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Basic Report (15-124)

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Atara Biotherapeutics, Inc.

Initiating Coverage With an Outperform Rating

We are initiating coverage of Atara Biotherapeutics with an Outperform rating and an Aggressive Growth company profile based on the company's two promising platforms and lead candidates targeting indications that currently lack effective treatments. Atara's management has leveraged its extensive scientific and business development experience to adroitly acquire a pipeline of promising compounds. Our bullish investment outlook on Atara is based on our view that the combination of the company's wholly owned lead product candidates, technology platforms, and potential for additional strategic collaborations offers investors' significant upside to current share prices.

We believe that Atara's lead molecularly targeted product candidate, PINTA 745, could address a significant market opportunity in patients with end-stage renal disease, based on its mechanism of action, proof-of-concept data, and lack of current treatment options in the setting. Based on the reported literature, we estimate that over 50% of patients with end-stage renal disease on dialysis experience a phenomenon known as protein-energy wasting, causing a decrease in functional ability, quality of life, and ultimately an increase in mortality. With an estimated 800,000 patients worldwide, clinical success in this indication alone would create significant stock appreciation over current levels. We highlight the expected results from a Phase II trial with PINTA 745 by the end of this year as a near-term catalyst and describe the potential market opportunities with additional molecularly targeted product candidates in further detail in this report.

Atara's second platform was recently acquired in a licensing deal with Memorial Sloan Kettering Cancer Center. This platform focuses on the use of third-party "off-the-shelf" T-cells that are specifically activated against opportunistic viral infections. Proof-of-concept data for these product candidates have been reported in multiple Phase I and Phase II trials, and the lead candidate, targeting the Epstein-Barr virus (EBV), has received breakthrough designation from the Federal Drug Administration in the treatment of EBV-associated malignancies in patients who have received hematopoietic stem cell transplantations. Although this lead indication is relatively small, the platform has the potential to address a broad range of additional malignancies across many indications.

We believe Atara's shares represent an attractive value with significant upside. While acknowledging the developmental risks associated with the biotechnology industry, we view Atara's two platforms as undervalued by the Street given the multiple product candidates with proof-of-concept data and broad optionality in potential indications. Trading at $36.32 with an estimated $350 million in cash, Atara is well capitalized to achieve multiple milestones in the advancement of its R&D effort into 2018.

Atara Biotherapeutics, based in South San Francisco, California, is a clinical-stage biotech company focused on leveraging its two proprietary technology platforms to develop therapies for indications that currently lack effective treatment options.

Stock Rating:	**Outperform**
Company Profile:	**Aggressive Growth**
Symbol:	ATRA (NASDAQ)
Price:	$36.32 (52-Wk.: $9.66–$65.56)
Market Value (mil.):	$1,036
Fiscal Year End:	December
Dividend/Yield:	None

Estimates	**2014A**	**2015E**	**2016E**
EPS FY	-$5.62	-$1.98	-$3.04

Valuation			
P/E	NM	NM	NM

Trading Data	
Shares Outstanding (mil.)	28.5
Float (mil.)	13.7
Average Daily Volume	341,774

Financial Data	
Cash (mil.)	$350
Enterprise Value (mil.)	$686

Portfolio Manager Summary

We believe that Atara offers investors the opportunity to own a stake in a well-managed company with two differentiated clinical platforms that are targeting areas of high unmet medical need. Lead clinical candidates from both platforms have generated encouraging clinical data to date. PINTA 745 showed positive proof-of-concept data in a Phase I study and has the potential to address a large market with no current therapies in patients suffering from end-stage renal disease (ESRD) with protein-energy wasting (PEW). We see increasing visibility into the clinical profile of PINTA 745 over the coming months, with Phase II clinical data in the dialysis PEW setting expected to be released in the fourth quarter of this year. In addition, we believe the cytotoxic T-lymphocyte (CTL) platform has consistently demonstrated strong efficacy in immunosuppressed patients with viral malignancies and offers certain advantages over other adoptive T-cell therapies, such as the ability to quickly administer the product in an "off-the-shelf" setting. Breakthrough therapy designation with EBV-CTLs in the EBV- lymphoproliferative disorders (LPD) following hematopoietic stem cell transplant (HCT) indication offers increased communication with the FDA regarding trial designs, and we believe there is potential for a priority review if clinical data is supportive at the time of FDA regulatory filing. With an estimated $350 million in cash, Atara is well capitalized and positioned to aggressively invest in the advancement of its research-and-development (R&D) effort into 2018, in our view.

Company History and Overview—Two Distinct Platforms With Broad Therapeutic Potential
Atara Biotherapeutics was launched in 2012 with a mission to develop therapeutics in areas of unmet medical need and little innovation. The company is built on two distinct platforms and technologies. The first is a set of seven compounds, licensed from Amgen, which target proteins in the transforming growth factor beta, or TGF-β, superfamily of proteins; clinical development is in progress in the areas of muscle wasting, including protein-energy wasting in patients on dialysis and cancer cachexia. Management dubs this platform and the associated compounds the *molecularly targeted programs*. The second group of assets was licensed more recently from Memorial Sloan Kettering Cancer Center (MSK) and is focused in the area of immuno-oncology. These therapies aim to target viral or cancer-specific antigens using off-the-shelf (allogeneic) T-cells from healthy donors. The most advanced of these candidates is in Phase II clinical testing in EBV-LPD, and the platform and its other product candidates are referred to as the *allogeneic T-cell therapies*. We believe that management has done an exceptional job of executing against its clinical, strategic, and financial goals during its relatively short life as a public company, and we anticipate an active news calendar over the coming 18-24 months.

The profile of Atara is highlighted by five clinical candidates in Phase I or Phase II trials. Projecting 12-18 months forward, we see potential for Atara to have significantly more critical mass clinically, which we believe will translate into shareholder value creation. Exhibit 1 provides an illustration of the company's current and projected pipeline.

Exhibit 1
Atara Biotherapeutics, Inc.
Product Portfolio

Drug	Description	Preclinical	Phase I	Phase II	Phase III	Indication
Molecularly Targeted Product Candidates						
PINTA 745	Peptibody that blocks myostatin signaling	NCT01958970				Protein-energy wasting in dialysis patients
STM 434	Soluble receptor designed to block Activin A	NCT02262455				Ovarian cancer or other solid tumors
ATA 842	Highly selective anti-myostatin antibody					IND enabling studies in cancer cachexia
T-Cell Product Candidates						
EBV-CTL	Allogenic T-cells activated against Epstein-Barr virus	NCT01498484				EBV-lymphoproliferative disorders due to immunosuppression from HCT, SOT, or other
	Allogenic T-cells activated against Epstein-Barr virus	NCT00002663				EBV-lymphoproliferative disorders due to immunosuppression from HCT, SOT, or other
CMV-CTL	Allogenic T-cells activated against cytomegalovirus	NCT02136797				Third-party donor-derived CMV-CTL for CMV viremia following HCT
	Allogenic T-cells activated against cytomegalovirus	NCT01646645				Primary transplant donor-derived CMV-CTL for CMV viremia following HCT
WT1-CTL	Allogenic T-cells activated against Wilms tumor protein	NCT00620633				Residual or relapsed leukemia following HCT
	Allogenic T-cells activated against Wilms tumor protein	NCT01758328				Relapsed/refractory multiple myeloma following HCT

EBV = Epstein-Barr virus
CMV = Cytomegalovirus
HCT = Hematopoietic stem cell transplant
SOT = Solid organ transplant
CTL = Cytotoxic T-lymphocyte
Source: Atara Biotherapeutics reports

Molecularly Targeted Products and Potential Therapeutic Benefits of Targeting TGF-β

Atara licensed seven compounds from Amgen in 2012 that formed the basis of the company. These compounds are biological molecules designed to modulate the signaling of proteins in the TGF-β superfamily. In general, the TGF-β superfamily consists of a highly conserved group of signaling proteins that play fundamental roles in biological processes at the cellular level, such as growth, differentiation, development, and tissue homeostasis. We believe these compounds have the potential to treat a broad range of clinical indications, including oncology, diseases of muscle regulation, and anemia. Although these pathways have been studied for multiple decades across numerous indications, limited clinical data have been reported to date with therapies designed to manipulate these signaling pathways in various disease indications.

Allogenic T-Cell Therapies—Immuno-oncology: Harnessing the Power of the Immune System to Fight Cancer

In many patients, certain immunosuppressive factors prevent the immune system from functioning properly. To bypass this immunosuppression, investigators have attempted to create and deliver activated T-cells to patients. In this technique, T-cells are isolated from the peripheral blood or the tumor environment (tumor infiltrating lymphocytes, or TIL) of the patient and grown in the laboratory. These cells can then be exposed to specific antigens or be genetically modified to express engineered receptors for specific antigens through chimeric antigen receptors (CAR) or T-cell receptors (TCR), and subsequently activated and stimulated to proliferate using cytokines such as interleukin-2 (IL-2). The activated T-cells are then transferred back into the patient to target and eliminate the cancerous cells displaying the specific antigen. Although still in the early phases of clinical testing, these therapies have shown impressive response rates in certain hematological cancers, such as CD19+ acute lymphoblastic leukemia (ALL). Continued research will be necessary to mitigate adverse events and adapt the technology to additional types of cancer, particularly in solid tumors. In addition, the personalized nature of the therapy creates a very labor-intensive manufacturing process and may present another set of challenges in achieving commercial-scale production and distribution.

As mentioned previously, Atara's entry to the immunotherapy space is through a collaboration with Memorial Sloan Kettering Cancer Center (MSK). As opposed to autologous CAR or TCR T-cells, which are collected from each individual patient, the allogenic T-cell technology from MSK is designed to be an off-the-shelf cellular therapeutic option for patients. The manufacturing process expands T-cells from healthy donors, selectively isolating T-cells that have been activated to a specific target and reducing the number of allospecific T-cells, thereby reducing the risk of the infused T-cells attacking off-target proteins. To date, MSK has developed libraries of activated cell lines that target Epstein-Barr virus (EBV), cytomegalovirus (CMV), and Wilms tumor protein (WT1). The potential clinical applications of effective therapies targeting antigens associated with these targets are quite broad, ranging from EBV-associated lymphoma to CMV opportunistic infections following hematopoietic stem cell transplant.

We provide an in-depth discussion of Atara's product candidates later in this report, while our goal here is to provide a brief overview. The company's lead allogenic T-cell program is designed to treat patients who have undergone a hematopoietic stem cell transplant, have subsequently developed an EBV-associated lymphoproliferative disease, and are refractory or relapsed following rituximab therapy. We see this as an area of unmet medical need and highlight that the FDA granted EBV-CTL breakthrough designation in this setting in February of this year.

We believe this technology has a highly attractive profile that may offer logistical and supply chain advantages over alternative approaches to cellular immunotherapy. Exhibit 2 provides a profile of Atara's allogenic T-cell therapies and draws a high-level comparison to engineered autologous T-cell therapies being tested in a number of clinical trials, primarily in hematologic malignancies. In comparison to the current generation of autologous T-cells, we highlight that two of the biggest advantages of Atara's platform include a reduced time from diagnosis to treatment and a seemingly more tolerable safety profile.

Exhibit 2
Atara Biotherapeutics, Inc.
Comparison Between Allogenic and Autologous T-Cell Therapies

	Allogenic T-Cell Platform Profile	Autologous T-Cell Platform Profile (CAR-T/TCR)
Manufacturing	Off-the-shelf technology manufactured ahead of time: therapy available within days of need	Cells isolated from blood of each patient must be genetically engineered, expanded, and reinfused into the patient
Immunogenicity	Proprietary human leukocyte antigen (HLA) matching algorithm suggests best donor therapy for each patient	Autologous cells naturally match the patient's HLA type
Current Results	Successful proof of concept in two viral-related malignancies (EBV-LPD, CMV) may support use in additional indications	Successful proof of concept by multiple companies and cancer centers supports use in certain highly advanced hemetological malignancies
Additional Indications	Potential for broad application beyond oncology in patients failing multiple antiviral therapies	Additional work needed to translate earlier success with hemetological malignancies into solid tumors
Safety	Limited adverse events and off-target effects reported to date	Cytokine release syndrome has proved to be a severe adverse event occasionally leading to patient deaths, particularly in older patients

Source: Company reports

Over the coming 12-18 months, we expect a number of key inflection points that could represent value-creating opportunities for investors. In particular, we highlight the Phase II top-line results from Atara's lead compound, PINTA 745, in ESRD patients with PEW, expected by the end of this year. A table of key anticipated events is shown in exhibit 3.

Exhibit 3
Atara Biotherapeutics, Inc.
Timeline

Date	Product	Event
2015	PINTA 745	Phase II trial results in ESRD patients with PEW (Q4)
	EBV-CTL	Phase III trial initiation in EBV-LPD following HCT
2016	STM 434	Phase I top-line results in solid tumors (1H)
	PINTA 745	Additional Phase II trial results in ESRD patients with PEW
	CMV-CTL	Clinical developments in broadened indications
	WT1	Phase I top-line results in HCT patients with AML or multiple myeloma

Source: Atara Biotherapeutics reports

Valuation and Financial Analysis

Our Outperform rating on Atara Biotherapeutics is based on our belief that the stock represents an attractive value at current price levels. Given the difficulty in assigning precise values to developmental-stage biotechnology companies, we have employed multiple strategies to elucidate the company's value, including sum of the parts, comparable analysis, and attribution of value to Atara's two platforms and pipeline. Atara's shares are trading at about $1 billion in market cap, and in light of its current cash position, the company's enterprise value is roughly $686 million. We see significant potential for upside in Atara shares.

Sum-of-the-Parts Evaluation

We performed a net present value analysis using a conservative set of criteria in which we attribute the majority of the company's value to PINTA 745 in the setting of dialysis patients suffering from protein-energy wasting (PEW). We believe that PINTA 745 alone, especially with consideration of the company's $350 million cash position, justifies Atara's current valuation. Therefore, we believe continued success with the additional molecularly targeted pipeline and/or CTL platform could provide significant value not currently reflected in the stock price to shareholders. In the following section, we outline our assumptions to perform the sum-of-the-parts analysis.

PINTA 745 Assumptions

- Given the lack of a current market in the PEW setting, it can be difficult to accurately estimate the potential market opportunity for PINTA 745. Based on published literature, we believe 50-60% of ESRD patients are at risk of developing PEW, putting the addressable patients at approximately 800,000 globally. In determining a possible price point for PINTA 745, we relied heavily on the commercial experience of Amgen's Epogen in the dialysis setting. Consistent with pricing trends associated with marketing pharmaceuticals in other global markets, we assume PINTA 745 will be priced at a 15%-20% discount to the U.S. price in Europe and other major markets. In addition, we assume promotional discounts ranging from 10% to 15%.

- We conservatively estimated the timeline of development and regulatory decisions on PINTA 745, with market penetration capped at 30% in the ESRD with PEW setting. Acceleration to the timeline, broader label expansion, and increased penetration rates provide potential upside to our estimates.

Comparable Analysis

Recognizing that the biotechnology sector is extremely heterogeneous and dynamic, we compiled a list of companies that we believe have comparable attributes to Atara, as shown in exhibit 4. Given the recent investor focus on biotechnology companies developing immuno-oncology therapies, we believe that a higher premium is being placed on exciting early-stage assets. As is often the case with comparable analysis, particularly in biotechnology, there are no perfect comparisons for Atara. However, we believe that when compared with many of the companies in our analysis, Atara shares seem to be a good value, particularly on the basis of enterprise value. We highlight that Atara is uniquely positioned because of its two differentiated technology platforms that could have the potential to address large markets.

Exhibit 4
Atara Biotherapeutics, Inc.
Analysis of Comparable Companies
(dollars in millions, except share price)

Company	Share Price	Market Cap	Rating
Acceleron	$24.50	$810	
Aduro Biotech	$21.23	$1,322	Outperform
Bellicum Pharma	$15.45	$409	
Cellectis	$27.25	$955	
Chimerix	$38.41	$1,770	Outperform
Kite Pharma	$64.57	$2,824	
Juno	$48.74	$4,901	
Novavax	$7.26	$1,958	
Atara Biotherapeutics	**$36.32**	**$1,036**	**Outperform**

Source: FactSet

Attribution of Value to Technology Platform and Earlier Pipeline Candidates
Atara has two platforms with several early-stage product candidates. In the original deal with Amgen, Atara received licenses to seven molecularly targeted products. These products target proteins in the TGF-b superfamily. In addition to the current clinical compounds PINTA 745 and STM 434, Atara is conducting preclinical studies on five additional compounds: ATA 842; ATA 777; ATA M43; STM 217; and ActR2B5. Atara's second platform, the adoptive T-cell products licensed from MSK, has three products in clinical trials. However, the technology platform and agreement with MSK have the potential to create additional T-cell products, including targeting other viral malignancies or engineered T-cells. Although we do not include these early-stage or future compounds in our valuations, we believe visibility into these products has the potential to provide further upside to current stock prices.

Risk Factors

We believe that Atara is affected by many of the same risks as other developmental-stage biotechnology companies. We believe that most developmental biotechnology companies, including Atara, share four broad categories of investment risk: clinical; regulatory; capital; and competitive.

Technical and Clinical Risk
Atara's unique approach to treating PEW and challenging cancers does not come without inherent risks. Specifically, while PINTA 745 has demonstrated encouraging results in patients with prostate cancer, the longer-term safety and efficacy profile of the drug in the dialysis population is unproven. Further, the use of modified T-cells requires significant technical expertise and logistical challenges. Miscalculations in any step of the clinical processes or trial design could ultimately cause a significant setback to the platform as a whole. In addition, although the expertise of the management team and the successful clinical data to date are encouraging, we acknowledge risks associated with the use of new therapeutic modalities such as adoptive T-cells in viral-associated malignancies and myostatin inhibition to address PEW in the dialysis setting.

Regulatory Risk
We believe that all development candidates in the biotechnology industry carry some level of regulatory risks. However, regulatory risks are especially apparent in the use of novel therapeutic strategies with little clinical precedence. Although inhibition of myostatin in the prostate cancer setting seems to be associated with clinical benefit, little is known regarding the safety of longer-term dosing. Moreover, cellular therapy is by no means a new concept, but Atara's unique approach to treating cancer and infectious disease with modified T-cells is novel. The success of multiple early-stage trials, both in safety and efficacy, helps reduce future regulatory risk, in our opinion; however, successful Phase II and Phase III trials are paramount to obtaining regulatory approval and ultimately commercialization.

Capital Risk
Biotechnology companies typically have a continued need for capital until reaching FDA drug approval, commercialization, and positive cash flow. Capital can come from a variety of sources, including strategic collaborations and equity financing. While we believe that Atara is well positioned to execute additional strategic deals, we acknowledge the potential for management to monetize one or more of its numerous clinical assets as a means of raising capital.

Competitive Risk
One of the attractive features of Atara is that the company is focused on areas of unmet medical need lacking innovation. Still, our analysis of the competitive landscape reveals other clinical candidates that pose either a direct or indirect competitive threat. We provide greater detail regarding the competitive landscape later in this report.

Company Management

Atara management consists of a number of individuals with significant experience in the biotechnology field. We believe that this team, led by Isaac Ciechanover, has the expertise and industry experience necessary to leverage the company's proprietary platform into successful clinical programs. The experience of the top executives is summarized in exhibit 5.

Exhibit 5
Atara Biotherapeutics, Inc.
Management Team

Management	Position	Previous Experience
Isaac Ciechanover, M.D.	President & Chief Executive Officer	Founder, Atara Biotherapeutics Partner, Kleiner Perkins Caufield & Byers Director for Business Development, Celgene
Christopher Haqq, M.D., Ph.D.	Chief Medical Officer	Vice President for Clinical Research and Development, Cougar Biotechnology
John F. McGrath, Jr.	Chief Financial Officer	Executive in Residence and Operating Partner, Kleiner Perkins Caufield & Byers
Gad Soffer	Chief Operating Officer	Global Project Leader for Abraxane, Celgene
Mitchall G. Clark	Chief Regulatory and Quality Assurance Officer	Senior Vice President of Global Regulatory Affairs, Abraxis Bioscience

Source: Atara Biotherapeutics reports

Key Program Analysis

PINTA 745

Mechanism of action. PINTA 745 is a peptibody that specifically inhibits the signaling of myostatin, an important protein in muscle regulation. The binding of PINTA 745 to myostatin prevents the docking of myostatin onto cell surface receptors of muscle cells, ultimately preventing downstream signaling effects within the cell. A peptibody consists of a peptide component with high specificity for the target protein fused to the Fc region of an antibody, thereby increasing the biological half-life of the compound. The average half-life of peptibodies in humans ranges from three to eight days, allowing for more convenient dosing schedules than peptide therapies, which are rapidly cleared from the body.

Muscle protein regulation. The regulation of muscle protein synthesis is a highly conserved process with multiple signaling pathways interacting with one another. In healthy individuals, the positive signaling of protein synthesis is largely balanced by countersignaling for protein breakdown. Insulin-like growth factor-1 (IGF-1) is an anabolic hormone responsible for stimulating the growth and differentiation of muscle cells through the IGF receptor on muscle cells. Binding of IGF-1 to its receptor activates intracellular-signaling molecules, particularly Akt kinase, which upregulates protein synthesis through mammalian target of rapamycin (mTOR) signaling and downregulates protein degradation by inhibition of FoxO signaling. Multiple studies have shown that resistance exercises can increase the levels of serum IGF-1 in healthy individuals, thus playing a significant role in muscle hypertrophy in response to resistance training.

The effects of IGF-1 are regulated through multiple mechanisms. For example, insulin-like growth factor binding proteins (IGFBPs) are capable of sequestering IGF-1 in the serum, thereby preventing IGF receptor binding. Myostatin, also known as growth differentiation factor 8, is almost exclusively expressed in skeletal muscle and acts as a negative regulator of muscle growth. Myostatin signaling occurs through the activin type IIB receptor (ActRIIB), activating Smad 2/3 which blocks Akt signaling and protein synthesis. In addition, myostatin signaling activates FoxO, upregulating protein degradation. Natural inhibitors of myostatin, such as follistatin, bind to myostatin and prevent the docking with ActRIIB, thereby blocking myostatin signaling. Disruption of myostatin signaling, either through a functional mutation or therapeutic modulation, has repeatedly led to an increase in lean muscle mass. One of the most notable cases of a natural myostatin mutation is in the Belgian Blue breed of beef cattle, in which the disruption of myostatin signaling leads to an almost 40% increase in lean muscle tissue compared with other breeds of cattle.

Despite the multiple mechanisms regulating muscle turnover, there are numerous disease states where this signaling balance is disrupted, and muscle wasting (also referred to as muscle atrophy, protein-energy wasting, cachexia, or sarcopenia, depending on contributing factors) occurs despite the consumption of adequate nutrition. The exact mechanisms that cause these disruptions in the natural balance of muscle turnover are unknown; however, muscle wasting is often correlated with chronic states of inflammation, such as in patients suffering from cancer, congestive heart failure, end-stage renal disease, or AIDS. In addition, the wasting of muscle tissue has been linked to poor patient prognosis and has a strong correlation with increased risk of mortality in patients across multiple indications. These conditions have created a significant interest in the medical community to gain a better understanding of the causes behind muscle wasting and develop effective treatments for these patients.

A variety of studies, both preclinical and clinical, have been conducted in an attempt to mitigate or reverse muscle wasting across multiple disease states. These include but are not limited to: resistance exercise; protein or amino acid dietary supplementation; anti-inflammatory therapies such as tumor necrosis factor-alpha antibodies; muscle hypertrophy agents such as anabolic steroids and

growth hormones; and muscle atrophy inhibitors such as myostatin antibodies. Overall, the results of these studies have been mixed. Given the heterogeneity in the underlying condition leading to muscle wasting, we believe there may not be a universal therapy for all forms of muscle atrophy. We also highlight the lack of large, well-controlled clinical trials showing that inhibiting or reversing muscle wasting directly improves patient prognosis in each underlying disease.

Target disease indication and potential market opportunity. Protein-energy wasting (PEW) is a state of muscle wasting, inflammation, and malnutrition that often occurs in patients with end-stage renal disease (ESRD) receiving dialysis treatment. Multiple studies have shown that PEW increases the risk of infections, cardiovascular disease, and other complications in dialysis patients, ultimately increasing the risk of mortality. A variety of mechanisms have been proposed as contributing to PEW, including chronic inflammation from dialysis, inadequate nutritional intake, loss of adequate kidney function, metabolic acidosis, and increases in muscle myostatin levels. These mechanisms are capable of disrupting the balance between protein synthesis and protein breakdown, which is highly regulated in healthy individuals through multiple signaling pathways.

Multiple large studies have examined the correlations between biomarkers of muscle wasting and mortality risk in ESRD patients on dialysis. The loss of muscle mass and decrease in serum protein albumin (a marker of protein turnover dysfunction) have both been consistently highlighted by these studies. In one such study, 120,000 patients receiving dialysis were analyzed for correlations between body mass index (BMI) and serum creatinine (a surrogate of skeletal muscle mass) and patient mortality. The results showed that patients with lower BMI and less muscle mass (as determined by lower serum creatinine) had an increased risk of death over a five-year time frame. Similarly, studies looking at protein serum albumin levels have shown decreased levels being highly correlated with poor patient prognosis. In a study by Lowrie and Lew *(Am J Kidney Disease,* 1990), logistical regression analysis was applied to more than 12,000 hemodialysis patients, and it was found that serum albumin levels below 4.0 g/dL (two-thirds of patients) had the highest correlation with death probability. Lastly, in a recent retrospective study conducted by Atara in collaboration with DaVita Clinical Research, a multivariate analysis of 56,000 patients receiving dialysis through DaVita centers was conducted to characterize the PEW condition and identify patients most at risk. The results of this study showed that patients with a serum albumin level less than or equal to 3.8 g/dL (54% of all patients) were at much greater risk of dying at one year compared with patients with higher serum albumin levels (11% versus 3%).

It is estimated that over 450,000 patients in the United States have ESRD and are receiving maintenance dialysis. Assuming about 50%-60% of these patients are at risk of PEW, we estimate the impact of this co-morbidity affecting roughly 250,000 patients in the United States and 800,000 patients globally. This is, therefore, a very large market opportunity with minimal current effective therapies.

Phase I proof-of-concept study in prostate cancer. A Phase I proof-of-concept trial with PINTA 745 (previously AMG 745) was conducted by Amgen in patients with nonmetastatic prostate cancer receiving androgen deprivation therapy (ADT), a patient population at high risk of cachexia. In this study, 38 patients were randomized 1:1 to receive either a weekly subcutaneous dose of 3 mg/kg or placebo. Patients remained on treatment for four weeks, at which point the changes in lean body mass and lower extremity muscle size were determined over baseline. Patients treated with PINTA 745 showed a significant increase in lean body mass at both the end of therapy and at a one-month follow-up over placebo control (exhibit 6). Treatment with PINTA 745 also led to an increase in lower extremity muscle size; however, the difference was significantly greater than placebo only at one month after treatment had ended. Both of these results show a time-dependent increase in skeletal muscle in patients who typically experience losses in muscle mass over time, even after treatment with PINTA 745 had been ceased. Interestingly, patients on PINTA 745 did not perform any better than placebo when measuring physical function, including maximum weight lifted for one repetition using a knee extension machine and a short physical performance battery (SPPB)

to assess standing balance, timed walk test, and five repetitions of chair stand. We note that the short duration of therapy and the high functional ability of patients at baseline likely confounded the ability to detect differences in physical function among the two patient groups.

Although the therapy was generally well tolerated, there was a slight increase in patients experiencing fatigue and diarrhea while taking PINTA 745. One patient discontinued treatment after the second dose of PINTA 745 due to moderate erythema of the abdomen, and the severity decreased following treatment discontinuation. Given the small size of this trial, greater patient numbers will be needed to fully elucidate the possible correlations between PINTA 745 and these adverse events. Overall, the Phase I results offer a proof of concept for the ability of PINTA 745 to safely and efficiently increase lean body mass, even in patients with an underlying imbalance in muscle protein turnover. However, it is important to note that the mechanisms of muscle wasting may be different in patients with prostate cancer receiving ADT, compared with patients with ESRD and PEW, when trying to predict successful outcomes in the continuing Phase II study.

Exhibit 6
Atara Biotherapeutics, Inc.
Phase I Trial Results With PINTA 745 in Prostate Cancer

	Phase I	
Disease	Nonmetastatic Prostate Cancer	
Enrollment	38 patients	
Patient Characteristics	73.5±6.4 years old Receiving ADT for 6 months Serum testosterone below 50 ng/dL	
Treatment Duration	Four weeks	
Treatment Arms	3.0 mg/kg PINTA 745 subcutaneous weekly	Placebo control
Change in Lean Body Mass at Four Weeks	1.5%*	-0.7%
Change in Lean Body Mass at Two Months	1.9%*	0.2%
Change in Lower Extremity Muscle Size at Four Weeks	1.2%	-0.7%
Change in Lower Extremity Muscle Size at Two Months	2.7%*	-0.1%
Adverse Events	Diarrhea 21% Fatigue 16% Contusion 16%	Diarrhea 9% Fatigue 4%

* denotes p <0.05 compared with placebo

Source: Atara Biotherapeutics reports

Phase II study design. Shortly after receiving the rights to PINTA 745, Atara initiated a Phase II study in ESRD patients on hemodialysis who have PEW. The differences between the Phase I trial in prostate cancer and the continuing Phase II trial are outlined in exhibit 7

Exhibit 7
Atara Biotherapeutics, Inc.
Comparison of Phase I and Phase II Protocols for PINTA 745

Design Element	Phase I (Prostate Cancer)	Current Phase II (PEW)	Rationale
Route of Administration	Subcutaneous injection	Intravenous injection	Enhances drug exposure and aligns with routine patient management in the dialysis setting
Duration of Therapy	One month	Three months	Longer-term dosing may enhance muscle growth
Dose of PINTA 745	0.3mg/kg 1.0 mg/kg 3.0 mg/kg weekly dosing	3 mg/kg weekly 3 mg/kg loading dose followed by 1 mg/kg maintenance dose 6 mg/kg loading dose followed by 2 mg/kg maintenance dose	Higher drug exposure may be more effective while similarly well tolerated
Duration of Follow-Up	One month	Two months	Extends information on durability of effect

Source: Atara Biotherapeutics reports

As specified in the Phase II protocol, following the dosing of the first eight patients in the 3.0 mg/kg dosage arm for one month, a review of all safety data was completed. At that time, there were no reports of serious adverse events, and the most commonly reported grade 1 or 2 event was muscle pain. In addition, it was observed that the half-life of PINTA 745 in patients with ESRD was significantly longer than in the patients in the Phase I trial. Based on this additional data, Atara amended the protocol to add two new dosing regimens that include a loading dose followed by a maintenance dose. Exhibit 8 provides an outline of the Phase II trial design. In August 2015, Atara reported the completion of patient enrollment in the Phase II trial and reiterated the expected release of top-line results by the end of the year.

Exhibit 8
Atara Biotherapeutics, Inc.
Phase II Trial Design With PINTA 745 in PEW

	Phase II			
Disease	End-stage renal disease with protein-energy wasting			
Enrollment	48 patients			
Patient Enrollment Criteria	18 to 85 years old Maintenance hemodialysis for ≥6 months Receiving dialysis treatment at least 3 times per week Serum albumin ≤3.8 g/dL			
Treatment Duration	12 weeks			
Treatment Arms	3.0 mg/kg PINTA 745 IV weekly	3.0 mg/kg PINTA 745 IV for 3 weeks followed by 1.0 mg/kg PINTA 745 IV for 9 weeks	6.0 mg/kg PINTA 745 IV for 3 weeks followed by 2.0 mg/kg PINTA 745 IV for 9 weeks	Placebo
Percent Change in Lean Body Mass	Primary endpoint			
Change in Physical Function	Secondary endpoint			
Change in Inflammatory Biomarkers	Secondary endpoint			
Change in ESA Usage	Secondary endpoint			
Quality of Life Assessments	Secondary endpoint			

* denotes p <0.05 compared with placebo
ESA = Erythropoiesis Stimulating Agents
Source: Atara Biotherapeutics reports

Competitive myostatin inhibitor landscape. In addition to PINTA 745, multiple companies are developing experimental therapies targeting myostatin for a variety of indications. Exhibit 9, on the following page, provides an overview of selected continuing clinical trials. Notably, MYO-029, an anti-myostatin antibody developed by Wyeth and Pfizer, was evaluated in a small Phase I/II trial in adult patients with muscular dystrophies, including Becker muscular dystrophy, facioscapulo-humeral dystrophy, and limb-girdle muscular dystrophy. Although MYO-029 supported a trend in increased muscle mass, this did not translate into increases in muscle strength or function, and further study of the drug in this indication has been halted. We believe it is important to keep in mind the underlying causes of muscle atrophy in each indication. Although MYO-029 was able to increase muscle mass, the underlying muscular defect in these patients, such as dystrophin in Becker muscular dystrophy, may have limited a noticeable functional increase.

Exhibit 9
Myostatin Inhibitor Landscape

Drug	Company	Mechanism	Indication	Phase
PINTA 745	Atara Biotherapeutics	Anti-Myostatin Peptibody	Protein-Energy Wasting Secondary to Dialysis	Phase II
ATA 842	Atara Biotherapeutics	Anti-Myostatin Antibody	Cancer Cachexia	Preclinical
ACE-031	Acceleron Pharma/ Shire	Soluble ActRIIB	Duchenne Muscular Dystrophy	Phase II (Discontinued)
ACE-083	Acceleron Pharma	Activin and Myostatin Ligand Trap	Not Disclosed	Phase I (Healthy volunteers)
BMS-986089	Bristol-Myers Squibb	Anti-Myostatin Adnectin	Duchenne Muscular Dystrophy	Phase I/II
LY2495655	Eli Lilly	Anti-Myostatin Antibody	Cancer Cachexia Older Patients at Risk of Falling	Phase I Phase II
PF-06252616	Pfizer	Anti-Myostatin Antibody	Duchenne Muscular Dystrophy	Phase II
REGN1033	Regeneron/ Sanofi	Anti-Myostatin Antibody	Sarcopenia	Phase II
MYO-029	Wyeth/ Pfizer	Anti-Myostatin Antibody	Muscular Dystrophy	Phase I/II (Discontinued)
Bimagrumab	Novartis	Anti-Activin Receptor IIB Antibody	Sporadic Inclusion Body Myositis (sIBM) COPD Cachexia	Phase II/III Phase II
SRK-015	Scholar Rock	Latent Myostatin Inhibitor	Not Disclosed	Preclinical

COPD = Chronic obstructive pulmonary disease
Sources: Company reports and clinicaltrials.gov

Although we do not believe any myostatin inhibitors are being tested in the setting of PEW secondary to ESRD, successful outcomes with PINTA 745 may entice other companies to explore the indication. In addition, there is potential for PINTA 745 to be used in indications currently being tested by competitors' myostatin inhibitors or other muscle-wasting indications mentioned previously.

Intellectual property. Atara holds exclusive rights to five issued patents directed to PINTA 745 in the United States, with expiration dates ranging from 2025 to 2035. In addition, Atara has issued patents or pending patent applications in many countries worldwide, with expiration dates ranging from 2023 to 2035. These dates are exclusive of possible patent term extensions.

Amgen deal structure. Upon closure of the agreement between Atara and Amgen for the exclusive rights to PINTA 745, Atara paid Amgen an up-front license fee of $250,000 and issued Amgen 205,128 shares of Atara stock (converted to shares of common stock upon closing of Atara's IPO). Atara is obligated to make additional payments to Amgen upon certain developmental and commercial milestones, up to $129 million, and mid- to high-single-digit royalties on all sales of PINTA 745. In addition, Atara does not hold the rights to the commercialization of PINTA 745 in Japan because of a prior agreement between Takeda and Amgen.

Overview. The myostatin pathway has been fairly well characterized since its discovery about two decades ago. Despite this understanding and the important role of muscle biology in multiple indications, clinical success with therapies modulating the myostatin pathway has been limited. However, the Phase I results with PINTA 745 in prostate cancer offer positive proof of concept and strongly support the biological activity of the therapy. We, therefore, are encouraged by the potential for myostatin to show clinical benefit in the continuing Phase II trial. We view the continuing exploratory Phase II study as providing essential information to shape the design and endpoints for a registration-enabling trial. Given the lack of available treatment options for patients suffering from ESRD with PEW, we believe improvements in physical function, reduction of inflammatory biomarkers, enhancements in quality of life such as reduction of erythropoiesis-stimulating agents,

and any trends of survival benefit could all be used to design a primary endpoint in a subsequent pivotal trial. Based on the results of the continuing Phase II trial, we believe Atara may run an additional Phase II study with increased powering to determine the clinical and statistical significance of PINTA 745 on a potential primary endpoint to be used subsequently in Phase III.

Exhibit 10
Atara Biotherapeutics, Inc.
PINTA 745 Profile

	PINTA Clinical and Commercial Profile
PINTA 745: Potential First-in-Class Molecule for Protein Energy Wasting	• Blocks myostatin, preventing it from inhibiting muscle production • Improved lean body mass, physical function, inflammation in CKD* model • Statistically significantly increased lean body mass and lower extremity muscle size in randomized, blinded Phase I study in prostate cancer; acceptable safety profile
Attractive Market Opportunity	• Protein-energy wasting (PEW) is a state of muscle wasting, inflammation, and malnutrition in ESRD; no approved therapies • Decreased physical function, increased morbidity and mortality • ~250,000 patients in the U.S. and 800,000 worldwide
Upcoming milestones	• Randomized, blinded, placebo controlled Phase II trial (48 PEW patients) • Phase II data expected Q4 2015

* CKD = chronic kidney disease
Sources: Company reports, USRDS, and DaVita Clinical Research

STM 434

Mechanism of action. The therapeutic approach with STM 434 is to inhibit the signaling molecule activin. As a member of the TGF-β superfamily of growth factors, activin transfers cellular signals such as embryonic cell fate induction, wound healing, and proliferation through binding to activin receptor type II and IIB (ActRII/ActRIIB). During tumor development, activin A is implicated in the growth and proliferation of cancer cells, especially in ovarian and other solid tumors. In head and neck, colon, gastric, esophageal, pancreatic, and non–small-cell lung cancer, activin A has been shown to be overexpressed; we believe the totality of data suggests therapies that are designed to inhibit activin A's activity could be promising.

STM 434 is a soluble ActRIIB receptor-IgG fusion protein that binds to human activin. The approach of interacting with a signaling molecule (or a ligand, such as activin A) to inhibit its binding to its respective receptors is called a ligand trap. Taking advantage of trapping a ligand is more elegant, in our view, because targeting the activin receptors, which have multiple ligands, could perturb other biological processes not associated with cancer progression.

Target disease indication and potential market opportunity. While ovarian cancer accounts for only 3% of cancers among woman, it causes more deaths than any other cancer of the female reproductive system. Overall, it is the fifth-leading source of cancer death in women in the United States. According to the National Cancer Institute, there were an estimated 22,000 new cases and 14,000 ovarian cancer deaths in 2013, and roughly 192,000 women were living with ovarian cancer in the United States in 2012. In Europe, there are an estimated 44,000 new cases of ovarian cancer every year, and roughly 30,000 women will die from the disease. In exhibit 11, on the following page, we illustrate the epidemiology of ovarian cancer.

Exhibit 11
Ovarian Cancer Global Epidemiology

	Incidence	Prevalence	Deaths
United States	22,000	192,000	14,000
Europe	44,000	103,700*	30,000
Worldwide	230,000	593,000*	150,000

*Estimated five-year prevalence.

Sources: Atara and Roche reports, National Cancer Institute, World Health Organization, and International Agency for Research on Cancer

Progress in ovarian cancer treatment has been painfully slow in the past several decades, and even with new therapies, the clinical outcomes have not improved significantly. As shown in exhibit 12, with a five-year survival rate of about 44%, ovarian cancer's prognosis is better than only pancreatic (6%), liver (16%), esophageal (17%), lung and bronchial (17%), and stomach cancer (28%).

Exhibit 12
Five-Year Survival Rates by Cancer Stage at Diagnosis (2003-2009)

	All Stages	Local	Regional	Distant
Breast	89%	99%	84%	24%
Colon and Rectum	65%	90%	70%	13%
Esophagus	17%	39%	21%	4%
Kidney	72%	92%	64%	12%
Larynx	61%	76%	43%	35%
Liver	16%	29%	10%	3%
Lung and Bronchus	17%	54%	26%	4%
Melanoma (Skin)	91%	98%	62%	16%
Oral Cavity and Pharynx	62%	83%	59%	36%
Ovary	44%	92%	72%	27%
Pancreas	6%	24%	9%	2%
Prostate	99%	100%	100%	28%
Stomach	28%	63%	28%	4%
Testis	95%	99%	96%	74%
Thyroid	98%	100%	97%	55%
Urinary Bladder	78%	70%	33%	5%
Uterine Cervix	68%	91%	57%	16%
Uterine Corpus	82%	95%	68%	17%

Source: American Cancer Society, Surveillance Research 2014

While most ovarian cancer cases are detected at later stages, surgery can potentially be curative for early-stage disease that is contained within the ovaries. For late-stage ovarian cancer, palliative debulking surgery is often employed to remove most tumors larger than 1 centimeter. Following surgery, patients usually receive front-line chemotherapy, which consist of two or more drugs, including a platinum-based chemotherapy such as cisplatin or carboplatin and a taxane such as paclitaxel or docetaxel for three to six cycles (28-day cycle). Response rates are typically high for treatment-naïve patients (exhibit 13 summarizes the response rates based on ovarian cancer subtypes). However, most cancers eventually recur due to drug resistance. If the recurrence takes place longer than six months from the initiation of treatment (platinum sensitive), patients are usually retreated with another course of platinum-based chemotherapy. On the other hand, if the recurrence happens within the six-month time frame (platinum resistance), targeted therapies such as Roche's Avastin, which inhibits tumor angiogenesis, in combination with paclitaxel, is administered. Patients who relapse (third line) proceed to receive monotherapy chemotherapy such as docetaxel, paclitaxel, gemcitabine, or topotecan or alternatively, if BRCA mutation-positive, are treated with AstraZeneca's Lynparza, which blocks enzymes responsible for repairing damaged DNA.

Exhibit 13
Ovarian Cancer's Response to Treatment Based on Subtype

Ovarian Cancer Type	Prevalence	Typical Response Rate (Chemotherapy)	Typical Overall Survival
Serous Adenocarcinoma	63%	72%-73%	41 months
Clear Cell Tumors	11%	11%	21 months
Granulosa Cell	2%-5%	50%	147 months

Sources: Atara Biotherapeutics reports, *International Journal of Gynecological Cancer, and Journal of Gynecologic Oncology*

Study design. STM 434 is in a Phase I study that is enrolling roughly 75 patients with ovarian cancer and other solid tumors. The primary objective for the study is to determine the maximum tolerated dose for STM 434 monotherapy and STM 434 in combination with liposomal doxorubicin from a range of 0.25 mg/kg to 4.0 mg/kg. In addition, recommended Phase II dosing, radiographic response rate, muscle function, and body composition will be evaluated as secondary endpoints. Atara will also be exploring biomarkers that could identify patients who will be most likely to respond to treatment; these biomarkers include activin A and follicle-stimulating hormone levels and ARID1A and FOXL2 mutation status in patients. We anticipate top-line results from the study during the first half of 2016. In exhibit 14, we provide a summary of the Phase I trial design.

Exhibit 14
Atara Biotherapeutics, Inc.
Phase I Trial Design With STM 434 in Ovarian and Other Advanced Solid Tumors

	Phase I	
Disease	Ovarian cancer Fallopian tube cancer Endometrial cancer Solid tumors	
Enrollment	75 patients	
Patient Characteristics	Refractory or intolerant to all standard available treatment No increased risk of bleeding	
Treatment Arms	STM 434 every 4 weeks	STM 434 + Liposomal doxorubicin every 4 weeks
Doses	0.25 mg/kg STM 434 0.5 mg/kg STM 434 1.0 mg/kg STM 434 2.0 mg/kg STM 434 4.0 mg/kg STM 434	0.25 mg/kg STM 434 0.5 mg/kg STM 434 1.0 mg/kg STM 434 2.0 mg/kg STM 434 4.0 mg/kg STM 434 + 40 mg/m^2 liposomal doxorubicin
Maximum Tolerated Dose	Primary endpoint	
Recommended Phase II Dose	Secondary endpoint	
Radiographic Response Rate	Secondary endpoint	
Muscle Function and Body Composition	Secondary endpoint	

Source: Atara Biotherapeutics reports

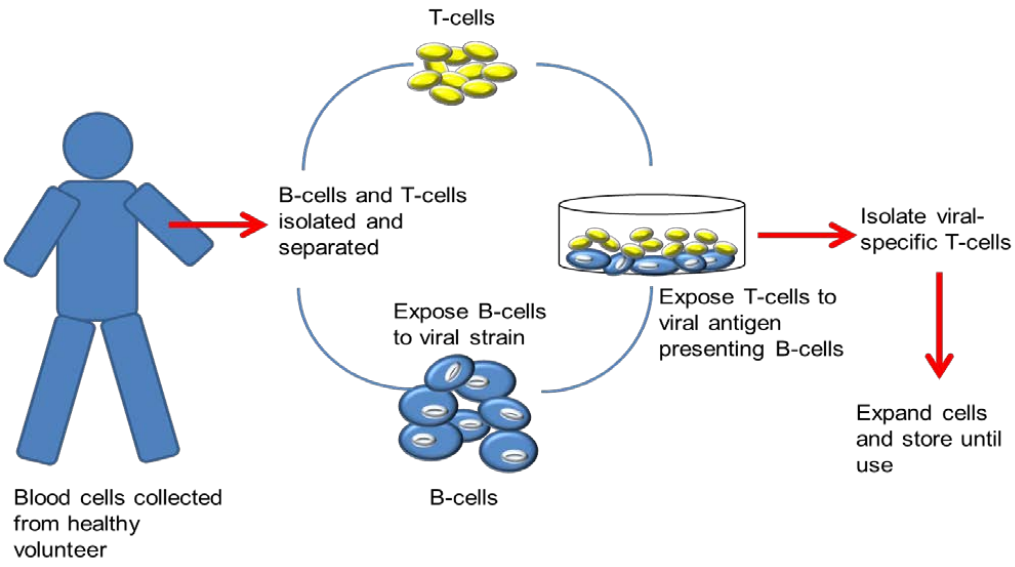
Intellectual property. Atara holds exclusive rights to four issued patents directed to STM 434 in the United States. In addition, Atara has issued patents or pending patent applications in many countries worldwide, with anticipated expiration dates ranging from 2023 to 2035. These dates are exclusive of possible patent term extensions.

Amgen deal structure. Upon closure of the agreement between Atara and Amgen for the exclusive rights to the six additional molecularly targeted products, Atara issued Amgen 410,256 shares of Atara stock (converted to shares of common stock upon closing of Atara's IPO). Atara is obligated to make additional payments to Amgen upon certain developmental and commercial milestones, up to $163 million, and mid- to high-single-digit royalties on all future sales of these compounds.

Cytotoxic T-Lymphocyte Platform

Mechanism of action. Third-party white blood cells are collected from healthy individuals via leukapheresis, and the T-cells and B-cells are then separated. Next, the B-cells are exposed to a specific antigen, such as a viral strain, activating the cells to the exposed antigen and creating B lymphoblastoid cells. The B-cells are then irradiated to prevent them from proliferating and co-cultured with the T-cells isolated from the same donor. In this co-culture, the B-cells present the specific antigen to the T-cells, activating and sensitizing the T-cells to the antigen of choice. The activated T-cells are then expanded and undergo a selection process to remove nonspecific T-cells. Once complete, the T-cells are assessed for their reactivity to the specific antigen and their HLA restriction prior to being cryopreserved and stored for future therapeutic use. In total, the process takes about eight weeks to generate a new T-cell line. However, the cells are then ready to be thawed and infused once a patient is ready for therapy. The CTLs will then specifically target and destroy cells presenting the antigen to which they were originally activated. See exhibit 15 for an illustration of the procedure.

Exhibit 15
Atara Biotherapeutics, Inc.
Allogenic T-Cell Manufacturing Process



Source: Atara Biotherapeutics reports

HLA restriction is an essential aspect of allogenic T-cell platforms. For a T-cell to recognize and destroy cells presenting a specific antigen, the HLA complex, also known as the major histocompatibility complex (MHC), must be compatible between the patient and the donor. MHC class I (HLA-A, B, or C) receptors are most often associated with viral infections. These receptors specifically present antigens on the cell surface that originated inside the cell. For example, peptides derived from viral proteins that are broken down inside the cell will be presented on the cell surface by MHC class I receptors. Therefore, cytotoxic T-cells seeking to destroy cells with specific viral peptides will need a matching MHC class I receptor to recognize the infected cell. Work conducted by MSK has resulted in the development of an algorithm for successful matching of a third-party CTL line for use in individual patients based on HLA restrictions. Based on the extensive library of donor T-cells that MSK has generated to date, it estimates being able to find an appropriate HLA match for 99% of patients. In addition, the library of donor T-cells provides the opportunity to treat a patient with multiple cell lines in the situation where a successful outcome is not achieved upon first treatment. MSK and Atara have reported specific instances of patients having variable response rates to treatments based on the matching of different HLA types.

Epstein-Barr Virus–Cytotoxic T-Lymphocytes (EBV-CTL)

Target disease indication and U.S. market opportunity. Epstein-Barr virus (EBV) is a member of the herpes family (also called human herpesvirus 4, or HHV-4) and is one of the most common viruses in humans. EBV is most commonly known for causing infectious mononucleosis, a relatively benign condition naturally resolving itself in healthy individuals. It is estimated that 90%-95% of all individuals have been infected with EBV by the time they are in their 40s. Once EBV is contracted, it will remain latent in B-cells for the rest of an individual's life. Although typically benign, EBV has been associated with various malignancies including Hodgkin lymphoma, Burkitt lymphoma, gastric cancer, and even diseases of the central nervous system such as multiple sclerosis. EBV is especially problematic in immunocompromised individuals, such as those who have undergone a hematopoietic stem cell transplant (HCT) or solid organ transplant (SOT). The immunosuppression necessary to prevent graft rejection creates an increase in the risk of lymphomas or lymphoproliferative diseases associated with EBV (EBV-LPD). Exhibit 16 shows the annual incidence of EBV-associated malignancies in the United States and the European Union.

Exhibit 16
Approximate Incidence of EBV-Malignancies

Indication	U.S. and E.U. Addressable Market (Patients)
LPD after HCT	1,300
LPD after SOT	1,700
EBV+DLBCL	2,900
EBV+Nasopharyngeal Carcinoma	6,000
EBV+Gastric Cancer	16,500

EBV = Epstein Barr Virus
HCT = Hematopoietic Stem Cell Transplant
SOT = Solid Organ Transplant
DLBC = Diffuse Large B-Cell Lymphoma
Source: Atara Biotherapeutics reports

EBV-LPD following HCT. Hematopoietic stem cell transplantation is a therapeutic option for a multitude of malignancies, most often blood cancers such as myeloma or leukemia. In patients with advanced diseases, HCT is often the only chance for a curative therapy. However, significant risks are associated with HCT, particularly in cases of allogenic HCT. The most common complication from HCT is infection resulting from the ablation of the patient's own bone marrow before HCT, significantly reducing the patient's immune system's ability to fight infections. In patients receiving allogenic HCT, there is a risk of graft-versus-host disease (GvHD), an incompatibility between the donor HCs and the patient's tissues that causes the donor's immune cells to consider the patient's tissues foreign. To limit the occurrence of GvHD, patients receiving allogenic HCT will typically receive immunosuppressants such as prednisone. The chronic state of immunosuppression places the patient at a higher risk for opportunistic infections. Although ultimately a rare complication, EBV-related malignancies, in particular EBV-LPD, are life-threatening conditions with limited treatment options. Patients will typically receive CD20-targeted therapies, such as rituximab, but a durable response is observed in only 20% of patients.

In 2012, there were slightly more than 22,000 allogenic HCTs in the United States and the European Union. While this is slightly less than the number of autologous transplants, the number of allogenic transplants is expected to rise in the coming years given the increased ability to match appropriate donors and general improvements in standard of care. It is estimated that 7% of patients receiving

an allogenic HCT will develop an EBV-specific LPD, of which 20% will receive durable benefit from rituximab therapy. Therefore, about 1,300 cases of EBV-LPD failing rituximab and without viable treatments are expected to occur in the United States and the European Union annually. The median overall survival (OS) for these patients has been reported to be in the range of 16-56 days.

EBV-LPD following SOT. Rejection of a transplanted organ is a major concern in all SOTs, and therefore, immunosuppressive medications are often given to patients receiving a donated organ. As in cases of allogenic HCT described previously, chronic immunosuppression exposes the patient to opportunistic infections or the reactivation of latent infections such as EBV. Treatment options are limited for patients who develop EBV-LPD due to already-weakened immune systems and other complications associated with SOT. Patients will typically receive rituximab therapy with or without chemotherapy as the first line of therapy, but the response rate is only about 50%. Patients who do not respond to rituximab historically show a 33% overall survival at two years and an even worse prognosis if they are considered high risk (60 years or older and elevated liver enzymes).

The incidence of EBV-LPD following SOT varies by organ transplant type and patient characteristics. As shown in exhibit 17, there is a significant variability in the incidence of EBV-LPD. This is largely dependent on patient age at transplant, co-morbidities, and level of immunosuppression.

Exhibit 17
Approximate Incidence of EBV-LPD Following SOT

Organ	Annual Transplants	Incidence of EBV-LPD
Kidney	36,000	1%-10%
Liver	13,000	2%-15%
Heart	4,000	2%-19.5%
Lung	3,200	2.5%-9.4%
Intestines	200	20%-30%

Sources: Global Observatory on Donation & Transplantation and Atara Biotherapeutics reports

Previous clinical experience with EBV-CTL. MSK has conducted two separate clinical trials (Study 95-024 and Study 11-130) with EBV-CTLs to date in a variety of patients with EBV-associated malignancies, including EBV-LPD after HCT and SOT. In these studies, a combined 34 patients with EBV-LPD after HCT were treated with allogenic EBV-CTLs. All 34 patients had received prior rituximab therapy. Patients received three weekly doses of $1x10^6$-$2x10^6$ cells/kg via intravenous infusion per cycle. If patients did not experience severe toxicities during the first cycle, they were eligible to receive additional cycles of therapy. A summary of the results presented at the recent American Society for Clinical Oncology (ASCO) Annual Meeting can be found in exhibit 18.

Exhibit 18
Atara Biotherapeutics, Inc.
Phase II Trial Results With EBV-CTLs in EBV-LPD After HCT

	ASCO 2015 Update
Disease	EBV-Associated Lymphoproliferative Disorder After HCT
Enrollment	34 patients
Patient Enrollment Criteria	Median age 25.9 years old (5.1-74.1) All received prior rituximab therapy
Treatment Duration	Three-week cycle (eligible to receive additional cycles if no toxicity observed)
Treatment	Weekly infusion of 1×10^6 - 2×10^6 cells/kg
Complete Response Rate	56% (19/34)
Partial Response Rate	9% (3/34)
Disease Control Rate	70% (24/34)
Safety Profile	No hematopoietic toxicity No cytokine release syndrome

EBV = Epstein-Barr virus
CTL = Cytotoxic T-lymphocytes
LPD = Lymphoproliferative disorder
HCT = Hematopoetic stem cell transplant
Sources: Atara Biotherapeutics and Memorial Sloan Kettering Cancer Center reports

Although median overall survival (OS) for all patients has not been reached, the two-year OS is 46.9% in Study 95-024 and 63.8% in Study 11-130. These results have the potential to be significantly better than the historically reported median overall survival of 16-56 days for patients failing rituximab. Notably, patients experienced minimal adverse events, and no cases of cytokine release syndrome were observed. Cytokine release syndrome has been a notable limitation in the use of engineered T-cell therapies, even leading to multiple cases of patient death.

Based on the promising results of Study 95-024 and Study 11-130, the FDA granted breakthrough designation for EBV-CTL therapy in patients who have received a HCT and failed rituximab therapy. Atara is preparing the registrational study protocol and plans to submit the protocol to the FDA as part of a special protocol assessment (SPA) by the end of this year.

Competitive landscape in EBV malignancies. The lack of a currently approved therapy for EBV-LPD has led to physician off-label use of other therapies, most notably rituximab, as mentioned previously. However, there are a number of continuing clinical trials evaluating different treatments for EBV-specific malignancies. Notably, Cell Medica is developing EBV-specific CTLs from patients suffering from EBV malignancies. These autologous EBV-CTLs are the result of a collaboration between Cell Medica and the Center for Cell and Gene Therapy at Baylor College of Medicine and are in a Phase II trial for the treatment of EBV+extranodal NK/T-cell lymphoma. In addition, Baylor College of

Medicine has numerous continuing clinical trials for various allogenic EBV-CTLs and multivirus-specific CTLs. ViraCyte has licensed viral-specific CTL therapies from Baylor, and Adcyte LLC has licensed multivirus-specific CTLs.

Exhibit 19
EBV Malignancy Treatment Landscape

Drug	Company	Mechanism	Indication	Phase	Trial Identifier
EBV-CTL	Atara	Allogenic EBV-CTLs	EBV-LPD and associated malignancies	Phase II	NCT01498484
CMD-003	Cell Medica	Autologous EBV-CTLs	EBV+ extranodal NK/T-cell lymphoma	Phase II	NCT01948180
LMP-CTL	Baylor College of Medicine	Allogenic EBV-CTLs	EBV-associated diseases	Phase I	NCT01447056
MABEL-CTL	Baylor College of Medicine	Allogenic EBV-CTLs	EBV-associated diseases	Phase I	NCT02287311
Multivirus-CTL	Baylor College of Medicine	Donor-derived multivirus-specific CTLs	EBV, CMV, adenovirus, HHV6, and BK virus associated diseases following stem cell transplant	Phase I/II	NCT01570283
GRALE-CTL	Baylor College of Medicine	Allogenic EBV-CTLs	EBV-associated lymphoma	Phase I	NCT01555892

Sources: Company reports and clinicaltrials.gov

Cytomegalovirus–Cytotoxic T-Lymphocyte (CMV-CTL)

Target disease indication and market opportunity. Cytomegalovirus (CMV) is a member of the herpes virus family and is also known as human herpesvirus 5 (HHV5). Similar to EBV and other herpes viruses, CMV has the ability to remain latent in the body well after the original symptoms of the viral infection have subsided. Given this latent characteristic, it is estimated that CMV infection affects 50%-90% of the global population. Although this latent infection is typically benign, immunocompromised patients, such as those who have undergone HCT or SOT, are at a high risk of developing significant CMV-associated malignancies, including retinitis, hepatitis, and encephalitis. Although many of these patients can be successfully treated with antiviral therapies, the emergence of resistance to antiviral drugs presents a significant challenge to the medical community. In addition to immunocompromised patients, it is estimated that 95% of glioblastoma multiforme (GBM) tumors express CMV-specific proteins. GBM is a very aggressive brain cancer with very poor patient prognosis. Exhibit 20 shows the annual incidence of CMV-associated malignancies in the United States and the European Union.

Exhibit 20
Estimated Incidence of CMV-Associated Malignancies

Indication	U.S. and E.U. Addressable Market (Patients)
Antiviral Resistant CMV After HCT	2,000
Antiviral Resistant CMV After SOT	1,300
Glioblastoma Multiforme	21,000
Congenital CMV	12,500

Source: Atara Biotherapeutics reports

Previous clinical experience with CMV-CTL. MSK previously reported interim results from three clinical studies with CMV-CTL in patients with CMV-associated diseases that developed resistance to antiviral drug treatment. At the interim point, 38 patients had been treated with CMV-CTLs after failing a median of four prior antiviral therapies, and 34 were evaluable for responses. In the 25 patients with CMV viremia, nine had a complete response and seven had a partial response (64% overall response rate). A similar response rate was observed in the nine patients with CMV disease, with five patients having a complete response and one with a partial response (67% overall response rate). These data demonstrate the benefit of CMV-CTLs in patients with limited treatment options, and the therapy was well tolerated with notably no cases of GvHD.

Exhibit 21
Atara Biotherapeutics, Inc.
Phase II Trial Results With CMV-CTLs in CMV-Disease Following HCT

	Memorial Sloan Kettering 2015 Update
Disease	CMV-associated diseases following HCT
Enrollment	34 patients
Patient Enrollment Criteria	Received allogeneic HCT Failed antiviral drug therapy
Treatment Duration	Three-week cycle (eligible to receive additional cycles if no toxicity observed)
Treatment	Weekly infusion of 1×10^6 cells/kg
Complete Response Rate	41% (14/34)
Partial Response Rate	24% (8/34)
Overall Response Rate	65% (22/34)
Safety Profile	No graft versus host disease

CMV = cytomegalovirus
CTL = Cytotoxic T-lymphocytes
HCT = Hematopoetic stem cell transplant
Sources: Atara Biotherapeutics and Memorial Sloan Kettering Cancer Center reports

Based on the promising results of CMV-CTLs in patients with CMV viremia and disease after HCT observed to date, Atara plans to test the use of CMV-CTLs in additional CMV malignancies. We look forward to increased visibility into clinical development plans with CMV-CTLs.

Competitive landscape in CMV viremia and disease. A number of antiviral therapies are currently approved or in clinical trials for the treatment of CMV diseases. Approved therapies include ganciclovir and valganciclovir, while Shire plc, Merck & Co., and Chimerix are all developing drug candidates for CMV infection. As seen in EBV malignancies, other companies are working on adoptive T-cell therapies for CMV. Cell Medica is developing CMV-specific CTLs for use with antivirals in patients suffering from CMV reactivation following allogeneic HCT. The donor-derived CMV-CTLs have completed a Phase III trial in the United Kingdom and are marketed under a "Specials" license. In addition, Baylor College of Medicine has numerous continuing clinical trials for various allogenic CMV-CTLs and multivirus-specific CTLs. ViraCyte has licensed viral-specific CTL therapies from Baylor, and Adcyte LLC has licensed multivirus-specific CTLs.

Wilms Tumor-1–Cytotoxic T-Lymphocyte (WT1-CTL)

Target disease indication and clinical development. Wilms tumor-1 is a transcription factor found in the nucleus of many cells and an important regulator of cell growth and differentiation, particularly during fetal development. After birth, the expression of WT1 is typically low outside the genitourinary tract. Mutations to the gene encoding the protein were first discovered as a key factor in the development of a childhood renal neoplasm, termed Wilms tumor, although the exact role of the protein in oncogenesis is under debate. Regardless of the exact role, the protein WT-1 is overexpressed in a broad range of tumor types, including acute myeloid leukemia, multiple myeloma, non–small-cell lung cancer, breast cancer, and ovarian cancer, among others.

MSK is conducting two Phase I trials with WT1-CTLs in patients who have received HCT and have relapsed/refractory multiple myeloma or residual/relapsed acute myeloid leukemia. Preliminary results from the continuing trial in patients with relapsed/refractory multiple myeloma or plasma cell leukemia were recently presented by MSK investigators at the 15th International Myeloma Workshop in Rome, Italy. In the seven subjects who had been treated with WT1-CTLs and had one year of follow-up, three patients achieved complete remission and 84% (6/7) had disease control. We believe the reported response rates, although in a small number of patients, are very encouraging and further support the use of the CTL platform in new indications. We look forward to additional results from these trials to be presented by MSK investigators at a future medical conference. Atara is also planning on evaluating the use of WT1-CTLs in combination with other therapies for the treatment of solid tumors.

Exhibit 22
Atara Biotherapeutics, Inc.
Phase I Trial Design and Top-Line Results With WT1-CTLs

	International Myeloma Workshop 2015
Disease	WT1+ Multiple Myeloma Following HCT
Enrollment	7 patients
Patient Enrollment Criteria	21-73 years old Relapsed or refractory multiple myeloma/plasma cell leukemia following HCT
Treatment Duration	Three-week cycle
Treatment	Weekly infusion of WT1-CTLs
Complete Remission Rate	43% (3/7)
Partial Response Rate	14% (1/7)
Disease Control Rate	86% (6/7)

WT1 = Wilms Tumor 1
CTL = Cytotoxic T-lymphocytes
HCT = Hematopoetic stem cell transplant
Sources: Atara Biotherapeutics and Memorial Sloan Kettering Cancer Center reports

Intellectual property. In collaboration with MSK, Atara has filed for patent protection covering the cytotoxic T-lymphocyte platform. The potential approval of these patent filings will directly cover specific methods-of-use claims and the methods of identifying and selecting third-party, donor-derived targeted T-cell lines for therapeutic use.

MSK deal structure. Atara entered into an exclusive option agreement with MSK in September of 2014, paying MSK $1.25 million in cash and issuing 59,761 shares of common stock. In June of this year, Atara exercised the option to obtain an exclusive license agreement for patents, know-how, cell lines, and other information regarding T-cell-specific products. In connection with this option exercise, Atara agreed to make an up-front payment of $4.5 million to MSK. Atara will also be obligated to make additional milestone payments up to $33 million based on the achievement of development, regulatory, and commercial milestones with the current T-cell products. MSK also has the potential to receive mid- to high-single-digit royalty payments based on sales of any licensed products.

Conclusion

We believe that Atara offers investors the opportunity to own a stake in a well-managed company with two differentiated clinical platforms that are targeting areas of high unmet medical need. Lead clinical candidates from both platforms have generated encouraging clinical data to date. PINTA 745 showed positive proof-of-concept data in a Phase I study and has the potential to address a large market with no current therapies in patients suffering from end-stage renal disease with protein-energy wasting (ESRD with PEW). We see increasing visibility into the clinical profile of PINTA 745 over the coming months with Phase II clinical data in the dialysis PEW setting expected in the fourth quarter. In addition, we believe the CTL platform has consistently demonstrated strong efficacy in immunosuppressed patients with viral malignancies and offers advantages over other adoptive T-cell therapies. Breakthrough therapy designation with EBV-CTLs in the EBV-LPD following HCT indication offers increased communication with the FDA regarding trial designs, and we believe there is the potential for a priority review if clinical data is supportive at time of regulatory filing. With an estimated $350 million in cash, Atara is well capitalized and positioned to aggressively invest in the advancement of its R&D effort into 2018. We are, therefore, launching coverage of Atara with an Outperform rating.

Exhibit 23
Atara Biotherapeutics, Inc.
Income Statement

(dollars in thousands except EPS and shares in thousands)

	2014A	Q1A	Q2A	Q3E	Q4E	2015E	2016E	2017E	2018E	2019E	2020E	2021E
Revenue												
PINTA 745	0	0	0	0	0	0	0	0	0	0	131,553	339,389
STM 434	0	0	0	0	0	0	0	0	0	0	0	0
EBV-CTL	0	0	0	0	0	0	0	0	191	2,711	6,845	14,770
Total revenues	**$0**	**$0**	**$0**	**$0**	**$0**	**$0**	**$0**	**$0**	**$191**	**$2,711**	**$138,398**	**$354,159**
Cost of revenue	0	0	0	0	0	0	0	0	48	678	34,600	88,540
Gross profit	0	0	0	0	0	0	0	0	143	2,033	103,799	265,619
R&D	**14,380**	5,767	7,007	8,759	10,073	31,605	66,337	85,791	95,833	106,577	119,954	135,009
R&D costs paid to Amgen	1,066	0	0	0	0	0	0	0	0	0	0	0
R&D costs paid to MSK	0	0	4,500	0	0	4,500	0	0	0	0	0	0
SG&A	**12,710**	3,544	3,601	3,961	4,357	15,463	23,255	29,297	33,304	38,505	48,566	54,662
Total operating expenses	28,156	9,311	15,108	12,720	14,430	51,569	89,592	115,088	129,137	145,082	168,520	189,671
Income (loss) from operations	**($28,156)**	**($9,311)**	**($15,108)**	**($12,720)**	**($14,430)**	**($51,569)**	**($89,592)**	**($115,088)**	**($128,994)**	**($143,049)**	**($64,721)**	**$75,948**
Interest income	125	153	163	0	0	24	0	0	0	0	0	0
Net income (loss) before income taxes	(28,031)	($9,158)	($14,945)	($12,720)	($14,430)	(51,253)	(89,592)	(115,088)	(128,994)	(143,049)	(64,721)	75,948
Income tax benefit (provisions)	(25)	2	0	0	0	2	0	0	0	0	0	(12,041)
Non-GAAP Net income (loss)	**($28,006)**	**($9,160)**	**($14,945)**	**($12,720)**	**($14,430)**	**($51,255)**	**($89,592)**	**($115,088)**	**($128,994)**	**($143,049)**	**($64,721)**	**$63,907**
Unrealized gains (losses) on investments	(100)	$0	$0	$0	$0	0	0	0	0	0	0	0
Comprehensive loss	(28,106)	(9,160)	(14,945)	(12,720)	(14,430)	(51,255)	(89,592)	(115,088)	(128,994)	(143,049)	(64,721)	63,907
Non-GAAP Net income (loss) attributable to stockholders	**(28,006)**	**(9,160)**	**(14,945)**	**(12,720)**	**(14,430)**	**(51,255)**	**(89,592)**	**(115,088)**	**(128,994)**	**(143,049)**	**(64,721)**	**63,907**
Non-GAAP net income (loss) per common share diluted	**($5.62)**	**($0.42)**	**($0.62)**	**($0.45)**	**($0.50)**	**($1.98)**	**($3.04)**	**($3.66)**	**($3.37)**	**($3.19)**	**($1.40)**	**$1.31**
Non-GAAP weighted-average common shares diluted	4,986	21,918	24,224	28,443	28,723	25,827	29,437	31,395	38,471	44,891	46,686	48,553

Sources: Atara Biotherapeutics, Inc. reports

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William Blair or an affiliate is a market maker in the security of Atara Biotherapeutics, Inc.

William Blair or an affiliate expects to receive or intends to seek compensation for investment banking services from Atara Biotherapeutics, Inc. within the next three months.

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DJIA:	16,912.29
S&P 500:	1,995.83
NASDAQ:	4,791.15

The prices of the common stock of other public companies mentioned in this report follow:

Amgen Inc. (Market Perform)	$148.05
AstraZeneca PLC	$31.92
Chimerix, Inc. (Outperform)	$38.37
Merck & Co. Inc.	$50.95
Pfizer Inc.	$33.29
Roche Holding AG	$32.55
Shire plc (Outperform)	$201.88
Takeda Pharmaceutical Co. Ltd.	¥5,486.00

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Outperform (Buy)	67%	Outperform (Buy)	16%
Market Perform (Hold)	31%	Market Perform (Hold)	3%
Underperform (Sell)	2%	Underperform (Sell)	0%

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