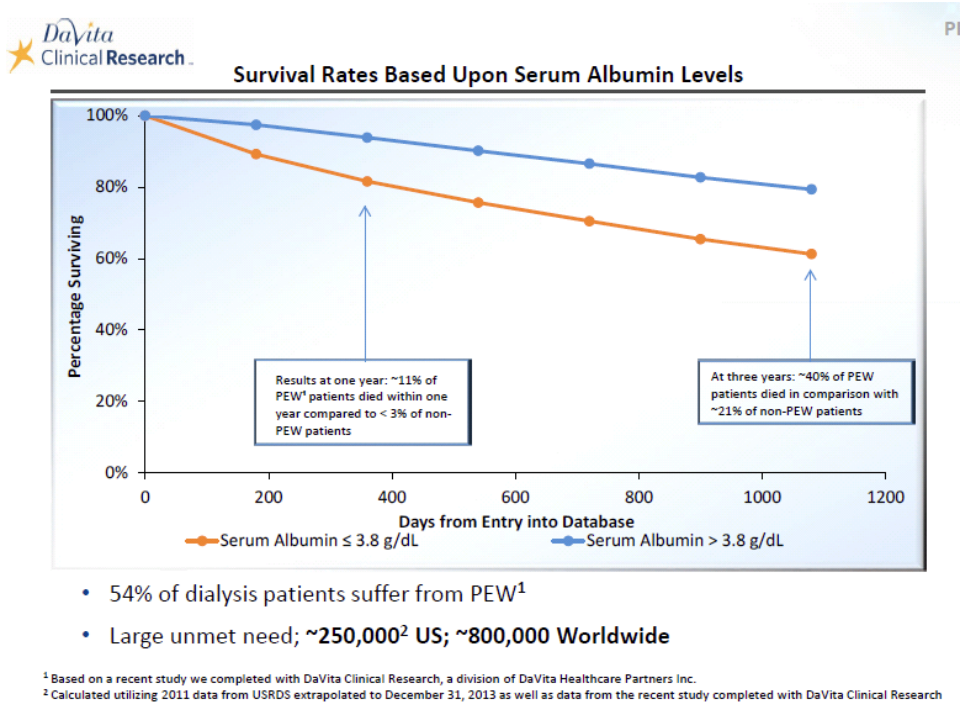
Atara is currently assessing PINTA-745 in a Phase I/II trial in ESRD patients on dialysis with top-line results anticipated by 2015 EOY and full 20-week durability results available in early 2016. The trial is enrolling 48 patients, with an eight-patient safety component and the other 40 will be randomized three to one to receive weekly PINTA-745 or placebo. The trial will determine the maximum-tolerated dose and will evaluate lean body mass (muscle) at 12 weeks as a primary endpoint with change in physical function as a secondary endpoint. The trial is a double-blind, placebo-controlled trial, but we note that it has successfully passed two pre-specified safety assessments.

Regulatory Strategy

Assuming data from the Phase II trial are positive, we anticipate that Atara will move directly to Phase III trials. Increasing skeletal muscle mass will certainly be a primary endpoint, with secondary endpoints including strength tests, such as a six-minute walk or hand grip strength. Our KOL checks indicate that clinically meaningful improvements in physical tests, such as a six-minute walk test, sit to stand, and stair climb would be approvable endpoints, particularly in conjunction with improvements in laboratory values, such as creatinine levels. We believe this represents the fastest route to approval, and model a potential approval by the end of 2018 or beginning of 2019. However, we believe the case for PINTA-745 will be particularly compelling if it is able to decrease PEW associated morbidity or mortality.

Therefore, we model that Atara will complete a large-scale trial with several thousand patients to expand the label. The difference in survival between PEW patients (defined as those with BSA levels below 3.8g/dL) and non-PEW patients is quite striking, with 11% mortality vs. 3% mortality at one year (Figure 15), according to a study conducted by Atara in collaboration with DaVita Clinical Research. We, therefore, model the PINTA-745 program receiving expanded regulatory approval by 2021.

FIGURE 15. Increased Mortality in Patients with PEW



Source: Company Reports

Commercial opportunity

We estimate that there are over 400,000 dialysis patients in the U.S. as of 2015. We arrive at this number based on 2011 prevalence, as reported by the United States Renal Data System (USRDS) 2013 Atlas of CKD and ESRD, grossed up by a 1.5% annual rate of increase. While ESRD rates grew in the 6% range for much of the last two decades, current estimates suggest that the rate of increase is leveling off (Hoerger, AJKD, 2015). Fully 55% of patients on dialysis have serum albumin levels of ≤3.8g/dL, which is a generally accepted cut-off used for diagnosing patients with PEW. By our estimates, in 2019, the first full year of marketing for PINTA-745, the addressable population will be ~267,000 patients in the U.S.

While PINTA-745 would not necessarily fall within the dialysis bundle, we have modeled the price conservatively at ~$215 per week, amounting to ~20% premium to Epogen, which is used for treating anemia in dialysis patients. With an approval based on improved physical condition, we model that patients would receive ~26 doses per year and that Atara could achieve a penetration rate of ~16% of the U.S. market. With an expanded label, based on a positive outcomes trial, we think there would be increased incentive for the major dialysis centers to prescribe PINTA-745, allowing Atara to capture an additional 26% of the market. We model peak sales of ~$1.0B in the U.S. by the time the patent expires in 2033.

FIGURE 16. PINTA-745 for Improving Muscle Mass and Physical Function in PEW Market Model

PINTA-745 for physical function in PEW ($MM)	2015E	2016E	2017E	2018E	2019E	2020E	2021E	2022E	2023E	2024E	2025E	2032E	2033E
Epidemiology													
prevalence of ESRD patients on dialysis	456,676	463,526	470,479	477,536	484,699	491,970	499,349	506,840	514,442	522,159	529,991	588,208	597,031
% growth	*1.5%*	*1.5%*	*1.5%*	*1.5%*	*1.5%*	*1.5%*	*1.5%*	*1.5%*	*1.5%*	*1.5%*	*1.5%*	*1.5%*	*1.5%*
% of dialysis pts with albumin <= 3.8g/dL	*55%*	*55%*	*55%*	*55%*	*55%*	*55%*	*55%*	*55%*	*55%*	*55%*	*55%*	*55%*	*55%*
# of ESRD Patients on dialysis with PEW	251,172	254,939	258,764	262,645	266,585	270,583	274,642	278,762	282,943	287,187	291,495	323,514	328,367
PINTA- 745 Penetration				***4.0%***	***8.0%***	***12.0%***	***16.0%***	***16.0%***	***16.0%***	***16.0%***	***16.0%***	***16.0%***	***16.0%***
# of Patients on PINTA-745				10,506	21,327	32,470	43,943	44,602	45,271	45,950	46,639	51,762	52,539
# of cycles per year (weekly cycle)				5.0	20.0	26.0	26.0	26.0	26.0	26.0	26.0	26.0	26.0
Total Patient Cycles on Therapy (Thousands)				**53**	**427**	**844**	**1,143**	**1,160**	**1,177**	**1,195**	**1,213**	**1,346**	**1,366**
Cost per week		$ 215	$ 221	$ 228	$ 235	$ 242	$ 249	$ 257	$ 264	$ 272	$ 281	$ 345	$ 355
% price increase			*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*
US Sales of PINTA-745 ($MM)				**$12.0**	**$100.2**	**$204.3**	**$284.8**	**$297.7**	**$311.2**	**$325.4**	**$340.2**	**$464.3**	**$485.4**
Royaly rate						4%	4%	6%	6%	6%	6%	6%	6%
US Royalties to AMGN ($MM)				$ -	$ 4.0	$ 8.2	$ 17.1	$ 17.9	$ 18.7	$ 19.5	$ 20.4	$ 27.9	$ 29.1

Source: JMP Securities LLC and Company Reports

FIGURE 17. PINTA-745 Improved Morbidity and Mortality in PEW Market Model

PINTA-745 for PEW Morbidity and Mortality ($MM)	2015E	2016E	2017E	2018E	2019E	2020E	2021E	2022E	2023E	2024E	2025E	2032E	2033E
Epidemiology													
prevalence of ESRD patients on dialysis	456,676	463,526	470,479	477,536	484,699	491,970	499,349	506,840	514,442	522,159	529,991	588,208	597,031
% growth	*1.5%*	*1.5%*	*1.5%*	*1.5%*	*1.5%*	*1.5%*	*1.5%*	*1.5%*	*1.5%*	*1.5%*	*1.5%*	*1.5%*	*1.5%*
% of dialysis pts with albumin <= 3.8g/dL	*55%*	*55%*	*55%*	*55%*	*55%*	*55%*	*55%*	*55%*	*55%*	*55%*	*55%*	*55%*	*55%*
# of ESRD Patients on dialysis with PEW	251,172	254,939	258,764	262,645	266,585	270,583	274,642	278,762	282,943	287,187	291,495	323,514	328,367
PINTA- 745 Penetration							***6.0%***	***12.0%***	***18.0%***	***24.0%***	***25.0%***	***25.0%***	***25.0%***
# of Patients on PINTA-745						0	16,479	33,451	50,930	68,925	72,874	80,879	82,092
# of cycles per year (weekly cycle)							20.0	25.0	30.0	30.0	30.0	30.0	30.0
Total Patient Cycles on Therapy (Thousands)						**0**	**330**	**836**	**1,528**	**2,068**	**2,186**	**2,426**	**2,463**
Cost per week		$ 215	$ 221	$ 228	$ 235	$ 242	$ 249	$ 257	$ 264	$ 272	$ 281	$ 345	$ 355
% price increase			*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*
US Sales of PINTA-745 ($MM)					**$0.0**	**$0.0**	**$82.1**	**$214.7**	**$404.0**	**$563.2**	**$613.3**	**$837.1**	**$875.2**
Royaly rate					4%	4%	6%	6%	6%	6%	6%	6%	6%
US Royalties to AMGN ($MM)						$ -	$ 4.9	$ 12.9	$ 24.2	$ 33.8	$ 36.8	$ 50.2	$ 52.5

Source: JMP Securities LLC and Company Reports

Based on our research, we estimate there are ~350,000 dialysis patient in the EU as of 2015. For Europe, where the treatment of dialysis patients is less aggressive, we model total market penetration of 30%, resulting in peak sales for PINTA-745 of ~$390M by 2033. In Japan, Amgen had already licensed PINTA-745 to Takeda Pharmaceuticals. Therefore, we calculated the market size for the rest of the world (ROW), excluding U.S., Europe, and Japan. Based on data from the WHO, we estimate there are ~800,000 ROW dialysis patients in 2015. For ROW, we only assume a peak market penetration rate of 26% resulting in peak 2033 sales of ~$1.0 B.

ATA-842 for Cancer Cachexia.

ATA-842 is an antibody that specifically binds myostatin and prevents it from activating the ActRIIB or ActRIIA receptors. The background biology and mechanism of action are essentially the same as for PINTA-745, with two notable distinctions that differentiate the two products. First, as an antibody, ATA-842 is highly specific and binds exclusively to myostatin. Atara has observed that it does not seem to have the same anti-inflammatory effects that PINTA-745 has, presumably because it does not bind to other TGF-β family members. In cancer cachexia, myostatin can be produced by the tumor cell itself (Lokireddy, Biochem J, 2012), rather than being induced by the inflammatory environment as in PEW. The more limited mechanism of action of ATA-842 may thus prove more appropriate for treating cancer

patients. Second, ATA-842 has a longer half-life than PINTA-745. Dialysis patients already have to visit a clinic three times weekly for intravenous-based therapy, and nephrologists have expressed a preference for tighter control and regulation of dosing in dialysis patients. However, monthly dosing would be preferable for cancer patients, who are not necessarily receiving IV treatments. ATA-842 is in IND-enabling studies and we do not ascribe any value to the program at this time.

COMPETITIVE LANDSCAPE

There are a number of companies developing anti-myostatin therapeutics. However, at this time, Atara is the only company pursuing PEW as an indication, putting it several years ahead of any potential competitor. Atara has also been collaborating with DaVita Healthcare partners (DVA, NC) which is one of the two largest dialysis and kidney care companies in the United States. While the two companies do not have a specific development or marketing agreement, we view Atara's access to DaVita's expertise and prodigious patient database as providing the company with an enviable competitive edge.

Early entrants in the anti-myostatin space included Wyeth's MYO-029 anti-myostatin antibody and Acceleron's ACE-031 ActRIIb receptor fusion. MYO-029 was terminated due to limited signs of efficacy and rashes at higher doses in a Phase I/II trial in muscular dystrophy. ACE-031 demonstrated significant increases in lean mass, but was discontinued after healthy patients and boys with Duchenne Muscular Dystrophy treated with multiple high doses developed nosebleeds and telangiectasia. ACE-031 was a soluble ActRIIb-Fc fusion receptor that bound a broad array of TGF-β family members including activins, bone morphogenetic proteins and other growth and differentiation factor members.

Currently active programs include PF-06252616 from Pfizer (PFE, NC), BMS-986089 from Bristol-Myers Squibb (BMS, NC) and LY249565 from Eli Lilly & Co. (LLY, NC) (Figure 18). PF-06252616 is an antibody that recognizes myostatin complexed with its propeptide. This program is enrolling patients in a Phase II trial for Duchenne muscular dystrophy after demonstrating signs of efficacy and no notable adverse events in a Phase I trial. BMS is also pursuing muscular dystrophy, with a Phase I trial in healthy volunteers using its Adnectin based therapeutic to bind and sequester myostatin. Eli Lilly is pursuing both cancer cachexia and sarcopenia, which is muscle wasting in elderly patients, with an anti-myostatin antibody.

FIGURE 18. Competitive Landscape

Investigational Therapeutic	Company	Mode of action	Indication	Status
MYO-029	Wyeth	anti-myostatin antibody	muscular dystrophy	program terminated: Phase I/II limited signs of efficacy, rashes at higher doses
ACE-031	Acceleron/Shire	ActRIIb-receptor fusion	muscular dystrophy	program terminated: Phase I/II good signs of efficacy; but poor AE profile: nose bleeds, bleeding gums, skin vascular rash
ACE-083	Acceleron	cysteine knot ligand trap of TGF-β family members	not specified	Currently enrolling Phase I in healthy volunteers
PF-06252616	Pfizer	anti-myostatin antibody	Duchennes muscular dystrophy	Phase I: signs of efficacy; no notable AE; Phase II: PCD 2017
BMS-986089	BMS	anti-myostatin adnectin	Duchennes muscular dystrophy	Ongoing: Phase I radomized dose escalation in healthy adults
LY249565	Eli Lilly	anti-myostatin antibody	muscle loss and weakness in advanced cancer	Ongoing: Phase I dose escalation
			sarcopenia: elderly (≥75 years) with low muscle strength and ≥ 1 fall per year	Phase II completed: lean body mass significantly increased; good AE profile primarily injection site reactions
SAR391786	Regeneron	anti-myostatin antibody	sarcopenia	Phase I and Phase II completed, Phase II results by EOY 2015
AAV1-FS344	Milo Biotechnology	Adenovirus mediated gene therapy delivery of follistatin (myostatin binding)	muscular dystrophy	Phase I/II demonstrated signs of activity in three of six Becker muscular dystrophy patients. Phase II ongoing
			Sporadic inclusion body myositis	Phase I/II ongoing

Source: JMP Securities LLC and Company Reports

STM-434 FOR THE TREATMENT OF SOLID TUMORS

Background

STM-434 is a soluble activin receptor IIB (ActRIIB)-Fc fusion protein. It binds to a number of TGF-β family members including activin A, activin B, bone morphogenetic protein 11 (BMP-11), and myostatin. Activin A has been extensively characterized for its role during embryogenesis where it is a potent morphogen that directs tissue development by modulating mesodermal cell fate, differentiation and proliferation. Post embryonically, the expression of activin A is tightly controlled, but it plays a role in a number of processes and organs. It regulates follicle stimulating hormone (FSH) production in the ovaries, spermatogenesis in testes, promotes wound healing by stimulating keratinocytes and stromal cells and plays a role in the migration and invasive properties of immune cells and endometrial cells. Given its effect on fundamental processes such as cell differentiation, proliferation and migration, it is perhaps not surprising that activin A has been found to play a role in the biology of cancer cells and metastases.

Activin A binds to the same receptor as myostatin and triggers the same downstream signaling events. However, as for all TGF-β family members, the effect of activin A signaling is very context specific. Different cell types contain various repertoires of co-receptors, transcription factors and epigenetic modifications. Thus, activin A may induce proliferation in one cell type while suppressing proliferation in another cell type. This has been found to be the case in various tumor types. Activin A demonstrates tumor suppressive effects in prostate and breast cancer, while in lung, renal cell carcinoma and head and neck squamous cell carcinoma, its expression consistently correlates with increased proliferation, invasion and poor patient prognosis.

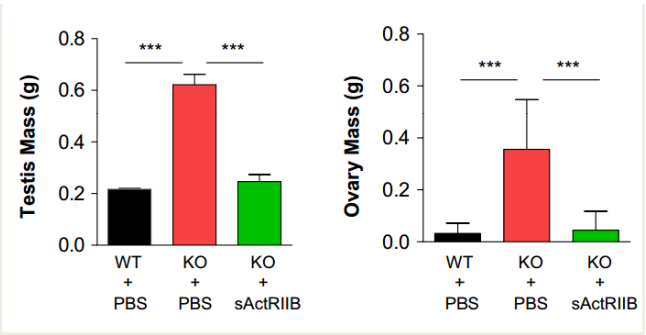
Atara is particularly interested in using STM-434 to treat two types of ovarian cancer, granulosa and clear cell, as they provide a potential pathway to accelerated approval. Granulosa cancers are relatively rare, accounting for ~2-3% of ovarian cancers while the prevalence of clear cell tumors is higher, at about ~5-10% of ovarian cancers in North America and Europe, and around 15-25% in Japan. Neither responds well to platinum based chemotherapy. As a result, alternative therapies are needed (Anglesio, GO, 2011; Cheng, BBRC, 2014). Tumor growth for both cell types is driven by activin A, and STM-434 suppressed tumor growth in preclinical models of both granulosa and clear cell cancers (Figure 19).

Mechanism of action

Nearly all granulosa tumors (97%) have a mutation in FOXL2, a member of the forkhead/winged-helix family of transcription factors. Activin signaling activates FOXL2, which triggers a negative feedback loop that keeps activin signaling in check. FOXL2 drives the transcription of follistatin, a broadly expressed glycoprotein whose main function is to bind and neutralize members of the TGF-β superfamily. Granulosa cells normally express high levels of follistatin, but in the absence of FOXL2 they are exposed to a constant proliferative stimulus from activin A. By binding and sequestering activin A, STM-434 would remove the proliferative signal that drives the growth of granulosa cells.

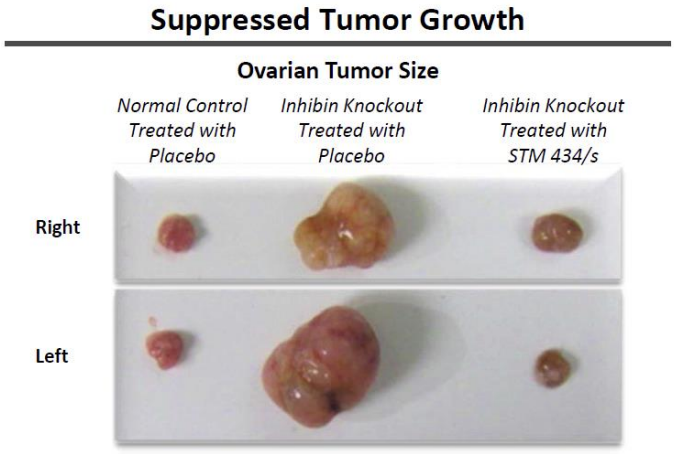
There is preclinical evidence demonstrating that a mouse homolog of STM-434 can inhibit tumor growth in another model of dysregulated activin signaling. Inhibin is another TGF-β family member that binds to the ActRIIb receptor. In most biological systems, inhibin counters the biological activity of activin. Mice with a targeted deletion of inhibin develop spontaneous gonadal tumors. Treatment of these mice with STM-434 inhibits tumor growth, as demonstrated in Figures 19-20.

FIGURE 19. A Mouse Form of STM-434 (sActRIIB) Inhibits Testis and Ovary Tumor Growth in Inhibin Knockout (KO) Mice



Source: JMP Securities LLC and Company Reports

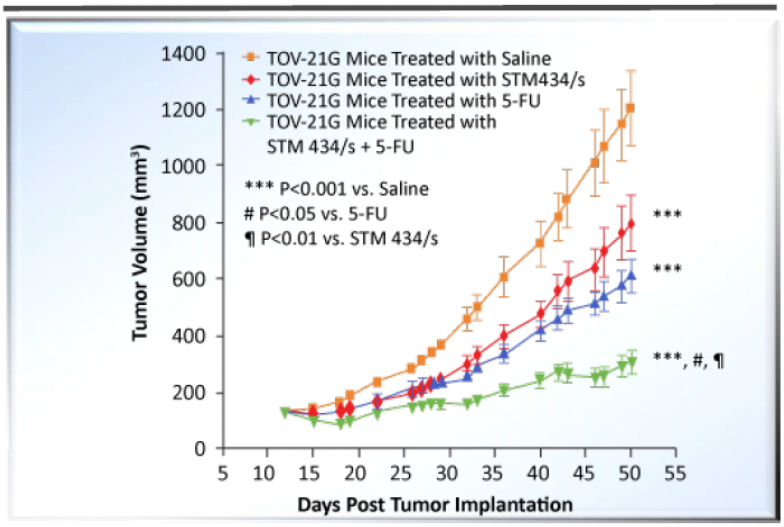
FIGURE 20. A Mouse Form of STM-434 (sActRIIB) Inhibits Ovary Tumor Growth in Inhibin Knockout Mice



Source: Company Reports

An alternate version of STM-434 (STM-217) has also demonstrated efficacy in a mouse model of clear cell carcinoma. Researchers implanted human TOV-21g (clear cell ovarian cancer cell line) xenografts in the abdominal flank region of athymic nude mice. After fourteen days, weekly subcutaneous injections of STM-217 were administered either alone or in combination with 5-fluorouracil (5-FU). STM-217 treatment statistically significantly reduced tumor growth by 43% (p<0.0001) when compared to the vehicle-treated tumor group. In the combination efficacy experiment, 5-FU monotherapy reduced tumor growth by 47% (p<0.0001), and the combination of STM-217 and 5-FU together inhibited growth by 73% (p<0.0001; Figure 21).

FIGURE 21. Significant Reductions in Clear Cell Carcinoma Tumor Size in Mice Treated with STM-434 (Variant STM-217)



Source: Lu & Haqq, ASCO 2013

Regulatory Strategy

Atara is currently enrolling patients in an open-label, all-comers Phase I trial in combination with liposomal doxorubicin to establish a maximum tolerated dose for advancing to Phase II. Atara will also collect radiographic response rates and preliminary anti-cachexia data. Atara has requested that participating physicians attempt to recruit ~30% ovarian cancer patients. If there are signs of efficacy in the Phase I and Phase II trials, we anticipate Atara will seek accelerated approval for treating granulosa and clear cell ovarian tumors, both of which are poorly controlled by first-line chemotherapy. We expect this initial path to approval will be followed by entry in the relapsed and refractory setting in other tumor types that are driven by activin A signaling, including lung, renal cell carcinoma and head and neck squamous cell carcinoma.

Commercial Opportunity

At this time, we only model the granulosa and clear cell ovarian tumor markets, for which there are pre-clinical evidence. We estimate that there are ~22,000 ovarian cancer patients in the U.S. as of 2015 and that this population will grow at a rate of ~1.5% per year (extrapolated from GLOBOCAN estimates). We estimate that the combined granulosa and clear cell populations account for ~10% of all ovarian cancer cases. Further, we assume that 50% of patients will be diagnosed at Stage II or later and that 80% of these patients will fail first-line chemotherapy. (Stage I patients are treated by surgical resection). Should STM-434 receive accelerated approval by 2019, the addressable population will be just under 1,000 patients per year. We model STM-434 achieving 40% market penetration. The estimated overall survival for women with paclitaxel-resistant ovarian cancer receiving a second-line chemotherapy is in the seven-month range (Anglesio, GynOnc, 2011). We assume Atara will have to demonstrate at least a three-month improvement in OS for final approval; therefore, we model patients receiving an average of 11 cycles per year. We model a price of ~$12,000 per month in 2019, extrapolating from current day prices for other anti-cancer biologicals, such as ramucirumab (Cyramza, Eli Lilly, NC). For these indications, we project peak sales of approximately $180MM combined in the U.S., Europe and Japan, While this represents the fastest path to market, other activin-driven tumor types, such as breast and prostate, represent larger markets.

We model STM-434 reaching the market for third-line metastatic breast cancer in 2022. According to SEER, the prevalent breast cancer population in the U.S. is ~3,000,000, of which ~5% have metastatic disease. We model 65% of patients receiving first-line therapy, of which 40% progress. Approximately 70% of the progressors will receive second-line therapy and 70% of those will progress (Anglesio, Gynecologic Onc, 2011). We, therefore, model an addressable population of ~19,000, which we anticipate will receive an average of six monthly doses, (Figure 23). We project peak U.S. sales of ~700MM by 2033.

FIGURE 22. STM-434 in Ovarian Cancer Market Model

US													
STM-434 in ovarian cancer ($MM)	**2015E**	**2016E**	**2017E**	**2018E**	**2019E**	**2020E**	**2021E**	**2022E**	**2023E**	**2024E**	**2025E**	**2032E**	**2033E**
Epidemiology													
# of ovarian cancer patients	22,148	22,458	22,772	23,091	23,415	23,742	24,075	24,412	24,754	25,100	25,452	28,053	28,446
% growth	*1.4%*	*1.4%*	*1.4%*	*1.4%*	*1.4%*	*1.4%*	*1.4%*	*1.4%*	*1.4%*	*1.4%*	*1.4%*	*1.4%*	*1.4%*
%with clear cell or granulosa tumors	*10.0%*	*10.0%*	*10.0%*	*10.0%*	*10.0%*	*10.0%*	*10.0%*	*10.0%*	*10.0%*	*10.0%*	*10.0%*	*10.0%*	*10.0%*
% stage II-IV	*50%*	*50.0%*	*50.0%*	*50.0%*	*50.0%*	*50.0%*	*50.0%*	*50.0%*	*50.0%*	*50.0%*	*50.0%*	*50.0%*	*50.0%*
# of Patients with clear cell or granulosa tumors	1,107	1,123	1,139	1,155	1,171	1,187	1,204	1,221	1,238	1,255	1,273	1,403	1,422
% relapsed/refractory to platinum	*80.0%*	*80.0%*	*80.0%*	*80.0%*	*80.0%*	*80.0%*	*80.0%*	*80.0%*	*80.0%*	*80.0%*	*80.0%*	*80.0%*	*80.0%*
Addresssable CC and granulosa population	886	898	911	924	937	950	963	976	990	1,004	1,018	1,122	1,138
STM-434 Penetration					***6.0%***	***12.0%***	***20.0%***	***27.0%***	***35.0%***	***40.0%***	***40.0%***	***40.0%***	***40.0%***
# of Patients on STM-434					56	114	193	264	347	402	407	449	455
# of cycles of treatment per year (monthly dosing)					3.0	6.0	8.0	10.0	11.0	11.0	11.0	11.0	11.0
Total Patient Cycles on Therapy				**0**	**169**	**684**	**1,541**	**2,636**	**3,812**	**4,418**	**4,479**	**4,937**	**5,006**
Cost per cycle		$ 11,000	$ 11,330	$ 11,670	$ 12,020	$ 12,381	$ 12,752	$ 13,135	$ 13,529	$ 13,934	$ 14,353	$ 17,652	$ 18,181
% price increase			*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*
US Sales of STM-434 (Millions)				**$0.0**	**$2.0**	**$8.5**	**$19.6**	**$34.6**	**$51.6**	**$61.6**	**$64.3**	**$87.2**	**$91.0**
Royaly rate	4%	4%	4%	4%	4%	4%	4%	4%	6%	6%	6%	6%	6%
US Royalties to AMGN ($MM)						**$ 0**	**$ 1**	**$ 1**	**$ 3**	**$ 4**	**$ 4**	**$ 5**	**$ 5**

Source: JMP Securities LLC and Company Reports

FIGURE 23. STM-434 in Breast Cancer Market Model

US												
STM-434 in breast cancer ($MM)	2015E	2016E	2017E	2018E	2019E	2020E	2021E	2022E	2023E	2024E	2025E	2033E
Epidemiology												
# of breast cancer patients	2,975,314	3,016,968	3,059,206	3,102,035	3,145,463	3,189,500	3,234,153	3,279,431	3,325,343	3,371,898	3,419,104	3,821,343
% growth	1.4%	1.4%	1.4%	1.4%	1.4%	1.4%	1.4%	1.4%	1.4%	1.4%	1.4%	1.4%
% with metastatic disease	5.0%	5.0%	5.0%	5.0%	5.0%	5.0%	5.0%	5.0%	5.0%	5.0%	5.0%	5.0%
1st line metastatic disease population	148766	148766	148766	148766	148766	148766	148766	148766	148766	148766	148766	148766
% patients treated with1st line	65%	65%	65%	65%	65%	65%	65%	65%	65%	65%	65%	65%
% progression in 1L metastatic breast cancer	40%	40%	40%	40%	40%	40%	40%	40%	40%	40%	40%	40%
% patients treated with 2nd line	70%	70%	70%	70%	70%	70%	70%	70%	70%	70%	70%	70%
% progression on 2L metastatic breast cancer	70%	70%	70%	70%	70%	70%	70%	70%	70%	70%	70%	70%
Addresssable 3rd line metastatic breast population	18,953	18,953	18,953	18,953	18,953	18,953	18,953	18,953	18,953	18,953	18,953	18,953
STM-434 Penetration								4.0%	12.0%	24.0%	36.0%	40.0%
# of Patients on STM-434								758	2,274	4,549	6,823	7,581
# of cycles of treatment per year (monthly dosing)								3.0	6.0	6.0	6.0	6.0
Total Patient Cycles on Therapy (Thousands)				0	0	0	0	2	14	27	41	45
Cost per cycle		$ 11,000	$ 11,330	$ 11,670	$ 12,020	$ 12,381	$ 12,752	$ 13,135	$ 13,529	$ 13,934	$ 14,353	$ 18,181
% price increase			3%	3%	3%	3%	3%	3%	3%	3%	3%	3%
US Sales of STM-434 (Millions)				$0.0	$0.0	$0.0	$0.0	$29.9	$184.6	$380.3	$587.6	$827.0
Royaly rate	4%	4%	4%	4%	4%	4%	4%	4%	6%	6%	6%	6%
US Royalties to AMGN ($MM)						$ -	$ -	$ 1.2	$ 11.1	$ 22.8	$ 35.3	$ 49.6

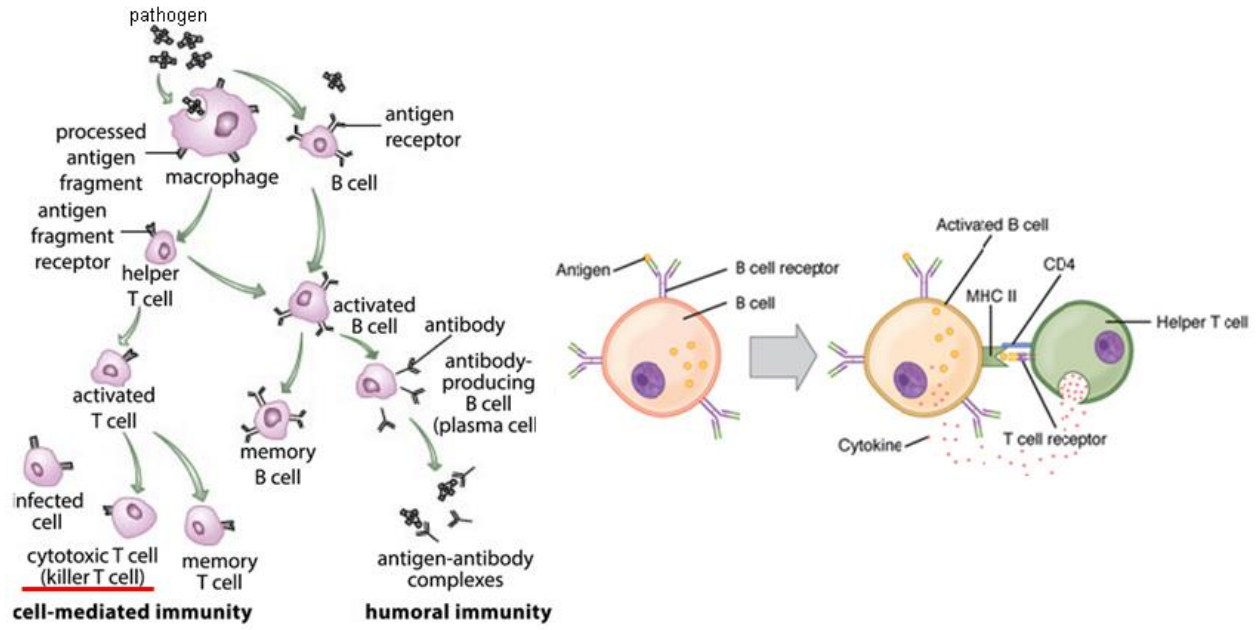
Source: JMP Securities LLC and Company Reports

ADOPTIVE CELL THERAPY: PERSONALIZED CANCER IMMUNOTHERAPY

The immune system plays a critical role in controlling tumor growth. Although it is an old concept, the idea of harnessing the power of the immune system to marshal an attack against cancer has only grown in popularity in recent times, especially with the successes of checkpoint inhibitors and cell-based therapies, such as CAR T and TCR. Even so, personalized immunotherapy has only begun to gain traction and some momentum in the clinic recently. As some experts in the field have stated, adoptive cell therapy (ACT) is “giving patients a living drug.” It involves the transfusion of T lymphocytes - immune cells in the blood that recognize and mount an attack against foreign invaders and diseased cells - to induce regression of cancer.

T cells have the ability to eliminate diseased or infected cells. They do so by coursing through the body, using their T-cell receptors (TCRs) to scan for antigens (short sequences of protein), presented by major histocompatibility complex (MHC) molecules on the surface of infected cells or distressed cells. When an antigen matches a receptor, the T cell activates and launches an attack. T cells are believed to play a central role in eliminating cells that accumulate potentially carcinogenic mutations, but tumor cells evolve to evade an immune attack by a variety of techniques, such as downregulating antigen presentation or expressing molecules that suppress immune activation. Adoptive cell transfer involves the identification, enrichment and ex vivo expansion of tumor-specific T cells (T cells with antitumor activity), which are then re-infused back into the cancer patient. The aim is to give the patient’s immune system the ability to overpower the tumor using these ‘trained’ T cells to initiate the response.

FIGURE 24. T Cells and B Cells

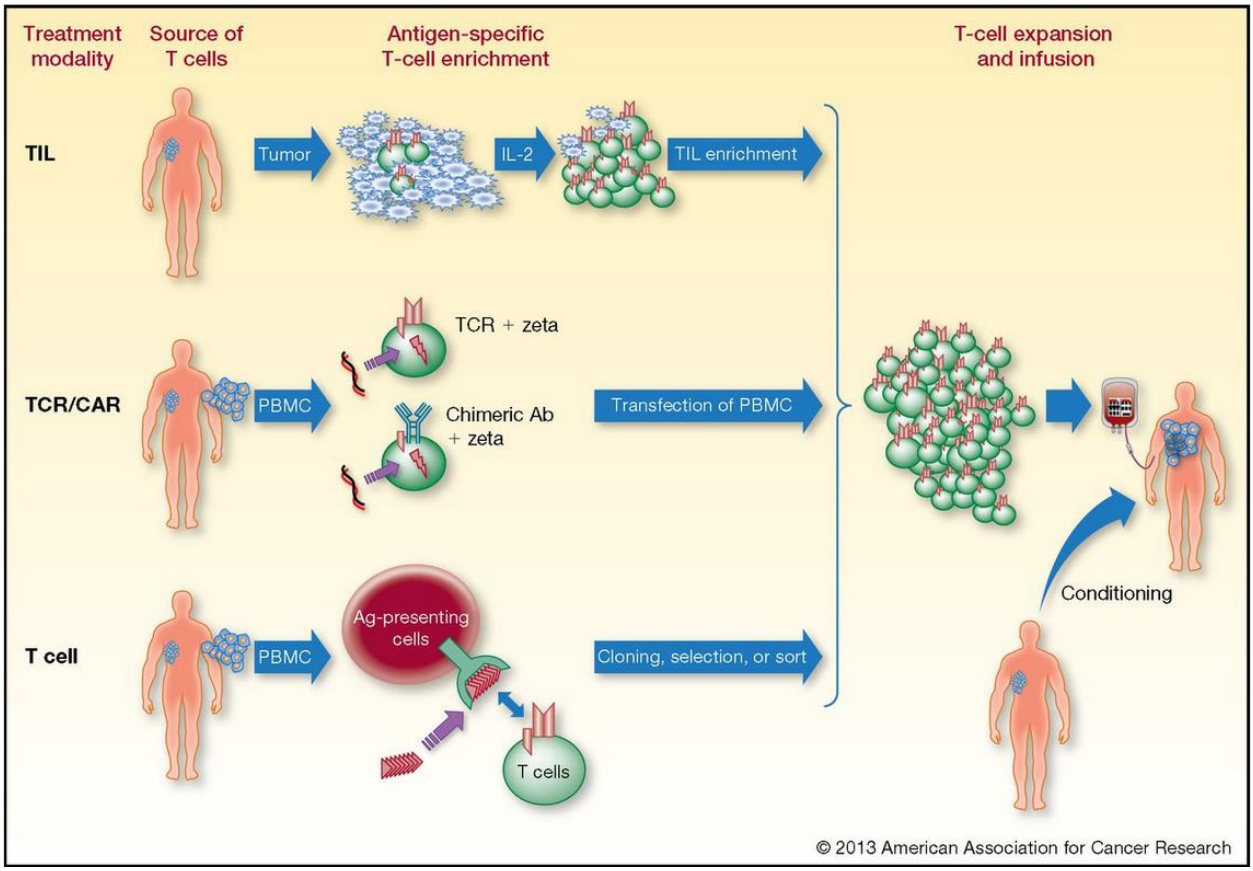


Source: www.courses.candelalearning.com/; www.esciencecentral.org

There are several forms of adoptive T cell therapies (Figure 25):

- Culturing tumor infiltrating-lymphocytes (TILs) from primary or secondary tumors, or the T cells that have migrated to the tumor microenvironment (such as being developed by Lion Biotechnologies (LBIO, NC)), followed by infusion of the expanded cells into the patient, together with Interleukin-2 (IL-2).
- Using genetically modified T cells for expression of a chimeric T cell receptor: modifying the T cells by encoding artificial, antibody-like proteins that bind the antigens studding the tumor cell's surface (CARs), such as those being developed by JUNO (NC), Novartis (NVS, NC), KITE (NC) and Cellectis (NC).
- Modifying T cells to alter the specificity of the T cell receptor (TCR), where the antibodies (as used in CARs) are excluded and therefore require presenting molecules such as HLA (internal antigens), such as those being developed by Adaptimmune (ADAP, NC); or modifying the T cells so that they are also enriched for a specific virus to treat virus-induced diseases (CTLs), which also require presenting molecules (HLA matching), such as treatments from ATRA and Cell Medica (private).

FIGURE 25. Adoptive T-cell Therapy



Source: Yee C, Clin Cancer Res, 2013

TARGETING VIRAL-SPECIFIC TUMORS WITH VIRAL-SPECIFIC T CELLS

Summary

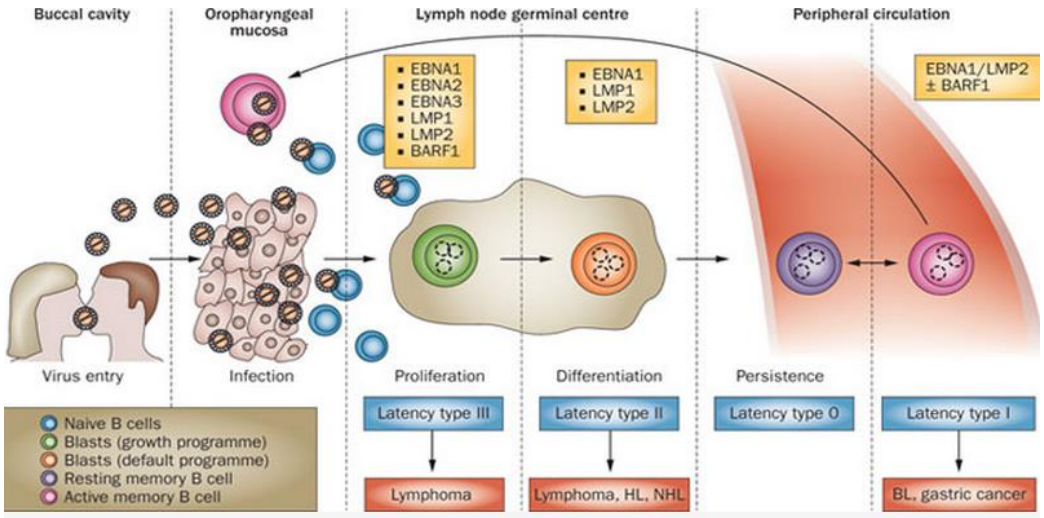
T cells represent a critical component of the body's immune and defense system. As mentioned, T cells may be harnessed to mount a response against viral infections and certain cancers by directing T cells to specific proteins that have been implicated in cancers or infections. Importantly, by retargeting primary human T cells, as a therapeutic tool to treat cancers that are otherwise greatly unmet or refractory to current therapies, adoptive therapies have the potential to expand an oncologist's armamentarium for the treatment of such cancers.

In June 2015, Atara exclusively licensed the worldwide rights to three clinical T-cell product candidates from MSKCC (EBV-CTL, CMV-CTL and WT1-CTL) – viral-specific T-cells for targeting viral-specific tumors. The company also has the option to exclusively license the worldwide rights to other undisclosed T-cell programs, either discovered or developed by MSK coming out of research funded by Atara.

Epstein-Barr Virus

A little over 50 years ago, Sir Anthony Epstein and his research assistant, Dr. Yvonne Barr, discovered the Epstein-Barr virus (EBV), which was subsequently revealed to be the first known human tumor virus. EBV is a common pathogen that is found widespread in humans, persisting as a life-long and largely asymptomatic infection. EBV belongs to the herpes virus family and is specifically categorized as human herpesvirus 4 (HHV-4). EBV is commonly spread by contact through bodily fluids and most often transmitted through saliva (Figure 26; EBV Life Cycle). Upon entering the host, the virus replicates in the oropharyangeal epithelial cells, and then preferentially infects B lymphocytes or cells. The virus then persists for the life of the host as a latent infection in a transcriptionally quiescent state within the B cell pool, as a harmless passenger circulating in the peripheral blood. The virus can alternate between two states: as active or latent (inactive) states, although the virus can only spread during the active state to cause infection. The infected B cells are phenotypically indistinguishable from normal memory B cells and are therefore undetectable, and do not generally cause an immune response.

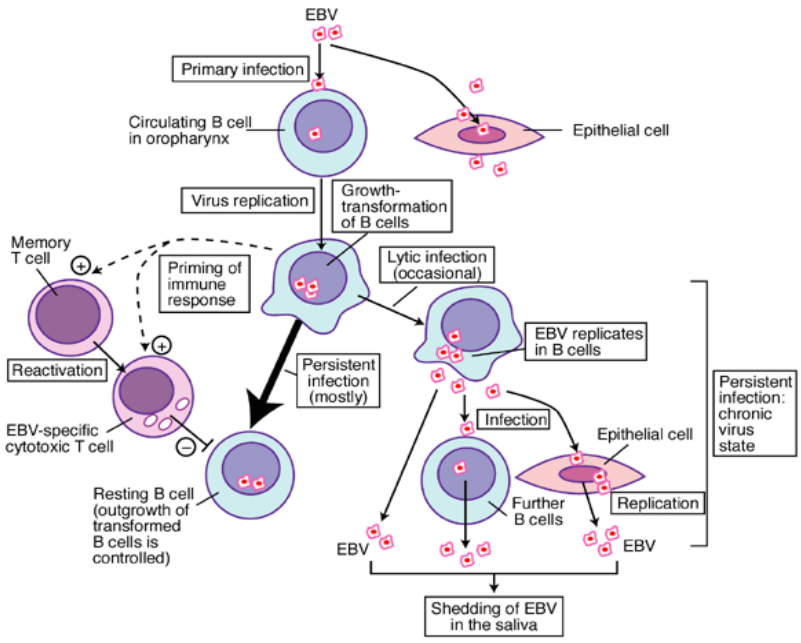
FIGURE 26. EBV Life Cycle



Source: Bollard et al, Nat Rev Clin Oncol 2012

Approximately 90-95% of adults are benignly affected by EBV and have evidence of EBV infection; they have antibodies present in their blood to indicate the presence of an active or latent EBV infection. Typically, when children are infected with EBV, they often display no symptoms, and then go on to carry the virus to adulthood. However, teenagers or young adults who acquire the virus may develop glandular fever (infectious mononucleosis). Nevertheless, most recover without incurring long-term effects.

FIGURE 27. EBV Infection in Normal Healthy Virus Carriers



Source: Expert Reviews in Molecular Medicine 2001

Even though nearly every adult is a carrier for EBV, it can present as a major health issue in individuals with compromised immune systems. In those specific types of individuals, this seemingly innocuous virus has the potential to cause cancer. EBV has a growth-transforming ability in these individuals and has been linked to the development of several forms of cancer, in particular three types of B cell malignancies: Burkitt lymphoma (BL), Hodgkin's lymphoma (HL), and post-transplant lymphoproliferative disease (PTLD). EBV is also known to cause other illnesses including gastric cancer, nasopharyngeal carcinoma, and numerous other types of cancers. Additionally, EBV has also been associated with progression of certain neurologic conditions, such as multiple sclerosis (MS).

EBV Lymphoma: EBV-associated PTLD after HSCT

Post-transplant lymphoproliferative disorder (PTLD) can occur as a serious complication resulting after hematopoietic stem cell transplantation (HSCT; bone marrow transplant) or solid organ transplantation (SOT), in up to 10% of adult patients. A majority of PTLD cases are EBV-driven. Thus, when there is an overgrowth of EBV-infected B cells, in the setting of chronic immunosuppression and decreased EBV-specific cytotoxic T cell immune surveillance (i.e., immune deficiency), lymphoproliferative (LPD) disease (or specifically, EBV lymphoma) results. The EBV-infected B cells that cause PTLD can originate from either the recipient that is the host or the donor. In the HSCT setting, PTLD usually results from donor B-cells, within 6-12 months of the post-transplant period.

Typically, EBV lymphoma following HSCT is aggressive and characterized by DLBCL (diffuse large B-cell lymphoma), wherein average survival without therapy is a mere 31 days (Figure 28). Rituxan (rituximab), a chimeric murine/human monoclonal antibody against CD20, an antigen expressed on the surface of B-cells, is generally given to such patients as "off-label" as a first line of therapy. However, rituximab treatment is associated with about a 50% disease response rate. Other patients with more extensive or rapidly progressive disease may also receive rituximab in combination with chemotherapy such as CHOP (cyclophosphamide, doxorubicin, vincristine, and prednisone). Given the toxicity associated with chemotherapy, especially in patients with co-morbidities, less-intensive regimens are sought. Of significance, patients who are refractory to rituximab treatment have a median survival of only 16-56 days.

FIGURE 28. Patient Characteristics

Treatment Setting	HCT	SOT
Incidence	Unmodified (1-3%), T cell depleted (5-7%), CBT (3-5%)	Heart-Lung (8-10%), Liver (6%)
Tumor Character	Aggressive-usually clonal DLBCL , survival w/0 therapy 31d	Indolent to Aggressive, polyclonal or clonal
High Risk Features	Age, GvHD, Extra-nodal disease, ≥3 sites, CNS, High risk patients 72% mortality (Styczynki 2013)	Age, KPS, LDH, CNS, time from transplant, 2 yr risk based OS 88%, 50%, 0% (Choquet 2007)
Rituxumab Responses	Viremia -80%; Disease - 50%-25% durable	Early lesions - 76%; High Grade lesions -47%
Rituxumab Refractory	16-56 day median OS (Fox 2014, Ocheni 2008, Uhlin 2014)	33% 2 yr OS (Choquet 2007)

Source: ASCO 2015, Company Reports

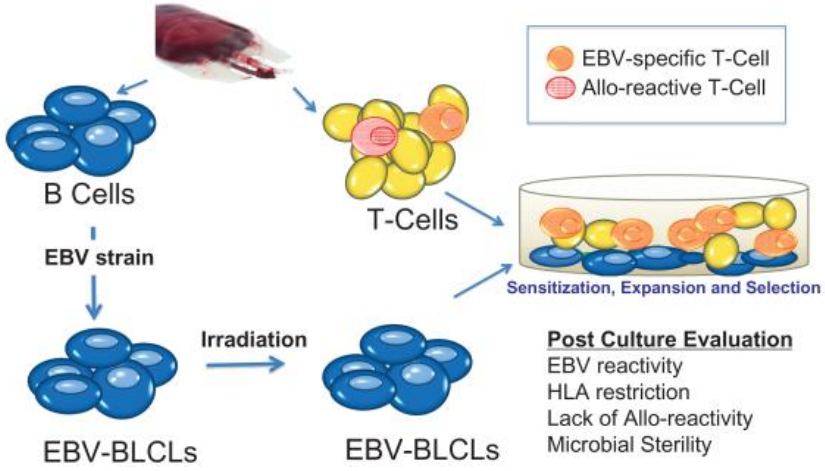
PTLD following SOT

In contrast to PTLD following HSCT, PTLD that occurs after solid organ transplantation is believed to arise from the lymphoid cells of the patient's own hematopoietic system (i.e., host-derived), in a majority of cases, due to the absence of normal T cell immune surveillance. Additionally, the incidence of EBV-lymphoma varies, ranging from 2-10%, depending upon the type of organ transplant (Figure 28). Patients undergoing SOT most commonly develop PTLD after exhibiting latency in the first year. In order to prevent or treat EBV-associated PTLD after SOT, two approaches are used: 1) to remove the infected B-cells; and 2) to restore and expand an EBV-specific T-cell mediated response. Thus, in the setting of SOT, the recommended guidelines include first reducing immune suppression (i.e., remove the immunosuppressive drugs), if possible. If this approach is unsuccessful, then rituximab is also used off label for these patients, either as a single agent or in combination with chemotherapy. The response rate is usually between 44-60%. However, many of these patients, like those following HSCT, are poor candidates for multi-agent chemotherapy. Historical survival for patients who have failed rituximab is only 33% at two years in this setting. For high-risk patients (older than 60 years of age, poor performance status and high LDH), the survival rate is 0% at two years. Therefore, adoptive cell therapy to reconstitute EBV-specific immunity offers an appealing approach for such patients.

ATARA'S T-CELL TECHNOLOGY PLATFORM: EBV TARGETED T CELLS

All three of Atara's T-cell product candidates share a common technology, which entails collection of blood from third-party donors. The EBV-cytotoxic T cell lines are generated by first exposing the B cells to a selected viral antigen, in this case, a laboratory strain of the Epstein-Barr virus 95.8, to activate the cells against that specific virus. The newly transformed B lymphoblastoid cells (EBV BLCLs), presenting with EBV antigen, are irradiated and co-cultured with T cells derived from the blood of the same third-party donor. With this co-culture process, the BLCLs present EBV antigen to the T cells to activate the T cells against the EBV virus. The activated EBV-specific T cells are next stimulated and sensitized weekly for a month and are expanded over that period of time, while the potentially alloreactive T cells are not. At the end of the time period, the cultures are assessed for their EBV specificity and HLA restriction, lack of reactivity and microbial sterility. After full characterization, the cell lines are cryopreserved and stored for future therapeutic use in an appropriate partially HLA (human leukocyte antigen) matched patient, as an off-the-shelf therapy.

FIGURE 29. Ex-vivo Generation of EBV-specific CTL

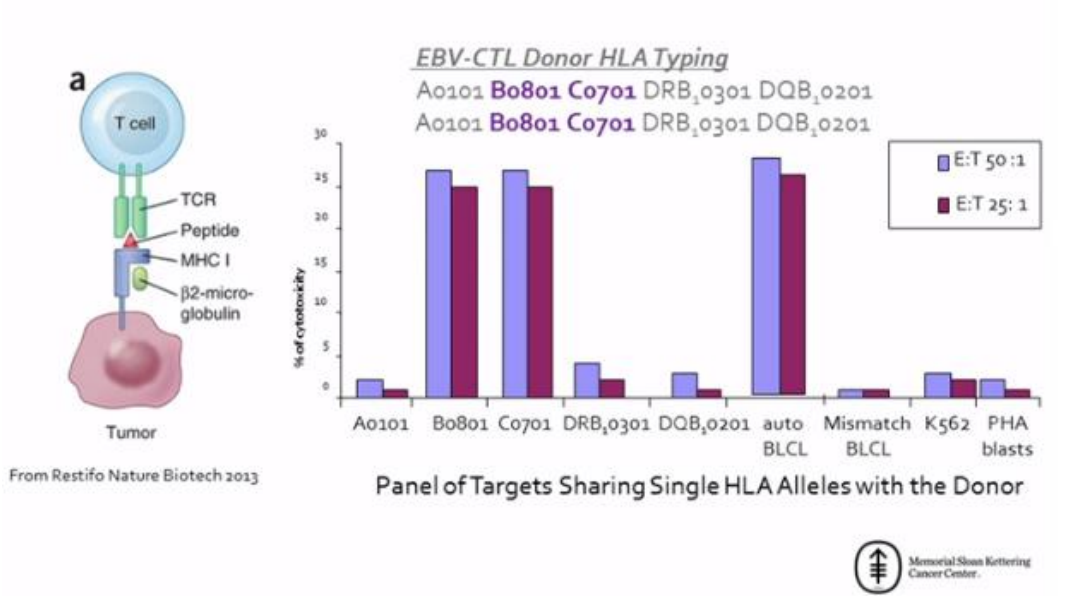


Source: Company Reports

Atara's EBV-CTL therapy offers multiple advantages as an allogenic, off-the-shelf therapeutic option: 1) Patients can have access to treatment, an average of three days from diagnosis, compared to ~8 weeks for an autologous treatment. 2) Cell lines that are generated in vitro may fail to recognize antigens of endogenous EBV, whereas Atara has the bank of 300 lines with multiple specificities to rely upon. 3) In the HLA disparate transplant setting, EBV T cells of donor origin may be restricted through an HLA allele that is not presented by the host lymphoma. 4) The bank consists of over 300 cryopreserved third-party (EBV-CTLs) cell lines, which permit selection of appropriate cell lines based on a partial HLA match at two alleles and desired HLA restriction for >98% of all patients referred. Thus, this large bank also offers the ability to treat an ethnically diverse population. 5) The lines have been depleted of non-virus specific alloreactive cells, which can induce graft versus host disease (GvHD). As such, minimal toxicities, no Grade 2 GvHD or organ rejection has been observed with these cells. 6) Atara has multiple microbiologically stable cells lines available to test in the patient if the first one fails to achieve a response. The bank allows for switching of cell lines if the patient does not respond to the first cell lines; and this has been done successfully a total of seven times. 7) The cells are not genetically re-engineered, which can contribute to other sets of challenges. 8) Maximum durability has been observed with these cells after a median of two cycles, where both CRs and PRs have been durable (unlike what is observed in the chemotherapy setting).

The EBV-CTL platform is based on the ability to use the lines effectively based on knowing the HLA restriction of each line. Therefore, these T cell lines, like all native T cells, recognize their target by seeing EBV epitope in the context of specific HLA alleles. Accordingly, the donor T cell line can be tested for cytotoxicity against EBV-positive targets that share a specific allele, but do not demonstrate any cytotoxicity against targets that do not bear other HLA alleles or EBV-negative targets (Figure 30). In our view, this level of specificity should prevent any off-target killing of cells and limit any side effect.

FIGURE 30. EBV-CTLs are HLA-restricted and Specific

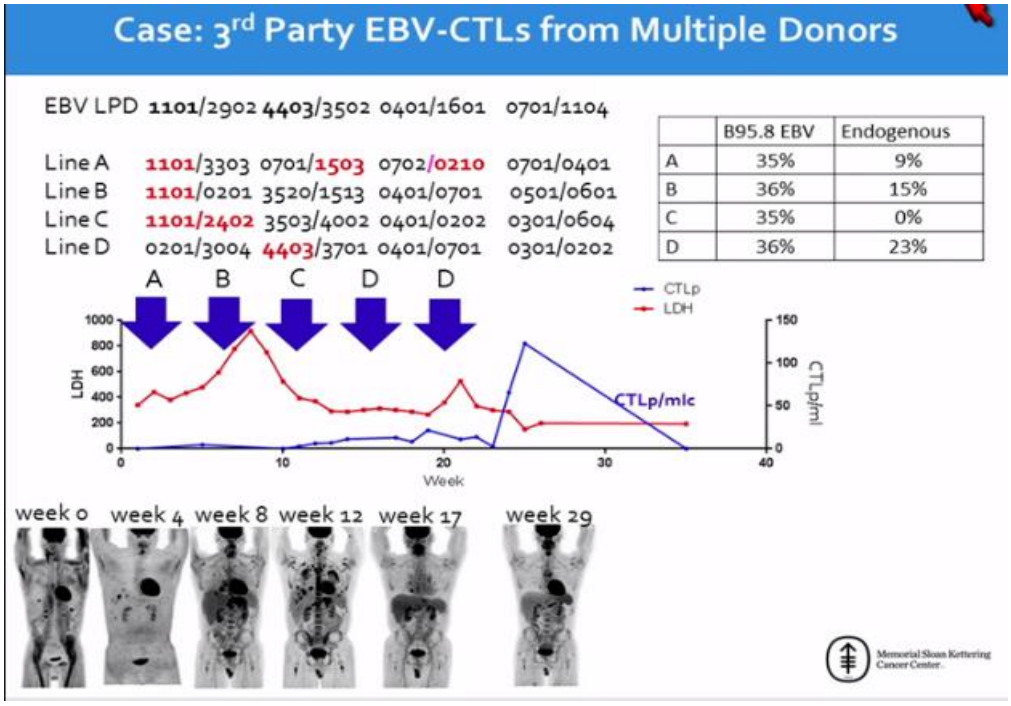


Source: Prockop S. et al., ASCO 2015

The power of the bank

Below is an example that demonstrates the utility of the EBV CTL bank as compared to using an autologous system. It offers the flexibility to switch from one donor T cell line to another which, in our view, sets the platform apart from an autologous treatment. Whereas one line may recognize an EBV epitope that is mutated in the endogenous tumor, switching to a different line that recognizes a different EBV epitope may actually mediate a better response.

FIGURE 31. Switching Capability with Atara's EBV-CTL



Source: Prockop S. et al., ASCO 2015

The above patient developed EBV lymphoma following a transplant for aplastic anemia and progressed through rituximab. After a first cycle of cells, he had a mixed response with improvement shown in nasopharyngeal disease, but demonstrated worsening of hepatic lesions. Subsequently, the patient was treated with two cycles of cells from two different donors through which he progressed. By week 12, the patient had disseminated disease. When he was switched to a fourth donor line D that was restricted by a different HLA allele, he had a complete clinical and radiographic response. In concert with the response in the patient, expansion of EBV-CTLS was observed. Thus far, seven successes have been observed in another ten patients that have been treated with the switching method.

Clinical Development of Atara's EBV-CTL

Atara's most advanced pipeline candidate from the T-cell platform is EBV-CTL, which is in Phase II clinical trials for the treatment of EBV-associated malignancies post HSCT and SOT. To date, the efficacy following treatment with EBV-CTL compares favorably with historical data. In two separate studies of HSCT patients with rituximab refractory PTLD treated with EBV-CTL, the one-year OS was 56% and 72%, whereas historically the median survival has been in the 16-56 day range. The treatment also compares favorably against other studies using similar modalities to address a similar patient population (Figure 32). We note that although there are several private companies (Cell Medica, Adcyte and Viracyte) that have licensed products from Baylor University, and a number of academic institutions that have ongoing similar programs, they are either autologous T-cell treatments, or allogeneic treatments, wherein the T cells have also been genetically modified. In our view, Atara's banked cells offer greater flexibility while demonstrating favorable levels of efficacy and safety.

FIGURE 32. T-cell Therapy for EVB-PTLD

Primary HCT Donor DLI	Patient no.	Response	GvHD	Sponsor
Papadopoulos et al *NEJM* 1994	5	100%	40%	MSKCC
Doubrovina et al *Blood* 2012	30	73%	17%	MSKCC
Primary HCT Donor EBV-CTLs				
Heslop et al *Blood 2010*	13	85%	8%	Baylor
Doubrovina et al *Blood* 2012	14	64	0%	MSKCC
Comoli et al *Blood Cells Mol Dis 2008*	5	100%	0%	Italy
Third Party EBV-CTLs				
Uhlin et al *CID 2012*	1	100%	0%	Sweden
Leen et al *Blood* 2013	9	67%	11%	Baylor
MSKCC *Blood* 2011 and 2013	5	80%	0%	MSKCC
Haque et al*Blood* 2007	33	64%	0%	UK
MSKCC ASCO 2015 (HCT)	34	65%	<1%	MSKCC
MSKCC ASCO 2015 (SOT)	13	62%	0%	MSKCC

Source: Prockop S. et al., ASCO 2015, Company Reports

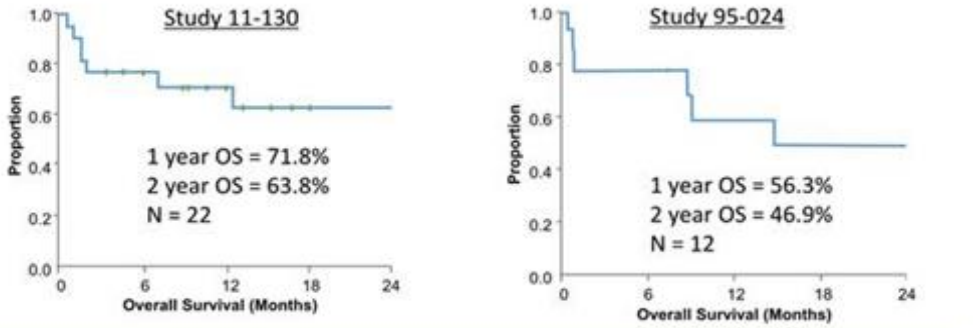
Thirty-four patients, who had reactivation of their EBV that led to lymphomas, were evaluated in a Phase II study. The study was composed of two sequential protocols (95-024 and 11-130) run at MSKCC since 1995. All 34 patients were refractory to prior rituximab treatment after HSCT, while 28 had high-risk disease features. Third-party EBV-CTL lines were matched for at least two of eight HLA alleles and restricted by an allele shared by the lymphoma. Patients received dosing three times weekly of $1x10^6$ cells/kg/dose via IV infusion over five minutes, and were assessed for toxicity, efficacy, and expansion of EBV cytotoxic T cells in vivo. The patients who did not demonstrate toxicity were eligible to receive additional cycles of cells. Maximal responses were observed after a median of two cycles of EBV-CTLs. Given the refractory patient population and the inclusion of high risk patients, the observed 65% overall response rate (ORR) and the disease control rate of 70%, once infused with Atara's T cells, are seen as a positive, in our view. Nineteen of the patients in the trial experienced a CR and three PRs, all of which have been durable.

FIGURE 33. Clinical Proof-of-Concept in EBV-LPD

Response Rate

EBV-CTL Source	N	Prior Rituximab	Response Rate (RR)[2]	Disease Control Rate (DCR)[2]
Third-party Donor[3]	34	34	65%	70%

Overall Survival



Received Breakthrough Therapy designation in February 2015
Pivotal study in rituximab-refractory EBV-LPD after HCT will be conducted under a SPA

[1] Prockop, S et al., Proc ASCO (2015); based on results from two clinical trials of EBV-CTL conducted by MSK
[2] RR = Complete response + partial response; DCR = disease control rate = complete response + partial response + stable disease
[3] Prockop, S et al., Proc ASCO (2015); includes 22 patients from Study 11-130 plus 11 patients from Study 95-024 plus one patient under a treatment IND

Source: Company Reports

The patient population from this study was segmented into two: one that was an older study initiated in 1995 (protocol 95-024) and the second (protocol 11-130) that commenced in 2011 (Figure 33 above). With the newer study, the median overall survival (OS) observed was 71.8% at one year and 63.8% at two years, while the median survival rate has yet to be met. Importantly, no GvHD was observed in these patients that required systemic treatment. Additionally, the treatment appears durable, as none of the patients have had a relapse of their EBV-related lymphomas that required additional treatment (Figure 34).

FIGURE 34. Response to EBV-CTLs

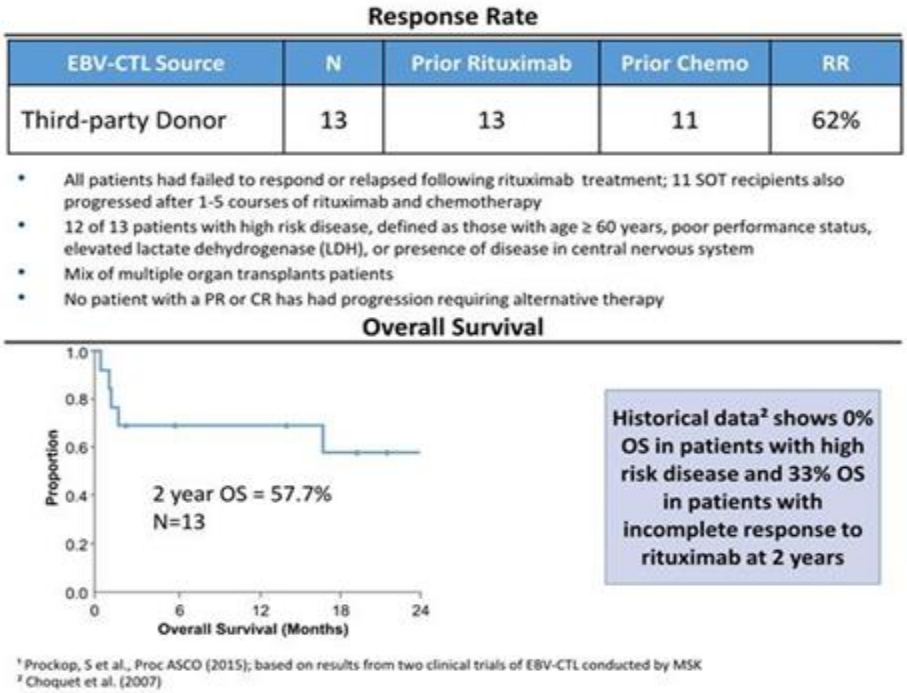
Cohort	N	CR	PR	SD	PD	CR+PR	Duration
HCT Recipients	34	19	3	2	10	65%	CR> 3-5 mo - >66 mo PR 6,>19,>27mo
SOT Recipients	13	1	7	0	5	62%	CR>22 mo PR6,7,17,>6>17>24>114

Source: Prockop S. et al, ASCO 2015, company reports

A second setting where Atara's EBV-CTLs have been tested as proof of concept was in patients that had a reactivation of EBV causing lymphomas following their solid organ transplant. The data were presented at this year's ASCO. In this study, 13 patients were evaluated: all had prior rituximab treatment, 11 had received multi-agent chemotherapy, seven had received R-CHOP or R-CHOP-like therapy, and six had received three or more combination chemotherapy regimens. In addition, 12 of the

SOT patients were considered high risk. Despite these features, the trial had seven PRs and one CR for an ORR of 62%. Of significance, no patient who achieved either a CR or a PR in the study has died from EBV lymphoma. Overall survival was 57.7% after two years, which compares favorably to the historical OS of 33% for those that have failed rituximab and 0% for those in the high-risk group.

FIGURE 35. POC in EBV-LPD After SOT



Source: Company Reports

Safety of EBV-CTLs

Another positive factor of Atara's banked EBV-CTL lines is the minimal toxicities that have been observed in either HSCT (Figure 36) or SOT studies, especially given how mismatched the T cell lines are. Only one episode of Grade 1 GvHD was seen in the combined studies, where the patient responded to topical steroids. In the SOT study, no transplant rejection occurred. Additionally, no de novo cytopenia, cytokine release syndromes or infusion site reactions occurred.

FIGURE 36. Safety of EBV-CTLs in HSCT

Cohort	SAEs	Patients	Related	Grade	Description
95-024	34	19	2 possibly	3	Neurologic
11-130	25	14	5 possibly	3	Febrile neutropenia Metabolic, Hypoxia
			2 possibly	4	Metabolic, Lymphopenia

Source: Company Reports

Another important investment attribute of Atara's EBV-CTL therapy is that it has received Breakthrough Therapy Designation (BTD) from the FDA in February for the treatment of rituximab-refractory EVP-LPD patients following HSCT, which was based on the two above-mentioned clinical trials (HSCT trials) conducted by MSKCC. Based on recent discussions to support a potential approval in this setting, ATRA expects to initiate a pivotal study in rituximab-refractory EVP-LPD patients after HSCT, and anticipates submitting a special protocol assessment (SPA) for the pivotal study as well. The company also plans to initiate another trial for approval in the setting of rituximab-refractory EBV-LPD after SOT.

CURRENT TREATMENT OPTIONS AND COMPETITORS IN DEVELOPMENT

Although the initial indications that Atara is seeking with its EBV-CTL program covers a relatively small patient population (combined U.S. and EU for EBV-LPD in HSCT is only 1,300 and in SOT is 1,700), there is a great unmet need for these patients. As mentioned previously, these patients have limited treatment options, one of which includes off-label use of rituximab or combination chemotherapy regimens. More importantly, there are currently no FDA or EMA-approved products in these indications. There are presently a number of trials that have explored the potential for the EBV-directed treatment directed towards EBV infection, with a majority at the Phase I stage of development. Figure 37 details some of these studies.

A number of the trials are being conducted by academic or private companies –some of which have spun out of the academic institutions. The private companies include: Cell Medica, which has Cytorex EBV, an autologous EBV specific T-cell therapy in a Phase II multi-center NK/T cell lymphoma study; while Adcyte and ViraCyte have licensed virus-specific T cells from Baylor University that are in multiple clinical trials. Given the dearth of options for this patient population, we believe, if Atara's product candidate can show similar positive results in a registration-directed trial, it will likely garner a sizeable portion of the limited, U.S. patient population.

FIGURE 37. Ongoing EBV-Directed Cell Therapies

Sponsor	EBV Directed Therapies	Phase	N	Identifier	Indication
Atara/MSKCC	EBV-CTL	II	112	NCT01498484	EBV-PTLD and EBV malignancies, NHL
Atara/MSKCC	EBV-CTL	I/II	84	NCT00002663	high risk or EBV-PTLD, leukemia, solid tumors
Cell Medica	Autologous EBV T cell	II	30	NCT01948180	Aggressive NK/T-cell lymphoma
MSKCC/Juno	EBV-CTL targeted to CD19	I	26	NCT01430390	ALL after HCT
University of Erlangen-Nürnberg M	Peptide stimulated EBV/CMV T Cells	I/II	50	NCT02227641	After HCT (pre-emptive/preventive)
Baylor	Tgfb resistant EBV cytotoxic T cells	I	14	NCT02065362	EBV-nasopharyngeal carcinoma
Baylor	EBV CTLs	I	19	NCT00085930	Neuroblastoma
Baylor	LMP-specific allogeneic T cells	I	18	NCT01447056	EBV-associated diseases
Baylor	EBV CTLs + GD2 chimeric TCR	I	19	NCT00085930	Neuroblastoma
Baylor	EBV CTLs	II	25	NCT00953420	r/r nasopharyngeal carcinoma
Baylor	LMP-specific allogeneic T cells	I	89	NCT00062868	relapsed lymphoma
Medical College of Wisconsin	Multi-virus CTLs	I	18	NCT01535885	Following HCT
Children's Research Institute	EBV CTLs	I	18	NCT01923766	Prevent Virus Infections
Baylor	LMP, BARF1, EBNA1 specific EBV-CTLs	I	42	NCT02287311	EBV lymphoma
Baylor	Multi-virus specific T cell	I/II	24	NCT01570283	Viral Infection
MSKCC	T Cells targeted to CD19	I	24	NCT01860937	relapsed ALL
Baylor	Allogeniec CTLs	N/A	N/A	NCT01945619	After HCT
Baylor	EBV CTLs	I	136	NCT01555892	EBV Lymphoma
Children's Research Institute	Multi-virus CTLs	I	28	NCT01945814	After HCT
Children's Research Institute	Multi-virus CTLs	I	24	NCT01956084	EBV lymphoma
Baylor	Multi-virus specific T cell	I	50	NCT02108522	After HCT
MSKCC/Juno	Autologous EBV T cell targeted to CD19	I	60	NCT01044069	ALL
New York Medical College	New York Medical College	II	45	NCT01636388	r/r Hodgkins Lymphoma

Source: Company Reports

Market opportunity & commercialization – EBV-PTLD patients following HSCT & SOT

We assume an annual incidence of ~600 EBV-PTLD patients in the U.S. in 2015, projected at a 2.7% growth rate. Currently all EBV-PTLD patients receive rituximab off label in this setting, but show a response rate of up to 50% to this treatment. Given the lack of any standard or durable treatment for this patient population, we see this as the addressable population for Atara's EBV-CTL treatment. While there are no clear guidelines as to the expectations for pricing and reimbursement in this setting, we have made certain assumptions based on other therapies for severe conditions.. We model EBV-CTL pricing at ~$55,000 for a course of treatment, with each course consisting of three infusions dosed over a period of one week. On average, we expect patients to receive two courses of treatment with up to four different cell lines from the cell bank. We assign a 50% market penetration in the HSCT market and 35% penetration in the SOT market where PTLD can be managed by titrating immunosuppression in some individuals. The combined markets yield peak sales of ~$150MM.

Based on our research, we estimate there are ~800 potential patients in the EU as of 2015. Given the sensitivity of the European market to cost as well as Cell Medica's prior presence in the region, we predict that EBV-CTL may compete less aggressively in the region and only capture 24% of the market. We model peak sales for EBV-CTL in the EU in 2025 of $60MM. Worldwide, including Japan, we calculate that there are an additional 1,660 EBV-PTLD patients in 2015. We assume 22% market penetration and model peak 2025 sales of $60MM.

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FIGURE 38. EBV-CTL for Treating PTLD after Hematopoietic Stem Cell Transplantation

US												
EBV-CTL in EBV-LPD after HCT ($MM)	2015E	2016E	2017E	2018E	2019E	2020E	2021E	2022E	2023E	2024E	2025E	2033E
Epidemiology												
Patients undergoing HCT	18,595	19,097	19,613	20,142	20,686	21,245	21,818	22,407	23,012	23,634	24,272	30,038
% growth	*2.7%*	*2.7%*	*2.7%*	*2.7%*	*2.7%*	*2.7%*	*2.7%*	*2.7%*	*2.7%*	*2.7%*	*2.7%*	*2.7%*
% of pts EBV-LPD after HCT	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*
# of Patients with EBV-LPD after HCT	599	615	632	649	666	684	703	722	741	761	782	967
EBV-CTL Penetration				***6.0%***	***12.0%***	***25.0%***	***35.0%***	***45.0%***	***50.0%***	***50.0%***	***50.0%***	***50.0%***
# of Patients on EBV-CTL				39	80	171	246	325	370	381	391	484
number of cycles per year				**1.0**	**2.0**	**2.0**	**2.0**	**2.0**	**2.0**	**2.0**	**2.0**	**2.0**
Total patient cycles per year				**39**	**160**	**342**	**492**	**649**	**741**	**761**	**782**	**967**
Cost per treatment (thousands)			$ 55	$ 57	$ 58	$ 60.10	$ 62	$ 64	$ 66	$ 68	$ 70	$ 88
% price increase			*0%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*
US Sales of EBV-CTL HCT ($MM)				**$2.2**	**$9.3**	**$21**	**$30**	**$41**	**$49**	**$51**	**$54**	**$85**
Royaly rate			10%	10%	10%	12%	12%	12%	12%	12%	12%	12%
US Royalties to MSKCC($MM)				**$ 0.2**	**$ 0.9**	**$ 2.5**	**$ 3.7**	**$ 5.0**	**$ 5.8**	**$ 6.2**	**$ 6.5**	**$ 10.2**

Source: JMP Securities LLC and Company Reports

FIGURE 39. EBV-CTL for Treating PTLD after Solid Organ Transplantation

US												
EBV-CTL in EBV-LPD after SOT ($MM)	2015E	2016E	2017E	2018E	2019E	2020E	2021E	2022E	2023E	2024E	2025E	2039E
Epidemiology												
Patients undergoing SOT	30,770	31,601	32,454	33,331	34,230	35,155	36,104	37,079	38,080	39,108	40,164	49,705
% growth	*2.7%*	*2.7%*	*2.7%*	*2.7%*	*2.7%*	*2.7%*	*2.7%*	*2.7%*	*2.7%*	*2.7%*	*2.7%*	*2.7%*
% of pts EBV-LPD after SOT	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*
# of Patients with EBV-LPD after SOT	769	790	811	833	856	879	903	927	952	978	1,004	1,243
EBV-CTL Penetration				***6.0%***	***12.0%***	***20.0%***	***28.0%***	***35.0%***	***35.0%***	***35.0%***	***35.0%***	***35.0%***
# of Patients on EBV-CTL				50	103	176	253	324	333	342	351	435
number of cycles per year				**1.0**	**2.0**	**2.0**	**2.0**	**2.0**	**2.0**	**2.0**	**2.0**	**2.0**
Total patient cycles per year				**50**	**205**	**352**	**505**	**649**	**666**	**684**	**703**	**870**
Cost per cycle per year (thousands)		$ 55	$ 55	$ 57	$ 58	$ 60	$ 62	$ 64	$ 66	$ 68	$ 70	$ 88
% price increase			*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*
US Sales of EBV-CTL SOT ($MM)				**$2.8**	**$12.0**	**$21.1**	**$31.3**	**$41.4**	**$43.8**	**$46.3**	**$49.0**	**$76.8**
Royaly rate				10%	10%	12%	12%	12%	12%	12%	12%	12%
US Royalties to MSKCC($MM)				**$ 0.3**	**$ 1.2**	**$ 2.5**	**$ 3.8**	**$ 5.0**	**$ 5.3**	**$ 5.6**	**$ 5.9**	**$ 9.2**

Source: JMP Securities LLC and Company Reports

CMV targeted t-cells

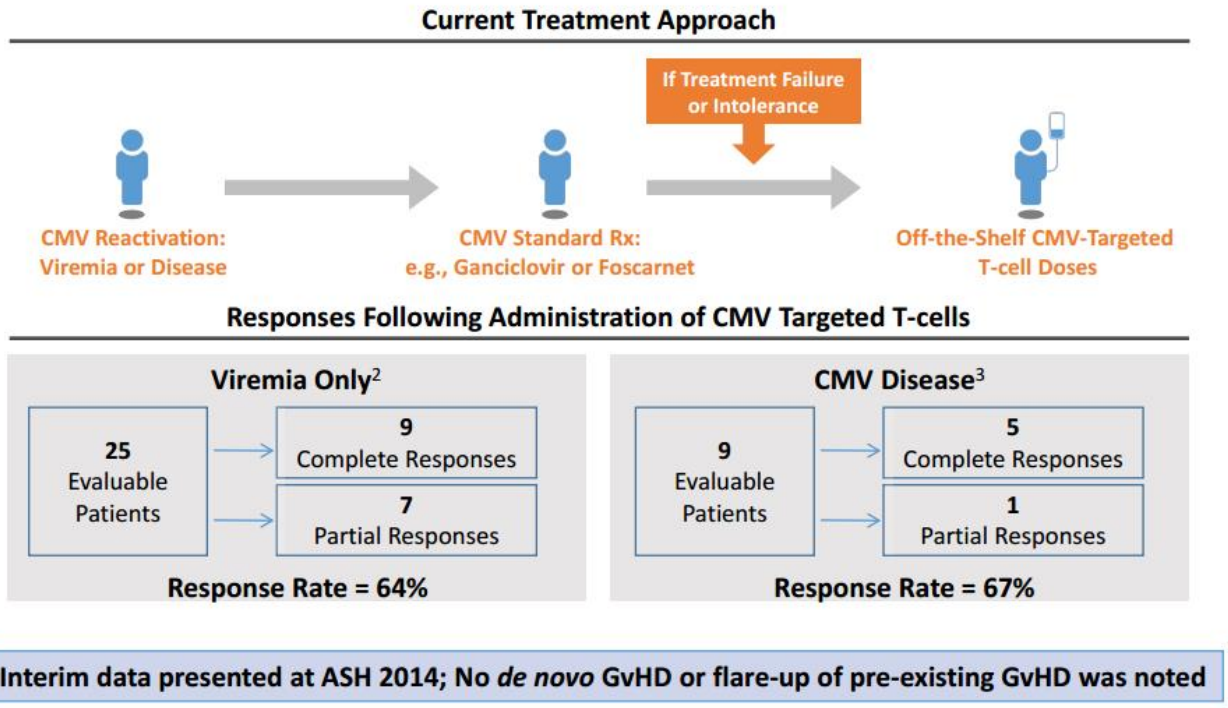
Atara's second T-cell candidate, CMV-CTL, targets cytomegalovirus. Also known as HHV5, CMV is a member of the Herpes virus family. Similar to EBV, CMV is a common virus that infects people of all ages, leading to life-long persistence, while generally remaining "silent" or in the latent state of infection (i.e., without showing any signs or symptoms). According to the CDC, the frequency of infection is in the range of 50% to 80% in the general adult population. Importantly, as with EBV, the reactivation of CMV in an immune-compromised state can cause severe and often fatal disease in people with a weakened immune system, including recipients of organ transplants, AIDS patients and in babies infected before birth. Depending on the tissue of those with a compromised immune system, CMV infection may cause CMV retinitis (leading to blindness), encephalopathy, interstitial pneumonia, gastrointestinal infection, central nervous system disease, hepatitis, retinitis, and encephalitis, prolonged hospitalizations of patients or even death. Therefore, immunocompromised patients, such as HSCT and SOT patients, are at the highest risk for developing significant disease syndromes from CMV. About one in 150 children is born with congenital CMV infection, which leads to nearly 400 deaths per year, or leaves ~8,000 children with permanent disabilities, including hearing loss, vision loss or developmental disabilities in the U.S. In the oncology setting, CMV is also associated with certain malignancies such as glioblastoma multiforme (GMB). Ninety-five percent of GBM tumors have been shown to express CMV. Even in a healthy individual, the virus can routinely reactivate, but is generally kept in check by the host immune response.

An estimated 2,000 patients in the U.S. and Europe combined have CMV infection following HSCT, the first indication which Atara expects to enter with its CMV-CTL treatment. Although there have been advances made in the detection as well as the management of CMV infections, the virus still presents a threat to immunocompromised patients. Currently, a patient is given small molecule antiviral therapies, provided prophylactically, which have reduced morbidity and mortality. However, undertaking those treatments can often lead to serious side effects, including neutropenia (when using ganciclovir, valganciclovir and cidofovir) and renal toxicity (when using foscarnet and cidofovir). Many post-HSCT patients may still progress to developing symptomatic CMV viral diseases such as retinal infections (CMV retinitis) with antiviral treatments. Additionally, the use of antiviral drugs has led to the emergence of resistance which has also presented a challenge to patient care.

Clinical evidence with Atara's CMV-CTL

To date, MSK has conducted one Phase I study and two Phase II clinical trials with the CMV-CTL treatment, which included patients with significant CMV viral counts (CMV viremia) before symptoms arose and overt CMV disease, where both types of patients were resistant to antiviral drug treatment. Compelling data from an interim look at the studies were presented at ASH 2014.

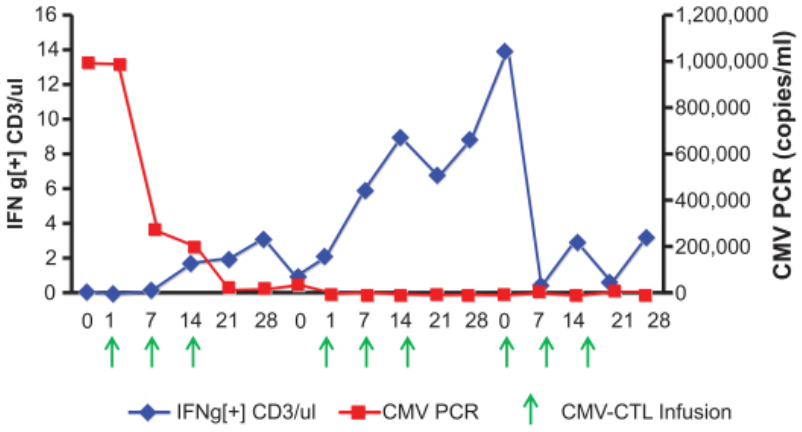
FIGURE 40. Phase II Proof of Concept in Anti-Viral Resistant CMV after HSCT



Source: Company Reports

Results from the ongoing Phase II studies have demonstrated similar efficacy in patients with refractory CMV infection after HSCT. The two Phase II studies combined had a total of 38 patients, who had failed a median of 113 days of prior antiviral therapy and a median of four antiviral therapies. Updated results in January (Figure 40) from MSKCC revealed that those patients with detectable viremia only had a response rate of 64% (including nine CRs and seven PRs), while those with symptomatic CMV disease had a 67% response rate (including five CRs and one PR), corresponding to RRs observed in the EBV studies with EBV-CTLs. Mechanistically, the effects of CMV-CTL administration can be seen in one patient example shown in Figure 41, where the drop in blood CMV DNA can correlates with the expansion in a population of activated interferon-gamma (IFN-γ) producing CMV-CTL. Importantly, the third-party matched donor cells were well tolerated in these studies with no de novo GvHD or even activation of prior GvHD associated with CMV-CTL infusion. In our view, the fact that these patients exhibited antiviral resistance resulting in 60+% response rates with the CMV-CTL treatment speaks to the power of the platform.

FIGURE 41. Patient Blood CMV DNA Count After CMV-CTL Infusion



Source: Company Reports

Market opportunity for CMV-CTL

Although multiple anti-virals are in the market for targeting CMV infection (such as Cytovene IV (ganciclovir), Valcyte (valganciclovir), Foscavir (foscarnet) and Vistide (cidofovir)), resistance to these agents has also become an increasing concern in immunocompromised patient populations with prolonged use. We believe that opportunities exist in CMV-expressing infections that are not adequately addressed by current therapies. CMV-CTL may also be superior to the some of the agents in development, although we also note the crowded space for the indication with the currently approved drugs and those in clinical development.

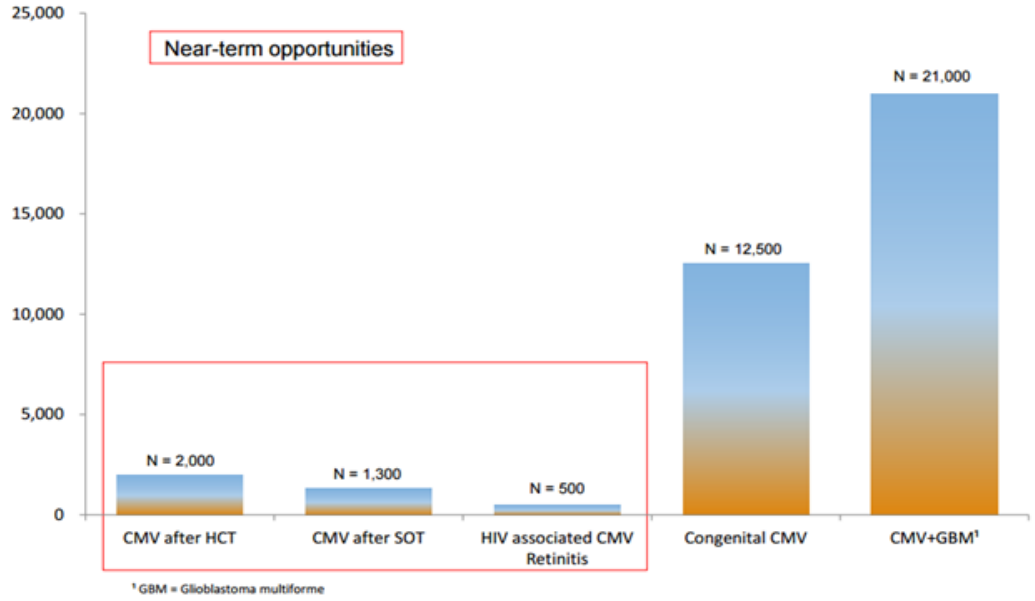
The latter group includes: Maribavir, a UL97 protein kinase inhibitor from Shire (SHPG, NC), which has already failed one Phase III study in liver transplant patients; letermovir (MK-8228), a CMV terminase inhibitor that is currently in Phase III trials from Merck (MRK, NC); and brincidofovir (CMX001), a lipid conjugated nucleotide analogue of cidofovir in development at Chimerix (CMRX, NC) that is also in Phase III trials. Other products in development that could directly compete with CMV-CTL, if approved, include: ASP0113, a DNA vaccine encoding CMV phosphoprotein 65 and glycoprotein B antigens from Astellas; Cytovir CMV, a CMV-specific cell therapy derived from primary HSCT transplant donors from Cell Medica (Private); as well as Adcyte (Private) and ViraCyte (Private) that have licensed virus-specific T cells from Baylor University that are in clinical trials. There may be near-term opportunities for Atara in the setting of CMV-PTLD (from reactivation of CMV infection) after HSCT and after SOT, in our view.

Revenue projections for CMV-CTL

In determining the market opportunity for the program, we initially model CMV-CTL use as second-line therapy in two indications: CMV-PTLD after HSCT and CMV-PTLD after SOT, assuming an annual incidence rate of ~1,025 patients in the U.S. at market launch beginning in 2021 receiving an average of three doses of therapy. We estimate sales in the U.S. approaching $50MM by 2033 for CMV-PTLD after HSCT. We also model our estimate peak sales in CMV-PTLD after SOT, since the company has

guided to potentially entering in this indication. In CMV-PTLD after SOT, we model an annual incidence of ~700 in 2018 for the U.S. launch. Given the crowded space in the setting, we model our pricing at a discount to the EBV-CTL product at $45,000 yielding peak revenues of $30MM. Market projections by locale and indication are detailed in Figure 42. The much larger market opportunity for CMV-CTL lies in treating CMV positive malignancies such as GBM. We currently model CMV-CTL capturing 40% of the U.S. market in GBM that has progressed after primary radiation and temozolomide therapy and second-line Avastin treatment. We model this market to yield worldwide revenues of ~$400MM by 2033. We note that we use a 35% discount rate on this program since the clinical data are indirect with respect to GBM. However, if the therapy does demonstrate efficacy, there is significant upside potential for moving into early stages of treatment.

FIGURE 42. Near-Term and Expansion Indications for CMV-CTL



Source: Company Reports

FIGURE 43. CMV-CTL for Treating CMV Infection After Hematopoietic Stem Cell Transplantation

US												
CMV-CTL HCT in CMV-LPD after HCT ($MM)	2015E	2016E	2017E	2018E	2019E	2020E	2021E	2022E	2023E	2024E	2025E	2033E
Epidemiology												
Patients undergoing HCT	18,595	19,097	19,613	20,142	20,686	21,245	21,818	22,407	23,012	23,634	24,272	30,038
% growth	*2.7%*	*2.7%*	*2.7%*	*2.7%*	*2.7%*	*2.7%*	*2.7%*	*2.7%*	*2.7%*	*2.7%*	*2.7%*	*2.7%*
% of pts CMV-LPD after HCT	*5%*	*5%*	*5%*	*5%*	*5%*	*5%*	*5%*	*5%*	*5%*	*5%*	*5%*	*5%*
# of Patients with CMV-LPD after HCT	874	898	922	947	972	998	1,025	1,053	1,082	1,111	1,141	1,412
CMV-CTL Penetration				***3.0%***	***9.0%***	***16.0%***	***22.0%***	***25.0%***	***25.0%***	***25.0%***	***25.0%***	***25.0%***
# of Patients on CMV-CTL HCT				28	88	160	226	263	270	278	285	353
number of doses per year				**1**	**4**	**4**	**4**	**4**	**4**	**4**	**4**	**4**
Total patient doses per year				**28**	**350**	**639**	**902**	**1,053**	**1,082**	**1,111**	**1,141**	**1,412**
Cost per cycle per year (thousands)		$ 30	$ 31	$ 32	$ 33	$ 34	$ 35	$ 36	$ 37	$ 38	$ 39	$ 50
% price increase			*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*
US Sales of CMV-CTL HCT ($MM)			**$0.0**	**$0.9**	**$11.5**	**$21.6**	**$31.4**	**$37.7**	**$39.9**	**$42.2**	**$44.7**	**$70.0**
Royaly rate							12%	12%	12%	12%	12%	12%
US Royalties to MSKCC($MM)						**$ -**	**$ 3.8**	**$ 4.5**	**$ 4.8**	**$ 5.1**	**$ 5.4**	**$ 8.4**

Source: JMP Securities LLC and Company Reports

FIGURE 44. CMV-CTL for Treating CMV Infection After Solid Organ Transplantation

US												
CMV-CTL SOT in CMV-LPD after SOT ($MM)	2015E	2016E	2017E	2018E	2019E	2020E	2021E	2022E	2023E	2024E	2025E	2033E
Epidemiology												
Patients undergoing SOT	30,770	31,601	32,454	33,331	34,230	35,155	36,104	37,079	38,080	39,108	40,164	49,705
% growth	*2.7%*	*2.7%*	*2.7%*	*2.7%*	*2.7%*	*2.7%*	*2.7%*	*2.7%*	*2.7%*	*2.7%*	*2.7%*	*2.7%*
% of pts CMV-LPD after SOT	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*
# of Patients with CMV-LPD after SOT	615	632	649	667	685	703	722	742	762	782	803	994
EBV-CTL Penetration				***3.0%***	***9.0%***	***15.0%***	***18.0%***	***22.0%***	***22.0%***	***22.0%***	***22.0%***	***22.0%***
# of Patients on CMV-CTL				20	62	105	130	163	168	172	177	219
number of doses per year				**1**	**4**	**4**	**4**	**4**	**4**	**4**	**4**	**4**
Total patient doses per year				**20**	**246**	**422**	**520**	**653**	**670**	**688**	**707**	**875**
Cost per cycle per year (thousands)		$ 25	$ 26	$ 27	$ 27	$ 28	$ 29	$ 30	$ 31	$ 32	$ 33	$ 41
% price increase			*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*
US Sales of CMV-CTL SOT ($MM)				**$0.5**	**$6.7**	**$11.9**	**$15.1**	**$19.5**	**$20.6**	**$21.8**	**$23.1**	**$36.1**
Royaly rate							12%	12%	12%	12%	12%	12%
US Royalties to MSKCC($MM)						**$ -**	**$ 1.8**	**$ 2.3**	**$ 2.5**	**$ 2.6**	**$ 2.8**	**$ 4.3**

Source: JMP Securities LLC and Company Reports

FIGURE 45. CMV-CTL for Treating GBM

US												
CTL-CMV Revenues, Resectable Recurrent GBM ($MM)	2015E	2016E	2017E	2018E	2019E	2020E	2021E	2022E	2023E	2024E	2025E	2033E
GBM incidence, US	8,500	8,585	8,671	8,758	8,845	8,934	9,023	9,113	9,204	9,296	9,389	10,167
% growth	*1%*	*1%*	*1%*	*1%*	*1%*	*1%*	*1%*	*1%*	*1%*	*1%*	*1%*	*1%*
% pts receiving chemoradiation (Rad plus Temodar)	*95%*	*95%*	*95%*	*95%*	*95%*	*95%*	*95%*	*95%*	*95%*	*95%*	*95%*	*95%*
# pts first-line pts treated with chemoradiation	8,075	8,156	8,237	8,320	8,403	8,487	8,572	8,657	8,744	8,831	8,920	9,659
% patients refractory or intolerant to chemoradiation	*9%*	*9%*	*9%*	*9%*	*9%*	*9%*	*9%*	*9%*	*9%*	*9%*	*9%*	*9%*
# pts refractory or intolerant to chemoradiation	727	734	741	749	756	764	771	779	787	795	803	869
% pts with recurrent disease at 1 year	*73%*	*73%*	*73%*	*73%*	*73%*	*73%*	*73%*	*73%*	*73%*	*73%*	*73%*	*73%*
# of patients with recurrent disease at 1 year	5,895	5,954	6,013	6,073	6,134	6,195	6,257	6,320	6,383	6,447	6,511	7,051
Addressable second-line patient population	6,621	6,688	6,755	6,822	6,890	6,959	7,029	7,099	7,170	7,242	7,314	7,920
% recurrent pts receiving avastin therapy	*50%*	*50%*	*50%*	*50%*	*50%*	*50%*	*50%*	*50%*	*50%*	*50%*	*50%*	*50%*
CTL-CMV target patient population	3,311	3,344	3,377	3,411	3,445	3,480	3,514	3,550	3,585	3,621	3,657	3,960
Market penetration						*4%*	*12%*	*20%*	*30%*	*35%*	*38%*	*40%*
Patients receiving CTL-CMV therapy				0	0	139	422	710	1,076	1,267	1,390	1,584
Number of doses per year				3	6	6	6	6	6	6	6	6
Total number of patient doses				0	0	835	2,530	4,259	6,453	7,604	8,338	9,504
Cost per dose				$27	$27	$28	$29	$30	$31	$32	$33	$41
% price increase				*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*
Total CTL-CMV Sales, US				**$ -**	**$ -**	**$ 23.5**	**$ 73.3**	**$ 127.2**	**$ 198.4**	**$ 240.8**	**$ 272.0**	**$ 392.7**
Royalties to MSKCC					**$ -**	**$ 2.3**	**$ 7.3**	**$ 12.7**	**$ 19.8**	**$ 28.9**	**$ 32.6**	**$ 47.1**
% Royalty					*10%*	*10%*	*10%*	*10%*	*10%*	*12%*	*12%*	*12%*

Source: JMP Securities LLC and Company Reports

WT1 targeted t cells

Atara's third clinical stage T-cell product candidate, WT1-CTL, targets cancers expressing the antigen Wilms Tumor 1 (WT1). Originally isolated as a tumor suppressor gene that was inactivated in a subset of Wilms tumors (a rare kidney cancer that afflicts children), the WT1 gene has been found subsequently to be overexpressed in a variety of human cancers of both hematologic and non-hematologic origin, including: multiple myeloma (MM), acute myeloid leukemia (AML), non-small cell lung, breast, pancreatic, ovarian cancer, and colorectal cancers. WT1 is a nuclear protein that is inaccessible to classical antibody therapy, but has been recognized as a promising target for immunotherapy. There is strong evidence that WT1 specific CTL can suppress AML. Conversely, we note that allogeneic T cells (not WT1 specific) have demonstrated activity in AML, including five complete remissions in patients infused with haploidentical allogeneic T cells (Colvin, BBMT, 2009). Importantly, this effect did not require engraftment. Not surprisingly, numerous groups are developing therapies targeting WT1 (Figure 47).

Atara's product candidate is currently in two separate Phase 1 clinical trials being sponsored and conducted by MSK, using primary HSCT donor derived WT1-CTLs that are manufactured from the blood of third-party donors. The first trial with WT1-CTL is a 36-patient, open-label, dose-escalation study in patients with residual or relapsed leukemia following allogeneic hematopoietic progenitor cell transplantation (NCT00620633). The primary endpoint is assessment of toxicity of the in vitro expanded WT-1 specific T cells derived from transplant donors. The second is also an open-label Phase I safety study (n=21) for patients with relapsed or refractory MM following T-cell depleted allogeneic HSCT. Primary endpoints include assessment of toxicities and determination of a maximum-tolerated dose.

Top-line results were released today (9/24/15), demonstrating promising signs of activity. Seven patients with relapsed/refractory multiple myeloma (including plasma cell leukemia) were treated with WT1-CTL following allogeneic HSCT. The WT1 antigen was present on the malignant plasma cells in the treated patients. Each patient received three WT1-CTL infusions. By one year, three patients achieved complete remission, one achieved a partial response, two exhibited stable disease, and only one had progressive disease. This promising initial data bodes well for ATRA's efforts to treat these hematologic malignancies with WT1-CTL. Given the lack of options for elderly patients with AML, we think Atara's WT1-CTL could achieve a high level of penetrance (55%) if it demonstrates clinical activity. We currently model the WT1 program generating $500MM in U.S. sales by 2033 with a 35% discount rate. Additionally, WT1-CTL may be tested in combination with other agents in solid cancers. The ability to treat multiple myeloma patients represents further upside to our model.

FIGURE 46. WT1-CTL for Treating AML

US												
WT1-CTL for Elderly AML ($MM)	**2015E**	**2016E**	**2017E**	**2018E**	**2019E**	**2020E**	**2021E**	**2022E**	**2023E**	**2024E**	**2025E**	**2033E**
Elderly AML												
AML indicence, US	19,143	19,430	19,721	20,017	20,318	20,622	20,932	21,246	21,564	21,888	22,216	25,402
%Growth	*1.5%*	*1.5%*	*1.5%*	*1.5%*	*1.5%*	*1.5%*	*1.5%*	*1.5%*	*1.5%*	*1.5%*	*1.5%*	*1.5%*
% pts > 70 yrs of age	*44.5%*	*44.3%*	*44.0%*	*43.8%*	*43.5%*	*43.3%*	*43.0%*	*42.8%*	*42.5%*	*42.3%*	*42.0%*	*41.3%*
AML expressing elevated WT1	*80.0%*	*80.0%*	*80.0%*	*80.0%*	*80.0%*	*80.0%*	*80.0%*	*80.0%*	*80.0%*	*80.0%*	*80.0%*	*80.0%*
Addressable ederly AML patient population	6,815	6,878	6,942	7,006	7,071	7,135	7,200	7,266	7,332	7,398	7,465	8,383
Market penetration					***0%***	***18%***	***26%***	***35%***	***48%***	***55%***	***55%***	***55%***
Elderly AML patients on WT1-CTL	-	-	-	-	-	1,284	1,872	2,543	3,519	4,069	4,106	4,610
Duration of therapy (cycles)		5.0	6.0	7.0	7.0	7.0	7.0	7.0	7.0	7.0	7.0	7.0
Total patient cycles on therapy	-	-	-	-	-	8,991	13,105	17,802	24,635	28,483	28,739	32,273
Cost of therapy (per cycle)		$ 12,000	$ 12,360	$ 12,731	$ 13,113	$ 13,506	$ 13,911	$ 14,329	$ 14,758	$ 15,201	$ 15,657	$ 17,109
% price increase			*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*
US sales of WT1-CTL		**$ -**	**$ -**	**$ -**	**$ -**	**$ 121.4**	**$ 182.3**	**$ 255.1**	**$ 363.6**	**$ 433.0**	**$ 450.0**	**$ 552.2**
Royalties to MSKCC					**$ -**	**$ 12.1**	**$ 18.2**	**$ 25.5**	**$ 36.4**	**$ 52.0**	**$ 54.0**	**$ 66.26**
% Royalty					*10%*	*10%*	*10%*	*10%*	*10%*	*12%*	*12%*	*12%*

Source: JMP Securities LLC and Company Reports

FIGURE 47. WT1 Vaccines and/or T-cell Directed Therapies

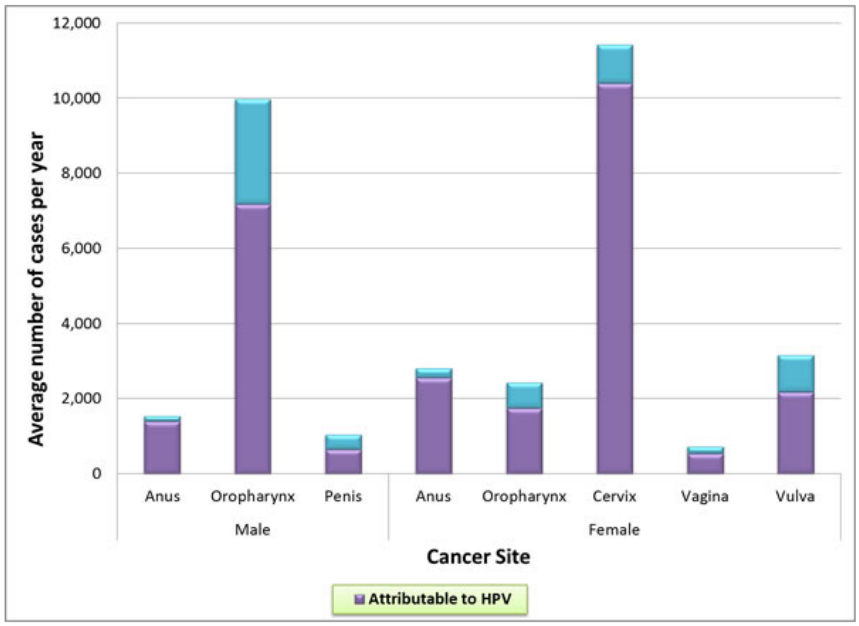
Sponsor	WT1 Directed Therapies	Phase	N	Identifier	Indication
MSKCC	WT1 Analog Peptide Vaccine	II	29	NCT01266083	AML,ALL
Cell Therapy Catapoult	WT1 TCR Gene Therapy	I/II	18	NCT01621724	AML,CML
MSKCC	WT1 Analog Peptide Vaccine	N/A	20	NCT01827137	MM following HCT
MD Anderson	WT1 Analog Peptide Vaccine	II	60	NCT01890980	MPM after MSK10-134
MSKCC	WT1 Analog Peptide Vaccine	II	60	NCT01265433	MPM
Fred Hutchinson	WT1 TCR Gene Therapy	I/II	20	NCT02408016	NSCLC or Mesothelioma
Fred Hutchinson	WT1 TCR Gene Therapy	I/II	55	NCT01640301	AML,MDS,CML after HCT
Atara/MSKCC	WT1-sensitized T Cells	I	36	NCT00620633	r/r Leukemia after HCT
MSKCC	WT1 Dendritic Cells	I	20	NCT01995708	MM undergoing HCT
Atara/MSKCC	WT1-sensitized T Cells	I	21	NCT01758328	MM after HCT

Source: JMP Securities LLC and Company Reports

Expansion of the t-cell platform

Although ATRA is beginning with treatment in the post-HSCT and post-SOT settings with its T-cell platform, the technology has utility in a variety of prevalent diseases and beyond the current set of targets (EBV, CMV and WT1-targeted conditions). To that end, ATRA anticipates collaborating with MSK on further research to develop additional cellular therapies, which could include T-cell programs targeted against other antigens and/or CAR-T (chimeric antigen receptor) cell programs, which the company has the option to license. ATRA has guided to the possible development of cellular therapies directed toward other viral targets, such as human papilloma virus (HPV) that is associated with cervical, anal, and head and neck cancers. According to the Centers for Disease Control and Prevention (CDC), HPV causes about 26,900 cancers each year (Figure 48).

FIGURE 48. Average Number of Cancers in Sites Where HPV is Often Found



Source: http:///www.cdc/gov

Another potential viral target under consideration is the John Cunningham (JC) virus, which causes infection of the brain and spinal cord (progressive multifocal leukocephalopathy, PML) in people with compromised immune systems, such as those with AIDS. In PML, the virus causes damage to the white matter of the brain, stripping nerve cells of their insulation (myelin). Others with a weakened immune system, with autoimmune conditions such as multiple sclerosis or rheumatoid arthritis are also at risk for PML. The virus is also associated with a subset of tumors (in pulmonary, digestive organs and in brain tumors).

Normally harmless, the JC virus is very common, often acquired in childhood, and is believed to be found in up to 85% of the general adult population. According to the National Institute of Neurological Disorders and Stroke (NINDS), 30% to 50% of patients with PML die within months (within one to nine months) of diagnosis, and those who survive may become severely disabled. It is estimated that 4,000 people develop PML in the U.S. and Europe combined each year, and have no effective treatment for the condition. We have not yet ascribed any value to either a possible HPV or JC program.

INTELLECTUAL PROPERTY

PINTA-745 and STM-434

Atara relies on a combination of patents, trade secrets, and confidentiality agreements to protect the intellectual property related to its technology and product candidates. For the two most advanced molecularly targeted product candidates, PINTA-745 and STM-434, Atara owns or licenses a number of issued patents and pending patent applications covering the product candidates' compositions of matter and methods of use. For PINTA-745, the anticipated expiration dates range from 2026 to 2035 for U.S. patents and patent applications, if issued, and from 2023 to 2035 for patents and patent applications, if issued, in jurisdictions outside the United States, exclusive of possible patent term extensions. For STM-434, the anticipated expiration dates range from 2027 through 2035 for U.S. patents and patent applications, if issued, and from 2026 through 2035 for patents and patent applications, if issued, in jurisdictions outside the United States, exclusive of possible patent term extensions.

Allogeneic t-cell therapies

The T-cell product candidates and platform technology that Atara has licensed from MSK are protected primarily as confidential know-how and trade secrets. If Atara does not adequately protect its intellectual property, competitors may be able to use the technologies and erode or negate any competitive advantage Atara may have. The patentability of inventions and the validity, enforceability, and scope of patents in the biotechnology field are generally uncertain because they involve complex legal, scientific and factual considerations, and have in recent years been the subject of significant litigation.

SUMMARY AND CONCLUSION

Atara is a biotherapeutic drug development company with two distinct platforms. One platform is based on a portfolio of bio-molecules that target TGF-β family members while the other platform takes advantage of recent developments in allogeneic cell-based therapies. In our opinion, this diversification represents a plus for the company since it offers multiple shots on goal with therapeutic candidates that have the potential to capture a significant market share of diseases that affect large populations. Moreover, these platforms come validated with clinical data that demonstrate compelling signs of efficacy. We anticipate that Atara will emerge as a leader in the advancing field of therapies to treat wasting diseases, including PEW and cancer cachexias, and we believe it also has the potential to make inroads in the competitive oncology space with groundbreaking new therapeutics.

APPENDIX A – MANAGEMENT TEAM

Senior Management

Dr. Isaac Ciechanover, M.D., President and CEO, founded Atara Biotherapeutics in August 2012. Previously, he was a partner in the life sciences practice at the venture capital firm Kleiner Perkins Caufield & Byers. As Celgene's Executive Director for Business Development, Dr. Ciechanover led the company's venture capital efforts and executed licensing and M&A activities with an aggregate value of more than $6.7 billion. He also served as Global Project Leader for the company's first clinical-stage biologic therapy. Dr. Ciechanover has also held business development and venture capital roles at Amylin Pharmaceuticals, Pequot Ventures' healthcare practice and Pfizer.

Dr. Christopher Haqq, M.D., Ph.D., Chief Medical Officer, brought 20 years of clinical, academic and drug development experience from biopharma companies when he joined Atara Biotherapeutics in September 2012. He previously served as Vice President for Clinical Research and Development at Cougar Biotechnology and Johnson & Johnson's Janssen, where he led a pivotal prostate cancer study supporting market approval for Zytiga® (abiraterone acetate). Dr. Haqq also worked at Amgen, where he directed early development studies for AMG 479 (ganitumab), an antibody against the insulin-like growth factor type 1 receptor. He has served as medical monitor on over 10 clinical trials and has contributed to drug development programs for a variety of molecules. Dr. Haqq has experience dealing with the European Medicines Agency, the U.S. FDA and other global regulatory agencies–filing IND applications, new drug applications, special protocol assessments and their international equivalents. Dr. Haqq got his start as a medical oncologist and principal investigator as an Assistant Adjunct Professor in the Division of Hematology/Oncology at the University of California, San Francisco.

John F. McGrath, Jr., Chief Financial Officer, joined Atara Biotherapeutics in January 2013. Prior to joining Atara, he served as Executive in Residence and Operating Partner at Kleiner Perkins Caufield & Byers. Mr. McGrath also held the positions of Vice President and Chief Financial Officer for Network Equipment Technologies, Inc., a company dedicated to developing network equipment for real-time communications. Earlier in his career, he was Vice President of Finance for Aspect Communications, Director of Finance for TCSI Corporation and Manager in the High Technology and Manufacturing practice at Ernst & Young. Mr. McGrath has served on the board of the Presidio Fund, a publicly traded mutual fund, and as Audit Committee Chairman on the boards of Actel and Endwave Corporation.

Gad Soffer, Chief Operating Officer, joined Atara Biotherapeutics in March 2013. Previously at Celgene, he led product development and lifecycle management for Abraxane. He also advised on venture investment and strategic partnerships resulting in ~$6 billion in business development transactions. Mr. Soffer also worked as a healthcare consultant with Easton Associates.

Mitchall Clark, Chief Regulatory and Quality Assurance Officer, brings over 30 years of global Regulatory Affairs, Quality Assurance, and Drug Development experience in the pharmaceutical industry to Atara. He has held senior management roles at a number of organizations including Schering AG (based in its UK and Berlin offices), American Pharmaceutical Partners (APP), Abraxis Bioscience, Celgene Corporation, and NantPharma, LLC. At NantPharma, LLC he established the Regulatory Affairs and Quality Assurance departments. As Senior Vice President of Global Regulatory Affairs at Abraxis, he led the regulatory team that filed the initial IND and obtained global regulatory approvals of Abraxane®.

Source: Company website

FIGURE 49. ATRA Income Statement

Income Statement ($MM)	2014A	1Q15A	2Q15E	3Q15E	4Q15E	2015E	2016E	2017E	2018E	2019E	2020E	2021E	2022E	2023E	2024E	2025E	2033E
Product Sales and Royalties																	
Total Product Sales and Royalties	**0.00**	0.00	0.00	0.00	0.00	0.00	0.00	0.00	18.45	201.25	581.30	1,095.58	1,806.32	2,778.75	3,739.26	4,649.99	6,576.26
Total Revenue		0.00	0.00	0.00	0.00	0.00	0.00	0.00	18.45	201.25	581.30	1,095.58	1,806.32	2,778.75	3,739.26	4,649.99	6,576.26
Royalties Paid																	
Total Royalties & Milestones						0.50	2.50	1.00	4.13	16.62	48.53	84.05	128.37	184.28	242.23	293.28	427.75
Cost of goods sold		0.00	0.00	0.00	0.00	0.00	0.00	0.00	2.77	30.19	87.19	164.34	270.95	416.81	560.89	637.41	900.53
Total Costs	*0.00*	0.00	0.00	0.00	0.00	0.50	2.50	1.00	6.89	46.80	135.73	248.39	399.32	601.10	803.12	930.68	1,328.28
Gross Profit	**0.00**	0.00	0.00	0.00	0.00	(0.50)	(2.50)	(1.00)	11.56	154.45	445.57	847.19	1,407.00	2,177.65	2,936.13	3,719.31	5,247.98
Operating expenses:																	
Research and development	*14.38*	5.77	7.01	8.25	9.49	30.51	46.64	74.95	111.22	140.70	150.89	165.98	182.58	200.84	220.92	243.01	502.65
R&D as % of US Sales										74%	32%	23%	18%	14%	13%	13%	20%
R&D costs paid to Amgen	*1.07*																
R&D license acquired from MSKCC			4.50														
General and administrative	*12.71*	3.54	3.60	3.66	3.72	14.52	16.33	18.13	19.94	25.39	41.16	52.25	59.07	77.46	92.14	101.14	149.76
Total operating expenses	**28.16**	9.31	15.11	11.91	13.20	45.03	62.96	93.08	131.16	166.83	192.37	218.46	241.83	278.44	313.19	344.28	652.61
Operating income (loss)	**(28.16)**	(9.31)	(15.11)	(11.91)	(13.20)	(49.53)	(65.46)	(94.08)	(119.60)	(12.38)	253.20	628.74	1,165.18	1,899.21	2,622.94	3,375.03	4,595.37
Other income (expense):																	
Interest income (expense), net	*0.13*	0.15	0.16	0.16	0.16	0.64	0.64	0.64	0.64	0.64	0.64	0.64	0.64	0.64	0.64	0.64	0.64
Total other income	*0.13*	0.15	0.16	0.16	0.16	0.64	0.64	0.64	0.64	0.64	0.64	0.64	0.64	0.64	0.64	0.64	0.64
Pre-tax income (loss)	**(28.03)**	(9.16)	(14.95)	(11.74)	(13.04)	(48.88)	(64.82)	(93.44)	(118.96)	(11.73)	253.85	629.38	1,165.82	1,899.85	2,623.58	3,375.67	4,596.01
Tax expense (benefit)	**(0.03)**	0.00									0.00	(94.41)	(291.45)	(569.96)	(918.25)	(1,181.49)	(1,608.60)
Tax rate											*0%*	*15%*	*25%*	*30%*	*35%*	*35%*	*35%*
Net Income	**(28.01)**	(9.16)	(14.95)	(11.74)	(13.04)	(48.88)	(64.82)	(93.44)	(118.96)	(11.73)	253.85	534.97	874.36	1,329.90	1,705.33	2,194.19	2,987.41
Other comprehensive gain (loss), net of tax:																	
Unrealized loss on investments	*(0.10)*	0.08	(0.05)														
Other comprehensive gain (loss)																	
Net loss applicable to common stockholders	**(28.11)**	**(9.16)**	**(14.99)**	**(11.74)**	**(13.04)**	**(48.88)**	**(64.82)**	**(93.44)**	**(118.96)**	**(11.73)**	**253.85**	**534.97**	**874.36**	**1,329.90**	**1,705.33**	**2,194.19**	**2,987.41**
Net loss per share basic	$ (1.43)	$ (0.42)	$ (0.62)	$ (0.40)	$ (0.44)	$ (1.64)	$ (2.13)	$ (3.01)	$ (3.74)	$ (0.36)	$ 7.64	$ 15.73	$ 25.12	$ 37.32	$ 46.73	$ 58.70	$ 65.14
Net loss per share diluted	$ (1.43)	$ (0.42)	$ (0.62)	$ (0.40)	$ (0.44)	$ (1.64)	$ (2.13)	$ (3.01)	$ (3.74)	$ (0.36)	$ 7.35	$ 15.10	$ 24.06	$ 35.67	$ 44.55	$ 55.82	$ 60.44
Basic share outstanding	*19.69*	21.92	24.22	29.55	29.75	29.78	30.42	31.09	31.78	32.50	33.24	34.01	34.81	35.63	36.49	37.38	45.86
Diluted Shares outstanding	*0.00*	25.23	25.57	30.32	30.57	30.67	31.39	32.13	32.91	33.71	34.55	35.43	36.34	37.29	38.28	39.31	49.43

Source: JMP Securities LLC, Company filings

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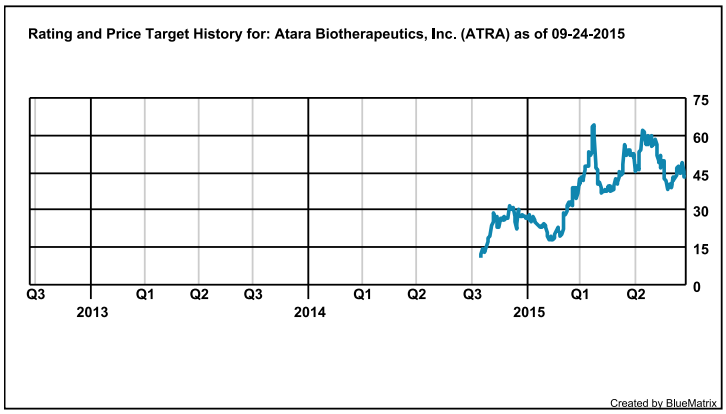
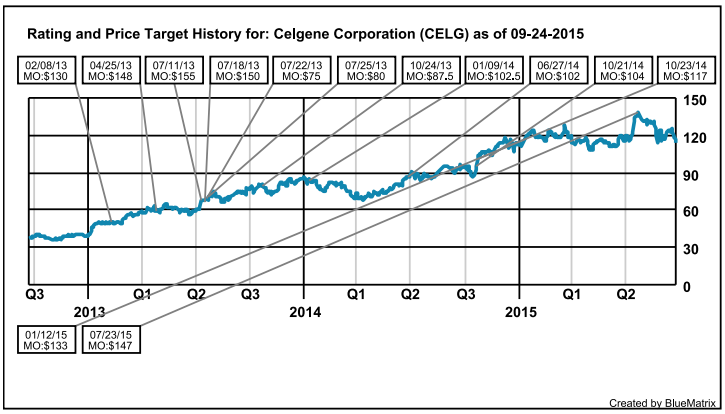
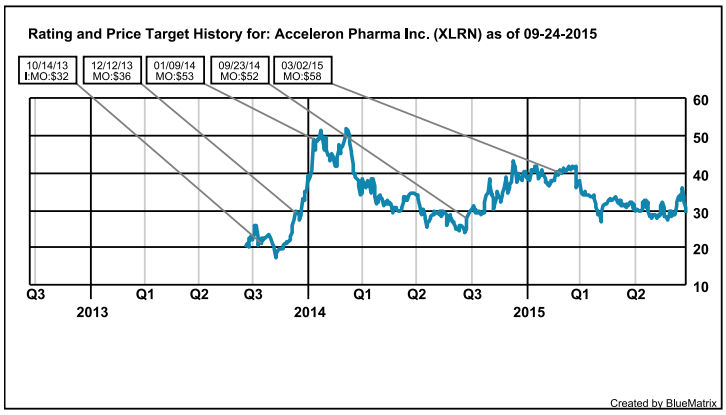
MARKET OUTPERFORM	Buy	302	*63.85%*	Buy	302	*63.85%*	88	*29.14%*
MARKET PERFORM	Hold	146	*30.87%*	Hold	146	*30.87%*	14	*9.59%*
MARKET UNDERPERFORM	Sell	6	*1.27%*	Sell	6	*1.27%*	0	*0%*
COVERAGE IN TRANSITION		19	*4.02%*		19	*4.02%*	2	*10.53%*
TOTAL:		473	*100%*		473	*100%*	104	*21.99%*

Stock Price Chart of Rating and Target Price Changes:

Note: First annotation denotes initiation of coverage or 3 years, whichever is shorter. If no target price is listed, then the target price is N/A. In accordance with NASD Rule 2711, the chart(s) below reflect(s) price range and any changes to the rating or price target as of the end of the most recent calendar quarter. The action reflected in this note is not annotated in the stock price chart. Source: JMP Securities.







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