Argos Therapeutics, Inc. (ARGS)

Initiating Coverage at Market Outperform; Differentiated Immunotherapy Platform for Cancer and HIV

MARKET DATA

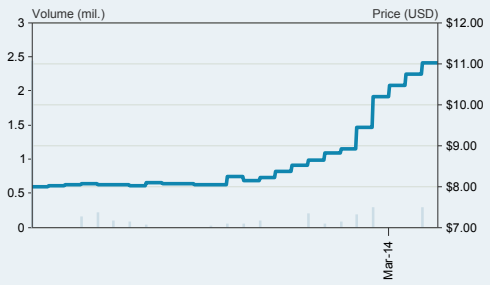
Price	$11.00
52-Week Range:	$7.97 - $10.96
Shares Out. (M):	19.0
Market Cap ($M):	$209.0
Average Daily Vol. (000):	289.0
Cash (M):	$85

Source: Thomson Reuters and JMP Securities LLC

FY DEC		2013E	2014E	2015E
Revenue ($M)	1Q	$0.8A	$1.2	--
	2Q	$1.0A	$1.0	--
	3Q	$1.5A	$0.8	--
	4Q	$1.7	$0.3	--
	FY	**$5.0**	**$3.3**	**$0.2**
EPS	1Q	($4.83)A	($0.50)	--
	2Q	($4.78)A	($0.51)	--
	3Q	($4.19)A	($0.57)	--
	4Q	($3.82)	($0.61)	--
	FY	**($17.36)**	**($2.19)**	**($1.50)**
	P/E	NM	NM	NM

Source: Company reports and JMP Securities LLC

STOCK PRICE PERFORMANCE



MARKET OUTPERFORM | Price: $11.00 | Target Price: $17.00

INVESTMENT HIGHLIGHTS

We are initiating coverage of Argos Therapeutics with a Market Outperform rating and $17 price target. Argos is a personalized immunotherapy company based on its proprietary Arcelis platform, targeting patient tumors and jump-starting immune responses even in the face of baseline immunosuppression. Argos' AGS-003 program is in Phase III development for metastatic renal cell carcinoma. Data from the pivotal study are expected in early 2016 and in our opinion, proceeds from Argos' completed IPO last month should see the company through to pivotal data. We believe data flow in the next 12-18 months should de-risk the Arcelis platform, increase investor confidence in Phase III success, and drive an increase in valuation for the company. Our $17 price target is based on a sum-of-the-parts NPV analysis consisting of revenue for AGS-003 in mRCC (~$14) and early stage RCC (~$3).

AGS-003 focused on patients with poor prognosis. Argos is focused on patients with intermediate and poor prognoses in metastatic renal cell carcinoma (mRCC). We believe this group can benefit from combination therapy on top of Sutent and other small molecule targeted therapies; however, side effect profiles have precluded this approach. The benign profile of AGS-003 allows positioning as first-line therapy in combination with Sutent; we believe AGS-003 may represent a paradigm shift in therapy with the potential to improve outcomes. We project peak sales of $634M in 2024.

Pivotal trial design optimized for success. In Phase II, AGS-003 in combination with Sutent led to a doubling of overall survival compared to a risk-matched historical control. Although the small numbers in Phase II and the use of a historical control comparator add risk to the pivotal trial design, there are several factors that give us confidence in a positive outcome in Phase III. These include: 1) overall survival in intermediate- and poor-risk RCC patients on Sutent is predictable, suggesting that the behavior of the control arm can be estimated with reasonable accuracy; 2) the trial is optimized to ensure that almost all patients receive the first five "priming doses" of AGS-003, which, in our view, is a key driver of positive outcomes; and 3) physicians can dose through progression and switch patients to drugs other than Sutent, reflecting current practice and allowing for continued dosing of AGS-003.

AGS-004 data provides near-term platform validation. We believe a near-term value driver for the company is data for Argos' second product, AGS-004, in 2Q14. AGS-004 is in development for HIV, designed to target patients' mutated HIV virus and restore immune response via a CD4+ T cell independent mechanism. In our view, these data should have read-through to the AGS-003 program by validating the mechanism of action of the Arcelis platform products. The AGS-004 program is being conducted with little to no cost for shareholders; therefore, we see this HIV program as upside.

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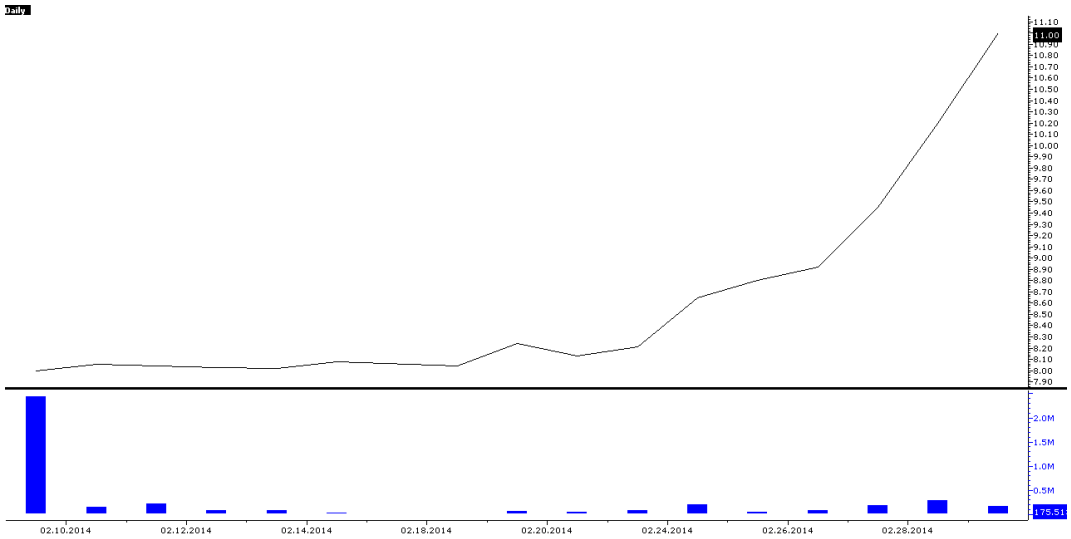
COMPANY DESCRIPTION

North Carolina-based Argos Therapeutics is a biopharmaceutical company focused on personalized immunotherapy for cancer and infectious disease. Its products are based in Argos's proprietary Arcelis platform with lead candidate AGS-003 in Phase III development for metastatic renal cell carcinoma in combination with Sutent as first line therapy. Argos is also developing AGS-004 for HIV eradication.

FIGURE 1. Price Chart



Source: Thompson Reuters

FIGURE 2. Upcoming Events

2Q14	Phase 2b Data in HIV	AGS-004
1H14	Initiate Core Biopsy study	AGS-003
2H14	Initiate early RCC study	AGS-003
2014	Initiate 2 HIV studies	AGS-004
YE14	Complete enrollment Pivotal study	AGS-003
2014, 2015	Interim blinded analyses	AGS-003
2016	Final data (OS)	AGS-003

Source: Company reports and JMP Securities LLC

INVESTMENT THESIS

Argos is differentiated from the immunotherapy field by targeting mutated tumor antigens and circumventing tumor immunosuppression.

Argos poised to win in immunotherapy

On the heels of the success of Bristol's Yervoy and impressive response rates from checkpoint (PD-1/PDL-1) inhibitors, investor focus is honed in on the potential for long-term clinical responses that result from circumventing tumor immunosuppression. We see Argos as a pure-play targeted immunotherapy company, differentiated from the field by its proprietary Arcelis platform. Argos products can target mutated tumor antigens from patients and jumpstart adaptive immune responses without relying on a patient's CD4+ T cells to help, lessening tumor immunosuppression. Argos's AGS-003 program is in Phase III development with data expected in early 2016 and we believe its mechanism of action should translate into a meaningful extension of overall survival in patients with metastatic renal cell carcinoma (mRCC). We anticipate supportive data in RCC in the next 12-18 months that, in our view, should increase investor confidence in the pivotal study and lift Argos' shares.

Phase II data robust despite small numbers

AGS-003 demonstrated impressive activity as a standalone therapy and in combination with first-line tyrosine kinase inhibitor (TKI) Sutent in two single-arm Phase II studies, supporting both safety and efficacy of AGS-003 in the mRCC intermediate and poor risk population. AGS-003 as standalone therapy demonstrated overall survival about equal to TKI therapy but without toxicities and in combination, overall survival doubled compared to TKI therapy alone. We acknowledge the risk associated with comparing single-arm clinical data to historical controls; however, recent meta-analyses of mRCC patients have demonstrated the predictability of overall survival in these patients on TKI therapy when stratified by risk. Further, we believe Phase II data support the mechanism of action of AGS-003 and therapy specific changes observed in immune response from baseline correlate to progression free survival and overall survival. In our view, these data are supportive of a true drug effect and give us confidence in a successful Phase III study.

Phase III designed for success

Argos needs one Phase III study for approval as was outlined in a special protocol assessment (SPA) with the FDA. The study is powered for a hazard ratio of 0.708, or a six-month increase in overall survival assuming a 16-month overall survival rate in the control arm, which we believe may be a conservative estimate based on the patient population being investigated. In our view, there are some key changes to the trial design from Phase II that give us confidence in a successful study.

- In the Phase II combination study, over half of the poor-risk patients did not survive long enough to receive five doses of AGS-003, which our analysis suggests is a driver of positive outcomes. For the Phase III study, these sickest patients will be excluded by capping risk factors at 4 (of 6), ensuring that more patients will receive five "priming" doses of AGS-003.
- AGS-003 can be dosed through disease progression, increasing the chance of patients receiving at least five doses of AGS-003 and possibly showing a clinical benefit. In Phase II, dosing was stopped at progression.
- Patients not tolerating Sutent can switch to another TKI or other second-line therapy – an option not permitted in Phase II. In our view, this will more appropriately reflect current practice, keep patients on study, and open up alternative treatment paradigms for AGS-003. We note this also increases the risk of the control arm outperforming; however, our analyses suggest most small molecule agents demonstrate similar mean overall survival as Sutent and we believe that the benefits of opening up the study to other agents outweigh the risks.

There are three event-driven interim analyses (25%, 50%, and 75% of events accrued) that we anticipate in 2015. Each analysis will assess if AGS-003 can provide at least a four-month improvement in overall survival; therefore, we see each analysis as incrementally de-risking with upside if the study is stopped early for efficacy. We anticipate final data in early 2016.

Upcoming supportive studies de-risk platform and support commercial viability

Although the pivotal study for AGS-003 will not read out until 2016, we anticipate news flow from supportive studies in RCC to increase confidence in the Arcelis platform and partially de-risk the AGS-003 pivotal study. We anticipate data before the pivotal trial reads out, generating more immunological data to support the mechanism of action of AGS-003. Additional open label studies will begin this year. These studies are focused on broadening the scope of AGS-003 – one will look at relapsing mRCC patients utilizing tumor samples from biopsy for AGS-003 production and others will be in early stage RCC patients. We also see upside in 2014 if the company begins a combination study with a PD-1/PDL-I inhibitor, as we believe AGS-003 and PD-1/PDL-1 inhibitors are likely synergistic and data on the combination may be important for competitive positioning in the future RCC market. We do not include PD-1/PDL-1 combinations in our valuation and consider them upside.

Platform validation in the near term

In 2Q14, we anticipate data from an NIH funded placebo-controlled Phase IIb study of AGS-004 in chronic HIV patients. We see these data as an important value driver for Argos as additional proof of mechanism for the Arcelis platform, as AGS-004 and AGS-003 are derived from the same process. However, AGS-004 uses HIV mRNA in lieu of tumor RNA. The data expected this summer are from a 53-patient trial looking at viral load reduction in HIV patients after discontinuing anti-retroviral therapy (ART) for 12 weeks. We believe that if patients with very few CD4+ T cells can launch an attack against the HIV virus due to AGS-004, it is supportive of the effect of AGS-003 where patients with tumor immunosuppression can launch an attack against a tumor. Minimally, we would expect successful ART interruption for 12 weeks to correlate with the number of new memory cells targeting the virus. If no increase in memory cells is observed, we would see it as a potential negative read-through to the AGS-003 program. The regulatory and commercial path forward for this indication is not clear; therefore, we consider this program as upside to our valuation.

VALUATION

We value Argos Therapeutics with a risk-adjusted, NPV sum-of-the-parts analysis including potential revenue streams for AGS-003 in metastatic and early stage renal cell carcinoma (RCC) in the U.S. and EU. AGS-003 is in Phase III development under a special protocol assessment with the FDA and we assign a 60% chance of the compound making it to market in this indication based on robust mechanistic data and Phase II results, accounting for ~$14 of our price target. Pivotal data is anticipated in 2016. We assume a U.S. launch in metastatic RCC (mRCC) in 2017 followed by EU launch in 2019 with a lag time of two years for launch into early stage RCC. We assign a 30% probability of success to the early stage RCC program due to a lack of clinical data, contributing ~$3 to our price target. As detailed on page 26, we estimate $463M and $171M at peak in for metastatic and early stage RCC, respectively.

Assumptions

We estimate a cost of $10,000 per dose, with patients receiving an average of eight doses in year one and four doses in subsequent years. We estimate that at steady state, 65% of new patients to therapy will receive all doses in year one and 45% of continuing patients will receive all doses for mRCC, changing to 70% and 50% in early stage RCC due to increased survival in this population. Due to a lack of pivotal data, we put in a placeholder of 15% and 10% penetration in the U.S. and EU, respectively, for mRCC and 5% penetration for early stage RCC. We assume 15% discounts in the U.S. and a 30% discount in price in the EU to arrive at our revenue estimates.

The near-term catalyst for the stock is a read-out of data from the AGS-004 study in HIV. While the regulatory path forward is not clear, we believe a positive result will provide further validation of the Arcelis platform and can act as a value driver for the stock. As a reminder, the AGS-004 study has been funded by NIH and we anticipate that studies beginning this year will likely be funded externally as well. Therefore, we see the HIV program as pure upside to shareholders and our analysis suggests that the potential market opportunity could approach $5B.

We derive our $17 price target by dividing the sum-of-the-parts NPV by ~22M diluted shares. We apply a terminal value due to the lack of generic competition as, in our view, intellectual property and trade secrets make the Arcelis platform difficult to replicate and we anticipate future expansion of the technology into other tumor types. We do not include cash in our valuation and we currently ascribe no value to the HIV program due to the lack of clarity on a path forward; we would consider this indication as upside.

FIGURE 3. Sum-of-the-Parts NPV Analysis

Product	Indication	Peak revenue	Peak revenue year	Probability of success	NPV	Per Share Value
AGS-003	mRCC	$463	2024	60%	$316,850	$14.17
AGS-003	RCC	$171	2024	30%	$63,481	$2.84
AGS-004	HIV	n/a	n/a	0%		
				Cash	$85,170	$3.81
				Total	**$380,331**	**$17.01**

Source: JMP Securities LLC

After completing its IPO at $8 last month, Argos has a current market cap of $209M. In our view, the company is undervalued compared to its peers in the targeted immunotherapy space and further undervalued compared to oncology companies with a late-stage un-partnered asset. We believe this is due to the lack of success in the field of targeted immunotherapies and, in our view, there is significant room for upside to Argos' shares as the differentiated profile of its platform becomes more apparent over the coming 12-18 months with the release of supportive data in HIV and RCC ahead of pivotal trial data in 2016.

FIGURE 4. Comparables Analysis

Company	Market Cap ($mil.)	EV ($mil)	Lead product	Dev. Stage
Dendreon	$521	$922	Provenge	Market
Agenus	$264	$240	QS-21	Phase III
Immunocellular	$79	$49	ICT-107	Phase III
New Link	$1,016	$971	Algenpantucel	PhaseIII
Northwest Biotherapeutics	$276	$269	DCVax	Phase III
Galena	$405	$359	NeuVax	Phase III
Average	**$427**	**$468**		
Argos	$209	$133	AGS-003	Phase III

Source: Company reports and JMP Securities LLC

Balance sheet

Excluding the over-allotment, Argos received $45M net cash from the IPO, which we estimate gives the company ~$85M pro forma, a sufficient amount, in our view, to get the company through to pivotal data in 2016. However, we believe the company may be opportunistic and supplement its balance sheet via a debt or equity financing accompanying a value inflection of the stock.

INVESTMENT RISKS

Clinical. Drug development is inherently risky and Argos' studies may not succeed. Argos is conducting a Phase III study based on a single-arm Phase II trial, which carries various risks. It is possible that the study may not be powered properly to demonstrate a benefit of AGS-003. There is also risk that the combination of AGS-003 and Sutent will not outperform Sutent alone due to changes in the patient population between Phase II and Phase III. Phase II data may be driven by factors outside of the mechanism of action of AGS-003 and may not translate into Phase III success. Manufacturing of AGS-003 may hinder enrollment rates, pushing out timelines to data. The company is also developing AGS-004 for HIV, an area where there is yet to be a successful vaccine. It is possible that AGS-004 will not succeed in allowing patients to discontinue anti-retroviral therapy.

Regulatory. The AGS-003 pivotal study, ADAPT, is being conducted under a SPA with the FDA; however, this does not guarantee approval. It is not known if the EMA will want more data, either clinical or manufacturing before allowing Argos to file for EMA approval. For AGS-004, the regulatory path forward and potential commercial market is unclear. The timing of validation of automated manufacturing could lead to a delay in filing the BLA for AGS-003.

Commercial. Argos is developing personalized immunotherapies for cancer and infectious disease. The development of these products requires a patient sample, either blood or tumor from surgery or biopsy, and patient cells obtained through leukapheresis. This may represent a paradigm shift in treatment and physicians may not be comfortable working these therapies into practice, creating commercial risk. Argos is finalizing automation of its manufacturing process; timing delays add risk to regulatory filing submissions. Unanticipated problems with automation may hinder Argos' ability to produce its therapies.

Competitive. Oncology drug development is competitive, with various companies bringing technologies forward that could make Argos' products obsolete. There are other targeted immunotherapies in development that, if successful, could be applied in RCC, adding direct competition to AGS-003.

Balance sheet. Following its IPO, Argos has cash to reach pivotal data; however, the company may seek additional equity financing, leading to dilution risk for shareholders. The company may seek debt financing for AGS-003 manufacturing facilities.

ARCELIS PLATFORM - BEST OF BOTH WORLDS OF IMMUNOTHERAPY

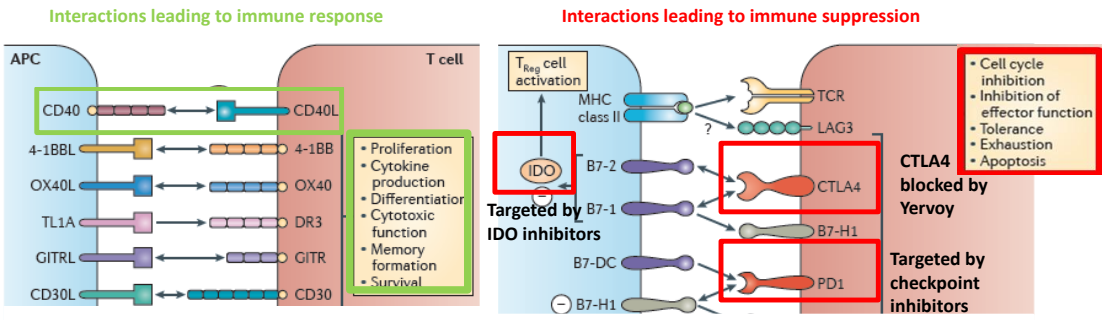
Argos' Arcelis technology – a proprietary targeted dendritic cell immunotherapy platform – differentiates Argos from the field of targeted immunotherapies by combining fundamental attributes of targeted cancer vaccines with key features of generalized immunotherapies.

- **Targeted approach**: The Arcelis technology uses antigens from patient tumors to generate dendritic cells primed to attack its mutated tumor antigens.
- **Avoids tumor immunosuppression**: The Arcelis technology programs dendritic cells to launch an immune response without needing to engage a patient's CD4+ T cells (T helper cells).

Tumors can become "invisible" to the immune system. Interfering with this process can shift clinical outcomes.

To launch an immune response, antigen presenting cells (APCs) interact with T cells to give stimulatory signals (Figure 5, left) that can attack tumors. However, in many cancer patients this attacking threat is neutralized by inhibitory interactions overexpressed by tumors, allowing tumors to become "invisible" to the immune system. Interfering with this balance and reawakening the immune system, therefore, has potential to shift clinical outcomes. This has been crystalized with the approval of Yervoy (ipilimumab), impressive results of the class of PD-1/PDL-1 "checkpoint" inhibitors, and the promise of indolamine 2,3 dioxygenase, or "IDO" inhibitors. All of these compounds block specific inhibitory interactions between T cells and APCs, either by inhibiting T cell receptors involved in immunosuppression (PD-1 or CTLA4) or by blocking the induction of T regulatory cells (IDO) (Figure 5, right).

FIGURE 5. Yin and Yang of T cell – APC Interactions – A Snapshot



Source: NATURE REVIEWS, IMMUNOLOGY VOLUME 13, APRIL 2013, pg. 227

Mechanism of action

AGS-003 is produced using Argos' proprietary Arcelis platform that generates dendritic cells (DCs) from patient monocytes and load them with his or her mutated tumor antigens to facilitate an immune attack against the tumor. The DCs are also loaded with RNA encoding CD40 ligand (CD40L), a CD4+ T cell signal that primes DCs (Figure 5, left). The CD40-CD40L interaction is an important one to activate DCs. By including CD40 ligand in the final product, AGS-003 DCs can prime T killer cells and secrete cytokines such as IL-12, needed to generate CD28+ memory T cells. Memory cells can, in turn, lead to a sustained immune response over time and are correlated with improved outcomes in various cancers. We believe the Arcelis platform overcomes previous limitations of other DC-based approaches. In our view, proof of mechanism for the Arcelis platform has been demonstrated in two different settings.

- **AGS-003**. Immunological data from Phase II in mRCC studies demonstrates an increase of tumor specific CD28+ memory cells from baseline that correlate with outcomes of overall survival, progression free survival, and tumor response.
- **AGS-004**. AGS-004, an Arcelis product using RNA isolated from the HIV virus instead of tumor cells, has shown decreased viral load in patients after a 12-week interruption in anti-retroviral therapy. In our view, generating an immune response in patients with HIV supports the proof of mechanism that these therapies can work in an immunosuppressed environment. We view the placebo-controlled Phase IIb data expected this summer as further validation of the platform.

ARGOS' DIFFERENTIATED DC PLATFORM TECNOLOGY

The Arcelis platform combines direct tumor targeting and the ability to overcome immunosuppression in one therapy.

The Arcelis platform combines tumor targeting and overcoming immunosuppression in one therapy, opening up potential for dramatic increases in patient survival. However, the area of targeted immunotherapies (cancer vaccines), in contrast to broader immunotherapies, has garnered mostly skepticism from investors. Most of this stems from the only approved therapy of this class – Dendreon's Provenge – which has been saddled with mediocre efficacy, high cost of goods and poor commercial performance. Below, we explore controversies surrounding targeted immunotherapies and why we believe Argos' Arcelis platform is set apart.

History of failure does not read-through to the Arcelis platform

Recent failures in the space, including GSK's MAGE-A3 therapy for melanoma and Immunocellular's ICT-107 for glioblastoma, have fueled questions of whether targeted immunotherapies can have an impact and improve clinical outcomes. MAGE-A3 is a recombinant protein vaccine with the MAGE-A3 antigen and ICT-107 is a DC therapy loaded with six peptide antigens. We believe there are two potential reasons for these failures – either incorrect targeting or an inability to overcome tumor immunosuppression. We believe the Arcelis technology is differentiated from these approaches by its focus on relevant targets, ability to mount an immune attack circumventing tumor immunosuppressed T cells, and demonstration of a clear mechanism of action.

The right target? MAGE-A3 and ICT-107 are loaded with peptides from MAGE-A3 and HER2, TRP-2, gp100, MAGE-1, IL13Ra2, and AIM-2 antigens, respectively. Each also contains an adjuvant to stimulate the immune system. The antigens targeted are self-antigens that are overexpressed on tumors. There are various risks, in our view, that accompany the use of tumor overexpressed antigens in a targeted immunotherapy.

- Previous exposure. Patients have been exposed to these antigens in the past; therefore, the body may be tolerant of these antigens and may not illicit a sufficient immune response.
- Mutations to antigens. Tumors may have mutated antigens to evade the immune system that cannot be recognized by "off-the-shelf" antigens. We believe this is especially likely if an antigen is not necessary for the survival of the cancer cell.
- One size fits all. Not all tumors will express the same antigens and many mutated antigens will differ between patients.

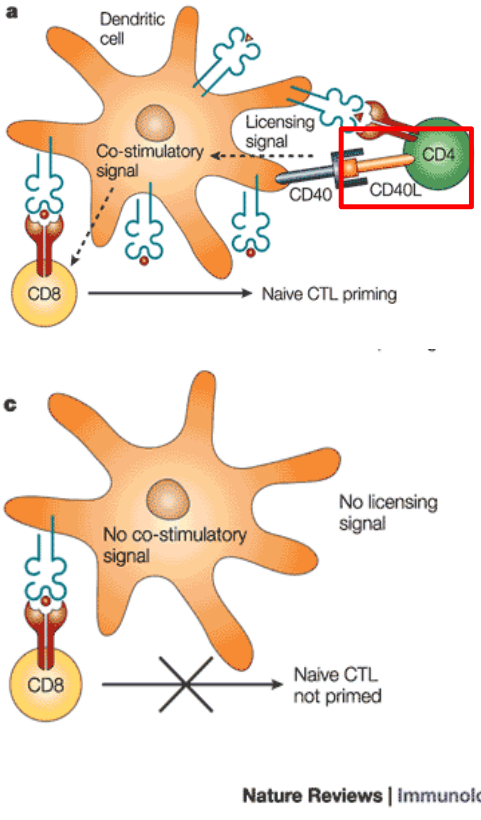
How is Arcelis different? The Argos platform uses RNA from a patient's tumor so that all antigens, including uniquely mutated ones, are available to prime immune responses. In our view, this gives Argos' technology a high chance of success.

We believe providing the right target is necessary, but not sufficient for targeted therapies to work.

Ability to overcome immune suppression? In our view, this is the most important shortcoming of the large majority of targeted immunotherapies. Generally, therapies have tumor antigens and include an adjuvant to stimulate the immune system and generate CD8+ T cells (T-killler cells, cytotoxic T lymphocyte ((CTL)); however, if the immune system is suppressed, this may not be sufficient to mount an immune attack. We believe providing the right target is necessary, but not sufficient for targeted therapies to work. In our view, the success of Yervoy and PD-1/PDL-1 inhibitors support the need to free the immune system to launch an attack.

How is Arcelis different? The Argos platform has engineered its dendritic cells (DCs) to activate an immune response without the need for a patient's CD4+ T cells. As illustrated in Figure 6, DCs need a "licensing signal" in the form of interactions with CD4+ T cells to prime CD8+ T killer cells and launch an attack. If these signals are not provided, the DCs, although loaded with antigens seen by T cells, are functionally inactive. The Arcelis platform encodes DCs with RNA for the CD40 ligand (CD40L), one of the key licensing signals for DCs. This allows Arcelis products to skip over the licensing step, launching CD4+ T cell independent priming, which we believe differentiates it from the field of DC and cell therapies.

FIGURE 6. Traditional Activation of CD8+ Effector T cells



CD40L is encoded in DCs from Arcelis platform

Source: Nature Reviews Immunology 1, 126-134

Predictability of RCC allows for single-arm studies to power controlled trials

A weakness in the development of many targeted therapeutics has been the lack of read-through from single-arm to controlled studies. In our view, two factors may contribute: 1) responders in early studies may respond due to robust baseline immune systems, not therapy; and 2) changing standards of care and a lack of data make it difficult to predict future control arm response, which may result in poor trial design and inaccurate powering of pivotal studies.

Outcomes correlate specifically with the *increase* in memory T cells, suggesting outcomes are related to therapy and not to a patient's baseline immune status.

How is Arcelis different? The Argos platform leads to generation of CD8+ CD28+ memory T cells. We are encouraged that clinical outcomes (overall survival (OS) and progression free survival (PFS)) correlate specifically with the *increase* in memory T cells from baseline to after the fifth priming dose of therapy. This correlation with the change of memory cells, not just the presence of immune cells, suggests to us that the clinical outcomes observed are related to therapy and not to a patient's baseline immune status.

Targeting a predictable population. Argos is pursuing its first indication in metastatic renal cell carcinoma (mRCC). mRCC was a logical choice for proof of concept, in our view, as there is a clear immunological component (cytokines have been used as therapy) and a clear way to access the risk of immunogenicity against the host by monitoring the potential attack of the healthy kidney. Another benefit of RCC is that recent publications of meta-analyses suggest that intermediate or poor-risk mRCC patients on tyrosine kinase inhibitor (TKI) therapy, the current standard of care for RCC, have predictable outcomes. In our view, these data mitigate the risk of a pivotal study design based on single-arm Phase II data, allowing the study to be powered correctly to observe a benefit.

Automated manufacturing designed for commercial success

Although Dendreon's Provenge demonstrated a modest benefit to prostate cancer patients, it had to be priced to account for the high cost of manufacturing. In our view, the majority of manufacturing issues were specific to Provenge, including the instability of the product, resulting in the need for localized processing facilities and the need for patients to be leukapheresed for each dose of Provenge.

How is Arcelis different? The Arcelis platform overcomes many of these hurdles by needing only one leukapheresis procedure for five years of dosing and by optimizing its manufacturing process to allow up to four days for the leukapheresis sample to get to a centralized manufacturing site (Figure 7). Once product is obtained, it can be frozen down in direct injectable containers that retain potency upon thawing for at least five years.

Argos will also complete the validation of an automated manufacturing process by the time of launch with the process validated in time for BLA filing. We see some risk associated with the transition from a manual to automated process, mostly from a timing of launch perspective; however, we are encouraged that the FDA will accept comparability data between manual and automated batches in the BLA for AGS-003 without the need for a bridging study. From a cost perspective, COGS should be ~$9,000, or ~10%, for five years of dosing in contrast to Provenge, where COGS has been close to $20,000 for each $31,000 dose. Automated capacity can also be built out in a modular fashion, allowing for spend to meet demand.

FIGURE 7. Differences between the Provenge and Arcelis Platforms

	Arcelis	Provenge
Antigen payload	• **ALL** tumor antigens • 100% **patient specific**	• Single antigen • May not apply to all patients
Immunogenicity	• Ability to culture 100% dendritic cells with high potency • **MOA correlates with survival**	• Only ~10% of cells are dendritic cells • No correlation
Applicability	• Potential for **ALL** cancers • **One** leukapheresis procedure • **Intradermal** injection	• Prostate cancer only • Three leukapheresis procedures • Intravenous infusion
Manufacturing	• One production run = **24 doses** • **Long-term stability:** can freeze cells and mRNA • **Automated, centralized production**	• One production run = one dose • Limited stability: only 18 hours; unable to freeze • Manual process; multiple facilities

Source: Company reports

We see manufacturing as a surmountable risk that will, at worst, delay the filing or launch of AGS-003 for a few months. However, considering the high-maintenance manufacturing process compared to most other therapeutics, we model a slower ramp than most oncology launches and cap penetration into advanced RCC patients at 15% and 10% in the U.S. and EU, respectively, with only 5% penetration into earlier stage patients. In our view, impressive clinical data and a fully automated manufacturing facility present meaningful upside to our estimates.

METASTATIC RENAL CELL CARCINOMA – HONING IN ON UNMET NEED

We give the pivotal study of AGS-003 a 60% chance of success based on the Phase II survival data, clear proof of mechanism, and the Phase III trial design.

AGS-003 is in Phase III development for metastatic renal cell carcinoma (mRCC). The pivotal trial is being run under the auspices of a special protocol assessment (SPA) with the FDA, with only one study needed for approval. Patients are randomized to standard of care tyrosine kinase inhibitor (TKI), Sutent, compared to AGS-003 in combination with Sutent and the trial is powered at 80% for a 6-month improvement in overall survival (OS) assuming a 16-month median survival of the control group. The target patient population is treatment naïve mRCC patients with intermediate or poor prognosis, defined minimally as less than one year of time from diagnosis to systemic treatment. We give the study a 60% chance of success based on Phase II survival data, clear proof of mechanism, and Phase III trial design, which we believe is optimized for success.

Renal cell carcinoma

Renal cell carcinoma, or kidney cancer, has an incidence in the U.S. of about 65,000 cases per year, or 2-3% of all cases diagnosed in the U.S. Incidence has grown in recent years, from 35,000 in 2005 to 49,000 in 2009, and 60,000 in 2013, likely due to improvements in early detection through imaging techniques; however, the mRCC population has stayed constant with about 10,000-12,000 patients newly diagnosed each year. We conservatively estimate 1% growth going forward. Cytokine therapy was used first line historically, with poor results and response rates hovering around 10% at best. Since first approval in 2006, tyrosine kinase inhibitors (TKIs), such as Sutent (sunitinib), inhibiting VEGF, and other kinases have become standard of care, with increases in progression free survival (PFS) and overall survival (OS) compared to cytokine therapy. Although VEGF is the main target of these drugs, Sutent and other TKIs have off-target effects, such as inhibition of c-kit, a cytokine receptor, leading to immune modulation, such as lowering levels of T regulatory cells (T regs), known to stimulate immunosuppression.

Current standard of care defines unmet need

There are two primary shortcomings to TKI therapies, in our view. The first is that the tolerability of Sutent precludes combination therapy, with side effects including GI events such as diarrhea, nausea, stomatitis and vomiting, fatigue, hypertension, and rash. Patients who cannot tolerate Sutent generally switch to another TKI therapy with a better tolerability profile, such as Votrient or Inlyta; however, these drugs have their own side effects.

The second shortcoming, in our opinion, is that despite impressive Phase III PFS (~11 months) and OS (~27 months) data for Sutent, recent retrospective meta-analyses of TKIs demonstrate that these data are mostly driven by patients with favorable risk profiles (zero risk factors), leaving a clear unmet need in high risk patients. Recent publications, including a study of 850 patients in the International Metastatic Renal Cell Carcinoma Database treated with first-line TKI therapy and a more refined data set presented at the ASCO GU conference in January, suggest there is a large drop off in benefit of TKI therapy in intermediate (1-2 risk factors) and poor risk (3+ risk factors) patients (Figure 8).

The six risk factors that have been validated to predict diminished overall survival are:

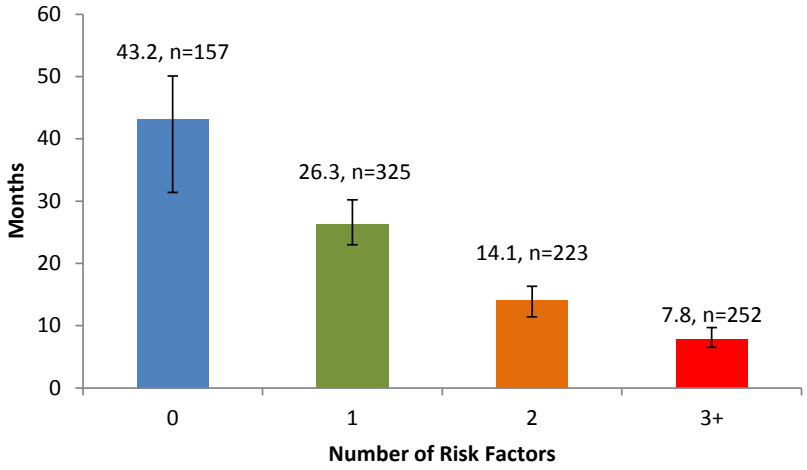
- Diagnosis to systemic treatment < 1 year (DxTx<1yr)
- Diminished performance status (PS)
- Elevated corrected calcium
- Anemia
- Elevated neutrophils
- Elevated platelets

Synchronous mRCC, where a patient presents at time of diagnosis with primary tumor intact and distant metastatic disease, by definition have at least the first risk factor. Generally, patients who are diagnosed, have surgery, and then relapse at a later date have much better survival prognosis.

The extensive analyses of survival in the mRCC population from patients treated with TKIs in the past five years have demonstrated the predictability of outcomes in intermediate- and poor-risk patients. We note that the range of survival decreases in the intermediate and poor prognosis groups compared to the group with zero risk factors, giving us further confidence in the predictability of these groups. In our view, this helps to mitigate the risk of uncertainty of performance of the control group in a pivotal study, allowing for a Phase III study to be powered correctly based upon historical data.

We believe a first-line therapy that improves survival in intermediate- and high-risk patients with a tolerability profile allowing for combination with TKIs would be an important advancement in the treatment of mRCC. In our view, if Phase II data for AGS-003 can be confirmed in the pivotal study, this will represent a significant step forward in the treatment of RCC.

FIGURE 8. Overall Survival (months) Stratified by Risk (literature reports)



Error bars = range of months

Source: Heng, Rini, Lee et al. J Clin Oncol. 2013

Argos is enrolling a pivotal study in intermediate- and poor-risk mRCC patients. By definition, these patients are newly diagnosed mRCC patients with the first risk factor of less than one year from diagnosis to systemic TKI treatment. To help us understand the potential performance of the control group in the pivotal study, we analyzed literature median overall survival per risk factor (Figure 9) and applied it to the pivotal trial population. As a reminder, the literature OS rates are based on patients seen in the last five years and there has been no change in standard of care during this time; therefore, we believe these data are relevant to a current study.

When we apply median overall survival predictions to patients enrolled in the ADAPT pivotal study as of January 2014, we see median overall survival of 15 months in the control arm; the AGS-003 pivotal study is powered for a control arm survival of 16 months (Figure 9). This analysis increases our confidence that the pivotal trial is properly powered to show a difference between arms.

FIGURE 9. OS Estimate Based on Enrollment Trends To Date

Risk factors	OS (months)	% Phase 3	Expected survival
1	26.3	18%	4.7
2	14.1	58%	8.2
3	9.7 *	13%	1.3
4	9.7 *	11%	1.1
Total anticipated OS in control			**15.2**

* assume upper end of the range

Source: Heng, Rini, Lee et al. J Clin Oncol. 2013, and Company reports

AGS-003 CLINICAL DATA

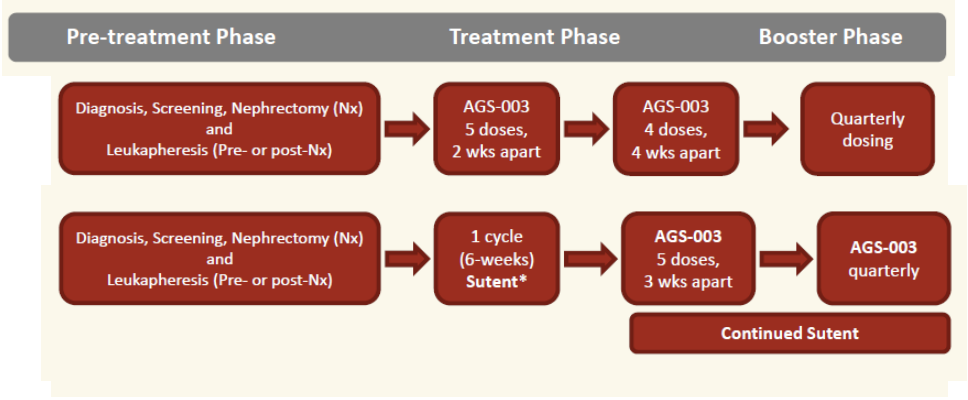
AGS-003 was tested both as monotherapy and in combination with Sutent, the accepted standard of care for first-line advanced RCC. The target population was synchronous mRCC patients newly diagnosed and scheduled for surgery. Tumor samples were obtained during surgery and shipped to Argos for AGS-003 manufacture. The leukapheresis procedure generally took place two weeks after surgery and the cells were shipped to Argos for processing.

Dosing schedule

AGS-003 monotherapy (trial AGS-003-004). For the monotherapy study, patients were given AGS-003 at a target dose of $1x10^7$ cells every two weeks for five weeks followed by four doses every four weeks, then quarterly dosing (Figure 10). This regimen was based upon literature data demonstrating a need for "priming" doses at the beginning of immunotherapy to kick-start a response.

Combination study (trial AGS-003-006). In the combination study, dosing was adapted to fit in with the dosing of Sutent, which is four weeks on and two weeks off. The first dose of AGS-003 is synchronized with the end of the Sutent cycle, 0-3 days before the first dose of Sutent in the second cycle in order to minimize dosing during periods of Sutent-induced lymphopenia, when immunity may be lowest. Based on the monotherapy study and literature data, five doses were administered as priming doses followed by "booster" doses once per quarter. If Sutent was stopped due to tolerability issues, AGS-003 was continued until progression of disease; however, if a patient progressed, dosing of all therapies was discontinued and the patient was likely given a second-line therapy, such as an mTOR inhibitor.

FIGURE 10. Trial Design of Phase II Studies



Source: Company reports

Patient population

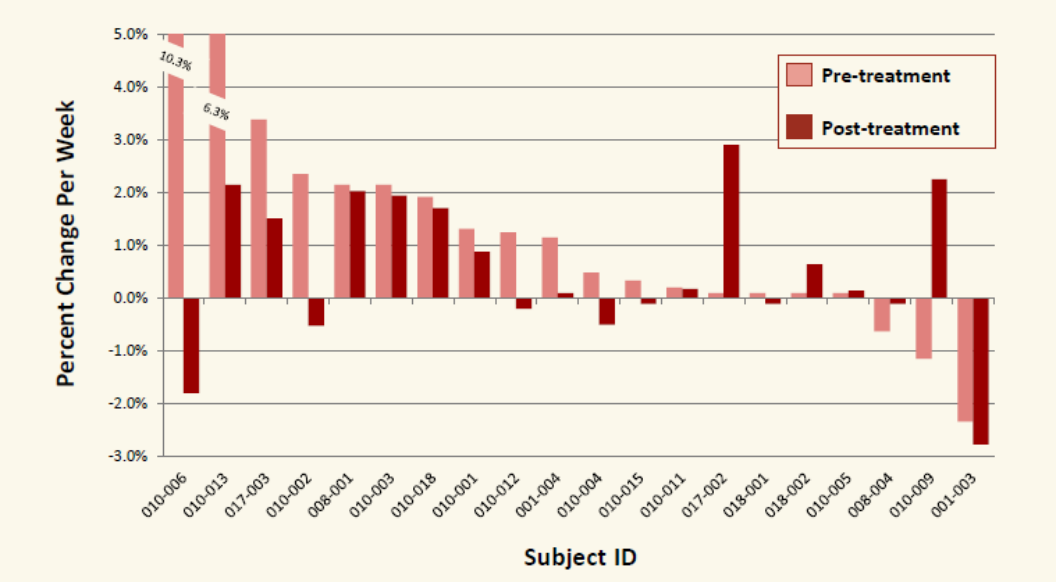
Intermediate- and poor-risk patients with newly diagnosed mRCC with a scheduled nephrectomy were eligible for therapy. In the 006 combination study, patients also had to be eligible for Sutent therapy. All patients had the risk factor of less than one year between diagnosis and systemic treatment. Patients could have 1-6 risk factors in each study, and 59% and 48% had three risk factors or more in the monotherapy and combination studies, respectively.

The overall survival of patients on standalone AGS-003 was 15.6 months, comparable to a historical control of 14.7 months survival on first-line TKI therapy.

Standalone efficacy data

The overall survival of patients on standalone AGS-003 was 15.6 months, comparable to a historical control of 14.7 months survival for a population of 66% and 34% of intermediate-and low-risk patients, respectively, on first-line TKI therapy. All but two patients received the first five "priming" doses of therapy and for those who received all five doses, ~55% of patients had a decrease in tumor growth rate (Figure 11).

FIGURE 11. Tumor Response After the First Five Doses of AGS-003



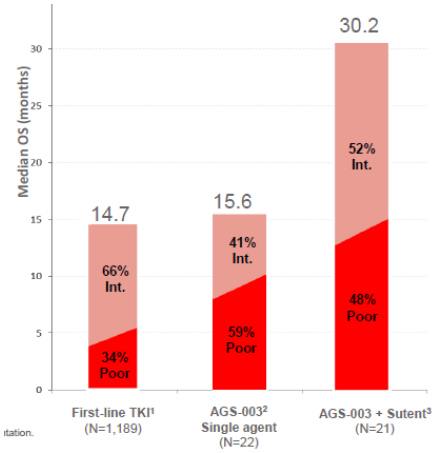
Source: Company reports

We believe these data support the efficacy of AGS-003, laid the groundwork for combination studies, explored dosing and established tolerability of therapy. Injection-site reactions were the most common adverse event (AE) along with transient flu-like symptoms and tender lymph nodes, which, in our view, are consistent with immune activation. All AEs were Grade 1 or 2 and no immunogenicity against the healthy kidney was observed.

Combination efficacy data

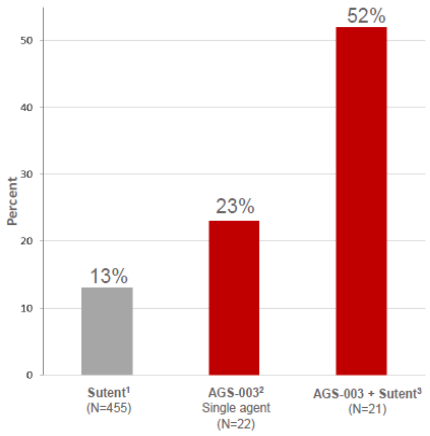
In the combination study of Sutent and AGS-003, median overall survival was 30.2 months, twice that of a risk-matched historical control (Figure 12). Long-term survival data (> 30 months) was 52% and although these data cannot be compared directly to historical data in all intermediate-risk patients on Sutent, we are encouraged that the combination of Sutent and AGS-003 appears to outperform TKI/Sutent therapy alone (Figure 13).

FIGURE 12. Median OS (TKI group historical)



Source: Heng, Rini, Lee et al. J Clin Oncol. 2013 and Company reports

FIGURE 13. Percent of Patients Surviving >30 Months (Sutent group historical)

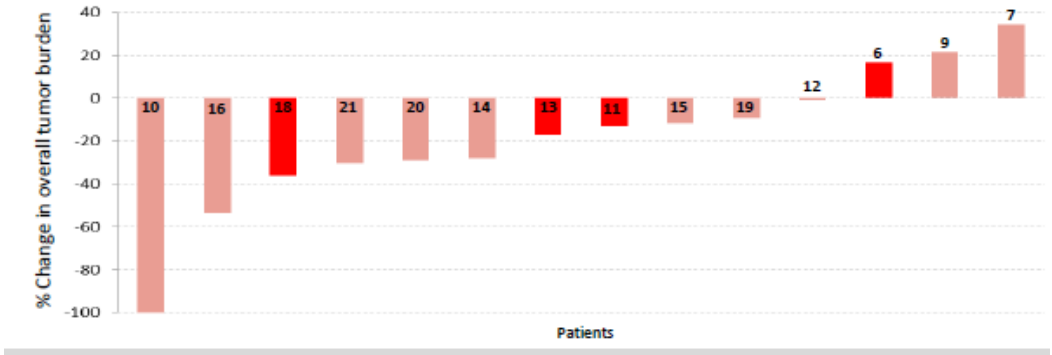


Source: Motzer et al. British Journal of Cancer (2013) 108:2470–247 and Company reports

Tumor response. In the AGS-003 combination study, most patients saw a decrease in tumor burden (Figure 14). Thirty-eight percent of patients met the criteria for partial responses (PR), defined as at least a 30% decrease in the longest diameter of target lesions, in line with or slightly higher than what is typically observed with Sutent alone.

Although it is difficult to say if Sutent or AGS-003 is driving the response as historical Sutent response data have never been risk stratified, the timing of the PRs gives us confidence in the contribution of AGS-003. It is established that most PRs observed with TKI therapy occur during the first few cycles of treatment, usually in the first six months of therapy. In the combination study, three of the eight PRs were in the first six months, two were at roughly one year and three occurred over two years into therapy, suggesting that AGS-003 is likely contributing to these responses. Overall response rate (PR + stable disease (SD)) was 62% from the first dose of AGS-003 and 81% from screening. In our view, the current Phase III study will inform the community on the response rate of higher risk populations and we believe there could be upside for Argos if the combination enhances this rate.

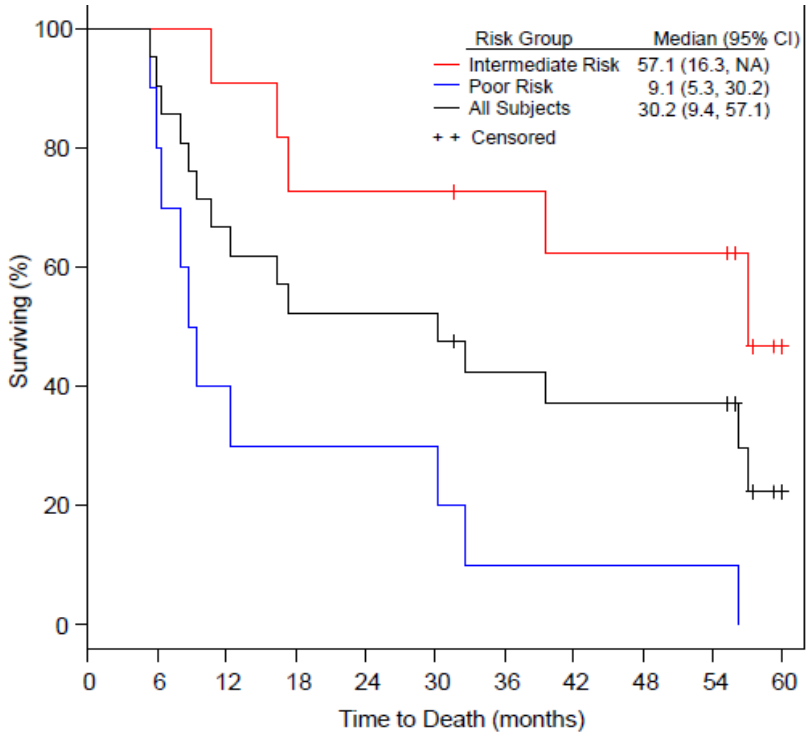
FIGURE 14. Best Tumor Response by Patient



Source: Company reports

Overall survival data stratified by risk. Median overall survival for the combination study was 30.2 months. However, stratifying survival by risk factors suggests an imbalance in response between intermediate- and poor-risk patients, with median survival of 57.1 months and 9.1 months for intermediate- and poor-risk patients, respectively (Figure 15). We believe that part of this difference is the expected lower survival of poor-risk patients; however, we also note a surprisingly large range (5.3-30.2 months at last cut) of survival in this group, which goes against historical data where the range of this group was tight (6.5-9.7 months). We believe part of this may be due to small numbers; however, we also believe these data can be partially explained by the observation that only 5/10 poor-risk patients in the combination study received the first five priming doses of AGS-003 and none of these patients survived for more than one year (Figure 16). In contrast, in the monotherapy study where 13/15 poor-risk patients received all five doses, outcomes appear better.

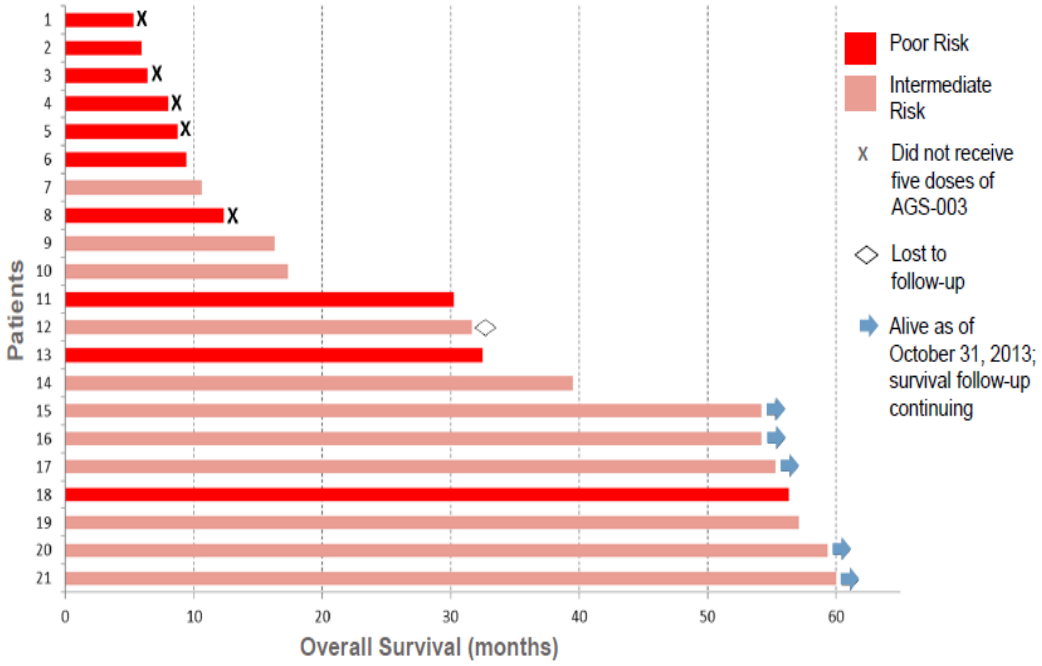
FIGURE 15. Kaplan-Meier Curves, Risk Stratified (Combination Study) as of YE13



** Data through Dec 2013*

Source: Company reports

FIGURE 16. Individual Patient Data – Combination Study

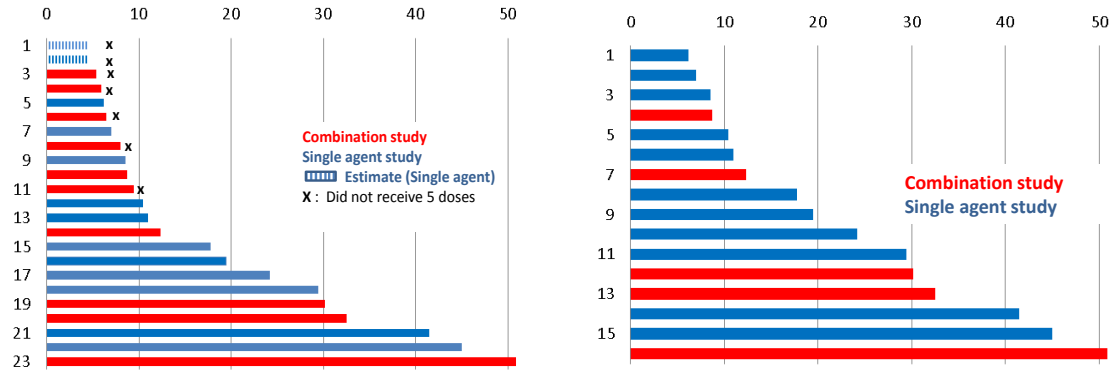


Source: Company reports

In poor-risk patients who received the first five doses of AGS-003, survival doubled historical expectations, with median overall survival close to 20 months.

To explore this further, we conducted an analysis of poor-risk patients from both the monotherapy and combination studies, specifically looking at the contribution of patients who received all five priming doses of AGS-003. In patients receiving all five doses, survival doubled historical expectations, with median overall survival close to 20 months, suggesting that AGS-003 can improve outcomes in this hard-to-treat group (Figure 17). We note that this number excludes one poor-risk patient who has survived 84+ months, but was rolled over from the monotherapy study to the combination study and did not fit the technical criteria (treatment naïve) to be included in this analysis.

FIGURE 17. OS in Poor-Risk Patients – All (left) and Those with ≥ 5 Doses

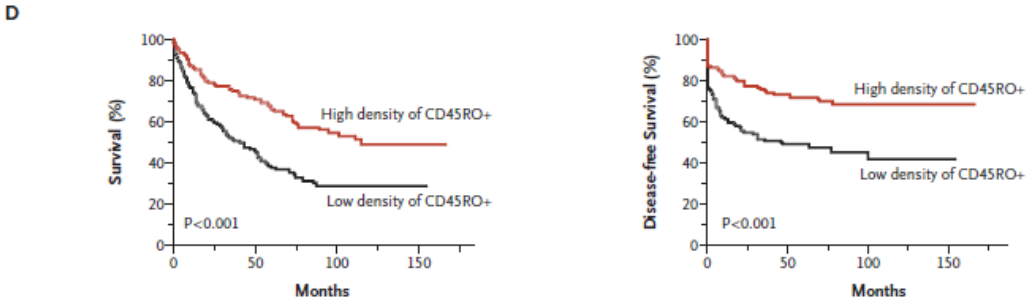


Source: Company reports, JMP Securities LLC

Proof of mechanism data

As a reminder, AGS-003 is engineered to jump-start an anti-tumor immune response via generation of memory cells that, in the literature, correlate with better outcomes. Specifically in RCC, memory cell (defined as CD45RO+) density correlates with disease free survival and overall survival (Figure 18).

FIGURE 18. Survival by Density of Memory Cells (CD45RO+) within RCC Tumors



Source: NEJM, 353: 25, 2005

During the 006 combination study, Argos collected blood from patients at time 0 and after three and five doses of AGS-003. Samples were exposed to AGS-003 or identical dendritic cells that lack tumor antigen RNA (negative control) to characterize immune responses using flow cytometry, a laser-based technique that can characterize cells by phenotypic and functional biomarkers.

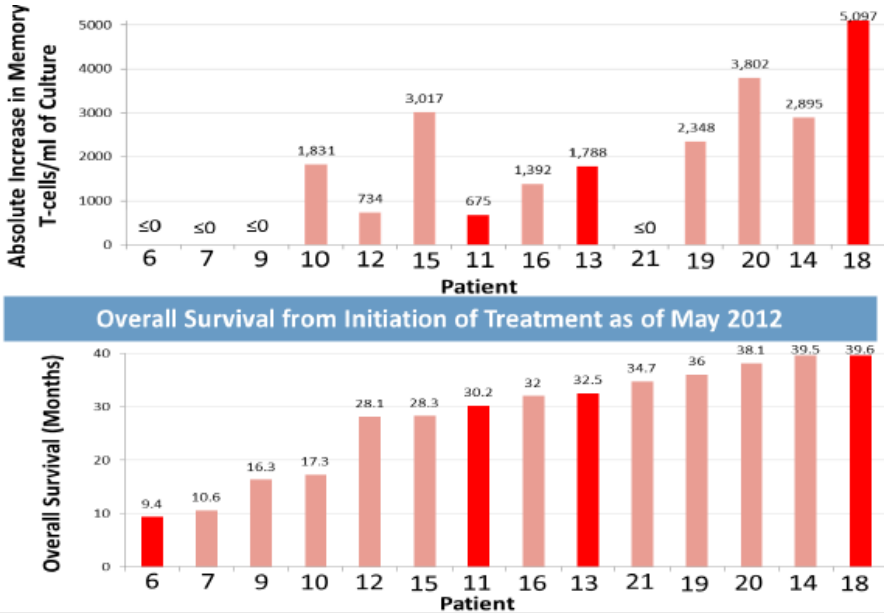
A key finding of these analyses, in our view, is that only after five doses of AGS-003 does the generation of memory cells correlate with outcomes of overall survival and progression free survival, supporting the five dose "priming" regimen (Figure 19). More importantly, the *change* in memory T cells from baseline to after the fifth dose correlates with outcomes with an increase in these memory cells correlated to longer PFS or OS. We believe these data confirm that the observed immune responses are specific to therapy and are not just an unrelated consequence of baseline immune responses that are therapy independent. We believe patient 21 in Figures 19 and 20 exemplifies this point. This patient did not see an increase of tumor-specific memory T cells; however, they demonstrated longer than average survival when the data was cut in May 2012.

Data accrued in the Phase II trial provides specific proof of mechanism for AGS-003, mitigating risk and giving us confidence in these data.

Our conversations with management suggest that this patient had higher than normal baseline levels of T cells, suggesting that either: 1) the delta in memory cells was too small to see with such a high background, or 2) this patient's response is not related to AGS-003 therapy. Even including this outlier in the analysis, the correlation of change in cells to PFS and OS is statistically significant, giving us confidence in the mechanism of action for AGS-003 and suggesting that the therapy should perform in a similar fashion in the pivotal study. We believe the clear mechanism of action bolsters clinical data from the Phase II study. In our view, accrual of supportive mechanism of action data over the next 12-18 months should partially de-risk AGS-003 ahead of pivotal data and be a catalyst for the stock.

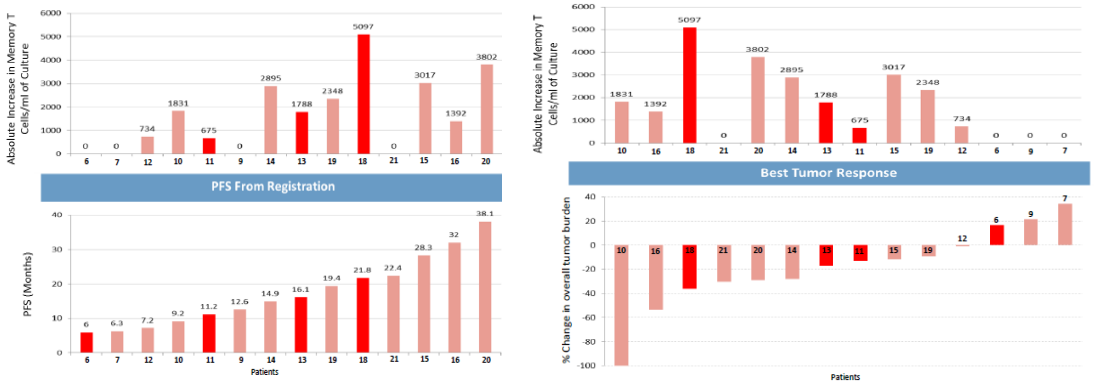
We look forward to the publication of these data in a peer-reviewed journal in the coming months, followed by a release of updated survival result at a medical conference this year. In our view, this will likely be at ASCO in June or at an immunotherapy conference in the fall.

FIGURE 19. OS Correlation



Source: Company reports

FIGURE 20. PFS (left) and Tumor Response (right) Correlation



Source: Company reports

Tolerability profile. The adverse events in the study were consistent with the Sutent profile with the exception of injection-site reactions expected to occur with AGS-003 therapy. No Grade 3/4 AEs were attributable to AGS-003 (Figure 21).

FIGURE 21. Side Effects in the Combination Study

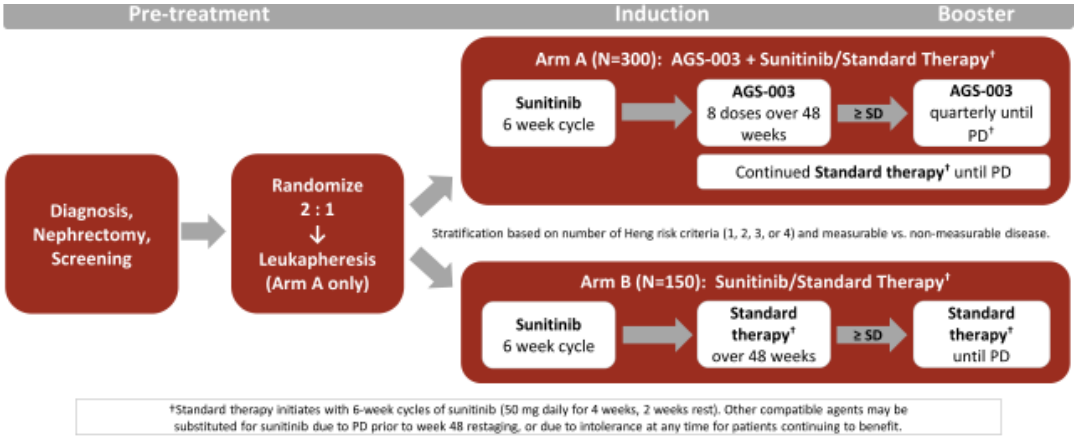
Adverse Event	N (%)	
	All Grades	Grade 3 or 4
Diarrhea	13 (61.9%)	1 (4.5%)
Fatigue	12 (57.1%)	2 (9.1%)
Nausea	11 (52.4%)	0 (0.0%)
Rash	9 (42.9%)	0 (0.0%)
Weight decreased	8 (38.1%)	2 (9.1%)
Peripheral edema	8 (38.1%)	0 (0.0%)
Headache	7 (33.3%)	0 (0.0%)
Injection site erythema	7 (33.3%)	0 (0.0%)

Source: Company reports

PIVOTAL TRIAL DESIGNED FOR SUCCESS

The pivotal study ADAPT is a 450-person, 2:1 open-label, randomized trial comparing Sutent alone to Sutent with AGS-003. The primary endpoint is overall survival; therefore, we are not concerned about the unblinded nature of the study. In the combination arm, patients will first receive one cycle of Sutent prior to the introduction of AGS-003 (Figure 22). The first cycle of Sutent serves two purposes – to provide time for manufacture of AGS-003 and to prime the immune system by decreasing T regs.

FIGURE 22. Pivotal Trial Design



Source: Company reports

The study is powered at 80% to demonstrate a six-month improvement in survival based upon control overall survival of 16 months. Argos intends to enroll 75% intermediate-risk patients and 25% poor-risk patients. All patients must have less than one year between diagnosis and systemic treatment and can have up to three other risk factors consisting of diminished performance status, elevated corrected calcium, anemia, elevated neutrophils, or elevated platelets. The study began enrolling patients in early 2013. Looking at enrollment trends as of January, 156 patients passed screening criteria and the majority of patients had two risk factors.

Phase II data set up an optimized Phase III study

Argos has maximized its chance for success with the design of the Phase III study, ADAPT.

Based on both the recent publication of risk-stratified survival data from patients on TKIs and the Phase II results of AGS-003 in combination with Sutent, we believe Argos has maximized its chance for success with the design of the Phase III study, ADAPT.

- **Patient population**. Similar to the Phase II combination study, the Phase III study will enroll synchronous mRCC patients who, by definition, have at least one risk factor. However, the patient population will be stratified by risk and skewed toward intermediate-risk patients (~75%) with recent enrollment suggesting that a majority have two risk factors. Intermediate-risk patients fared well in the Phase II study and, in our opinion, should lead to robust responses and separation from placebo in ADAPT. We note that although this makes the study population less sick than the Phase II study, these patients still represent those of highest need who are not adequately helped by the current standard of care due to their advanced disease.

In contrast to Phase II where patients could enroll with one-six risk factors, the Phase III study is capped at four risk factors. Patients with five or six risk factors, about 5-10% of the synchronous mRCC population, are unlikely to survive six months to allow for all priming doses of AGS-003 to be administered. Our analysis of patients receiving five doses of AGS-003 compared to those that did not suggests to us that capping risk factors should increase the OS of the population compared to the Phase II study, allowing for a potential AGS-003 benefit to be observed.

- **Predicted outcomes support powering**. We ran an analysis of potential survival rates on both arms of the pivotal study to evaluate whether we believe the study is adequately powered to show a treatment effect for the AGS-003 and Sutent combination. We compared literature data for intermediate- and poor-risk patients to risk stratified median overall survival observed in the Phase II AGS-003 combination study. We then applied these rates to the planned enrollment criteria for Phase III of 75% intermediate-risk patients. This analysis leads to a control group that outperforms the company's expectation; likely due to overestimating patients with one risk factor and not two. However, even with a more favorable median OS in the control arm, the OS observed in the AGS-003 combination suggests a 45-month median survival for the combination arm, almost twice as much as needed for the study to hit the parameters set in the SPA. This gives us confidence that the AGS-003 combination should adequately demonstrate a significant improvement over Sutent alone.

FIGURE 23. Analysis of Predicted OS Rates Based on Literature/Phase II Data

Population	OS (mos)	Phase 3 target enrollment	
Int	22.5 *	75%	16.9
Poor	7.8 *	25%	2.0
	Total anticipated OS in control		**18.8**
Int	57.1 **	75%	42.8
Poor	9.1 **	25%	2.3
	Total anticipated OS on AGS-003		**45.1**

* The Lancet Oncology Vol 14

**clinical data

Source: Lancet Oncology 2013, Company reports, JMP Securities LLC

- **Moving beyond Sutent**. In the AGS-003 Phase II combination study, patients stopped therapy with disease progression and Sutent was the only TKI able to be investigated in combination. In ADAPT, although patients must go through one cycle of Sutent, they are then allowed to switch TKI therapy if their disease progresses or they cannot tolerate the drug. It is expected that patients who cannot tolerate Sutent (~15% of patients) will be switched to another VEGF inhibitor and those progressing will be switched to an mTOR inhibitor, usually reserved for second-line therapy. In our view, these data will be useful commercially, as data generated with other TKIs in combination should give physicians comfort that AGS-003 can fit into normal treatment paradigms. The ability to dose with other TKIs also gives physicians the option to dose AGS-003 through progression. In our view, this will help ensure that patients receive all five priming doses of AGS-003, potentially increasing outcomes. We believe these are positives, although moving beyond Sutent does add potential for increased clinical risk.

- **Changing therapies can improve outcomes in the control population**. We believe the risk of this affecting the outcome of ADAPT is small, as all VEGF inhibitors show median overall survival that is equal to or worse than Sutent. mTOR agents used second line have shown overall survival of only about 10 months.

- **Other compounds may not be synergistic with AGS-003**. In our view, this is also a low probability scenario. Although not yet proven in the clinic, Argos has tested other therapies in combination with AGS-003 ex-vivo to look for changes in the outgrowth of memory cells, its intended mechanism of action. The only compound that interferes with this process is Nexavar, contraindicated for AGS-003. This interaction is likely due to an off-target effect; therefore, we believe the other compounds should work in a similar fashion to Sutent and not confound results. We note that literature data on the profile of all TKIs for RCC suggest that these compounds demonstrate c-Kit inhibition and therefore are likely to have the same immune modulating properties as Sutent.

Phase III challenges and expectations

- **Enrollment**. ADAPT in being conducted currently at 110 sites in North America, the EU, and Israel, with another 30 sites anticipated to open this spring. For a patient to be enrolled in ADAPT, they need to have newly diagnosed synchronous mRCC with surgery scheduled. Tumor collection occurs during surgery and samples are shipped to Argos. Only patients with clear cell mRCC are allowed into the study as TKIs are only approved in this population. Argos had anticipated about a 35% screen failure for the study due to presence of non-clear cell mRCC, too many risk factors or patient comorbidities. However, in reality, this rate has been close to 50%, driven mostly by a rate of 25% non-clear cell mRCC, higher than the expected 10-15% observed for RCC as a whole. Argos now expects to screen 900 patients to reach the requisite 450 patient enrollment and guidance of completed enrollment by the end of 2014 is in line with these numbers.

- **Interim analyses**. There are scheduled interim analyses at 25%, 50%, and 75% of events in the study, expected to occur in 2015. At each analysis, the trial continues if there is an opportunity to achieve at least over a four-month improvement in OS. Final analysis will be conducted when there are 290 deaths on trial, likely in early 2016. In our view, the Phase II data and powering of the study for a six-month improvement in overall survival on a control group of 16 months suggest to us that the study should continue without interruption and in our view, each interim analysis will be incrementally de-risking. We see upside for Argos shares if the study is stopped early for efficacy, which we see as possible at the 75% analysis, although we do not include this scenario in our valuation.

Regulatory path

As a reminder, ADAPT is being run under a SPA and is the only trial needed for approval. We believe if the six-month increase in overall survival is achieved, the regulatory path will be straightforward. AGS-003 has been granted fast-track status by the FDA; therefore, we would expect a priority review, setting up for 2016 approval and 2017 launch.

We believe the gating factor for filing and approval for AGS-003 will be manufacturing. Currently, the three components involved in the Arcelis manufacturing process (i.e., making the DCs, handling the patient RNA, and making the final product) are being conducted manually. However, Argos is working on the final modifications of an automated system that will mimic the manual process while increasing capacity and throughput. The company intends to break ground on a facility this year and have the automation process validated and ready for BLA inspection by the end of 2015. The FDA will not require a bridging study to switch the manufacturing over to an automated process and will only require comparability data between the two processes, which we believe simplifies BLA submission.

We note that the EMA may require a bridging study between the manual and automated processes; therefore, to be conservative we model a two-year gap between U.S. and EU approval and launch of AGS-003.

Phase II studies broaden opportunity and further de-risk platform

Argos plans to initiate studies this year that, in our view, will help to de-risk the RCC program by providing additional proof of mechanism data as well as broaden the potential scope of patients that can be treated with AGS-003.

Core biopsy study. In 1H14, Argos will sponsor a study at Duke in mRCC patients that will manufacture AGS-003 from core biopsy samples, rather than tumor samples acquired through surgery. In our view, this Phase II study will support the efficacy of AGS-003 in all untreated mRCC patients, either relapsed or synchronous. As a reminder, half of the mRCC patients are not newly diagnosed but have relapsed after undergoing nephrectomy at an earlier time point; therefore, tumor samples for AGS-003 manufacturing cannot be acquired through surgery. Our conversations with Argos management suggest that using biopsies as a source of tumor material is not much different than using tumor samples. However, we look forward to this being validated in a clinical trial setting. We view this as an important trial from a commercial standpoint as: 1) it will enable broader use of AGS-003 in mRCC, and 2) it could lead to expansion into other cancer types where surgery is not part of the standard of care. We see upside to our valuation if Argos expands into other tumor types.

Early stage RCC. In 2H14, Argos will sponsor investigator-led studies in early RCC patients, defined as those who, in standard practice, would have surgery and then be monitored with a "watch and wait" strategy. The goal of this study is to collect mechanistic immunological data from these patients and see if AGS-003 can prevent/delay relapse to metastatic disease. In our view, it will be more difficult to discern a clear benefit of AGS-003 in these patients as, by definition, many of the patients will be of favorable risk and thus, have more variable and longer term survival. To mitigate this risk, the study site will use matched controls from its database and strict entry criteria in order to control for variability and have a group for comparison. It is our expectation that these data will lead to a randomized pivotal trial for approval and launch two years after the mRCC population.

COMPETITIVE LANDSCAPE

The clean safety profile of AGS-003 enables its use in combination with small molecule inhibitors of VEGF and mTOR for RCC; therefore, we do not see new drugs in these classes as competitive threats to AGS-003. However, there are studies in progress now that may crowd the RCC landscape, especially those investigating checkpoint inhibitors. We believe AGS-003 could be synergistic with PD-1 inhibitors to prime the immune system and potentially improve upon AGS-003 activity. We anticipate that Argos will likely initiate a study of this combination by the end of this year. In our view, these data may become important as checkpoint inhibitors enter into the paradigm of treatment for RCC.

Checkpoint inhibitors: PD-1 or PDL-1 inhibitors are a logical fit for RCC as a cancer with a clear immunological component. Although the cells generated by AGS-003 are PD-1 negative, it is likely that PD-1 is expressed in RCC tumor cells present at baseline. Below we summarize some of the more advanced competitors in the immuno-oncology space.

- **Nivolumab**. Bristol is running a Phase III study of its PD-1 antibody, Nivolumab, against everolimus, an mTOR inhibitor, in advanced/metastatic RCC patients who have previously progressed on therapy. The primary endpoint is overall survival and the study is expected to read-out in 1H16. Though enrolling at the same time as ADAPT, we do not see this trial as a competitor for patients, as the treatment is second line. In a small Phase 1/2 study of 34 refractory patients, overall survival was 22 months.

  Bristol is also running a Phase 1 study in 72 advanced RCC patients to look at the tolerability of Nivolumab in combination with TKIs or Yervoy. The primary endpoint is safety, although tumor response is being measured. Data is expected this year. In our view, these data should give us an idea of the tolerability of these combinations and feasibility of using checkpoint inhibitors as first-line therapy in RCC. If positive, we believe continued development is likely. Although AGS-003 will be ahead in development, we see Nivolumab as a potential competitor. We believe positive data from this study would make a combination study of AGS-003 and checkpoint inhibitor(s) important from a commercial standpoint.

- **MK-3475**. Merck's PD-1 antibody, currently in registration with FDA, is being tested first line with Votrient in advanced RCC and will soon be tested in combination with Inlyta, although the population is not yet defined for the latter trial. Once again, if these combinations are tolerable, we believe it could put competitive pressure on AGS-003, increasing the need for Argos to run combination studies with these agents.

- **CT-011**. This checkpoint inhibitor is being investigated in a Phase II study sponsored with the NIH that explores combination with a DC vaccine. The vaccine is a fusion of DC cells and tumor cells. The vaccine itself is not likely, in our view, to overcome tumor immunosuppression; however, it is possible that combination with CT-011 may enhance its activity. We anticipate more details when the trial reads out, potentially as early as in 2H14. We view this study as early stage and not a commercial threat; however, these data may generate excitement for PD-1/vaccine combinations if successful.

Targeted immunotherapies. To our knowledge, most targeted immunotherapies are not being pursued in RCC at this time. However, if any of the therapies in development were to be successful, they could be explored in RCC. The one RCC targeted therapy in Phase III development is described below.

- **IMA901**. IMA901 is immactics' cancer vaccine, incorporating 10 HLA tumor-associated peptides discovered from patient tumors (Figure 24) and GM-CSF as an adjuvant. The compound is being tested in combination with Sutent against Sutent alone in favorable and intermediate-risk RCC patients. Interim data is expected in 1H14. In our view, IMA901 differs from AGS-003 in two ways: 1) use of antigens that may or may not be necessary for tumor survival, and 2) no inherent mechanism to overcome tumor immune suppression. These factors give us little confidence in a positive outcome for the study. However, in Phase 1, it was demonstrated that IMA901 decreases T regs; therefore, it may be synergistic with Sutent, which also decreases T regs. If successful, we believe that immatics may be a competitive threat to AGS-003 depending upon how the data look in intermediate-risk patients.

FIGURE 24. Antigens Targeted by IMA901

#	Peptide ID	Allele	Antigen	Common acronyms and synonyms
1	IMA-ADF-001	HLA-A*02	Adipophilin	adipose differentiation-related protein, ADRP
2	IMA-APO-001	HLA-A*02	Apolipoprotein L1	APOL1
3	IMA-CCN-001	HLA-A*02	Cyclin D1	CCND1, PRAD1, parathyroid adenomatosis 1, BCL-1
4	IMA-GUC-001	HLA-A*02	GUCY1A3	guanylate cyclase 1-soluble-alpha 3
5	IMA-K67-001	HLA-A*02	KIAA0367	--
6	IMA-MET-001	HLA-A*02	c-met proto-oncogene	MET, HGF (hepatocyte growth factor) receptor, HGFR
7	IMA-MUC-001	HLA-A*02	MUC1	mucin, CD227, episialin, epithelial membrane antigen
8	IMA-RGS-001	HLA-A*02	RGS-5	regulator of G-protein signalling 5
9	IMA-ADF-002	HLA-A*02	Adipophilin	adipose differentiation-related protein, ADRP
10	IMA-MMP-001	HLA-DR	MMP7	matrix metalloproteinase 7
11	IMA-HBV-001	HLA-A*02	HBV core Antigen	HBc, HBcAg, cAg

Source: Company reports

COMMERCIAL OPPORTUNITY

Renal cell carcinoma, or kidney cancer, has an incidence in the U.S. of ~65,000 cases per year, or 2-3% of all cases diagnosed in the U.S. Incidence has grown in recent years, from 35,000 in 2005 to 49,000 in 2009, and 60,000 in 2013, likely due to improvements in early detection through imaging techniques. However, the rate of newly diagnosed mRCC patients has stayed relatively constant at about 10,000-12,000 cases a year. With these factors in mind, we estimate 1% growth going forward.

mRCC – the first step

About 25-30% RCC patients have metastatic RCC - 24,000 and 20,000 in the U.S. and EU, respectively. AGS-003 is being tested in clear cell synchronous mRCC, or Stage IV patients, representing about half of the metastatic patients. The other half has recurred with metastatic disease after being diagnosed and undergoing surgery at an earlier date. Argos will run a supportive Phase II study in this population beginning this year, and we believe this expanded population will be the population likely on the label for AGS-003 at launch in 2017. In our view, these patients encompass the greatest unmet need in RCC. We model launch in 2017 in the U.S. and 2019 in the EU, assuming the need for a bridging study in the EU before MAA submission

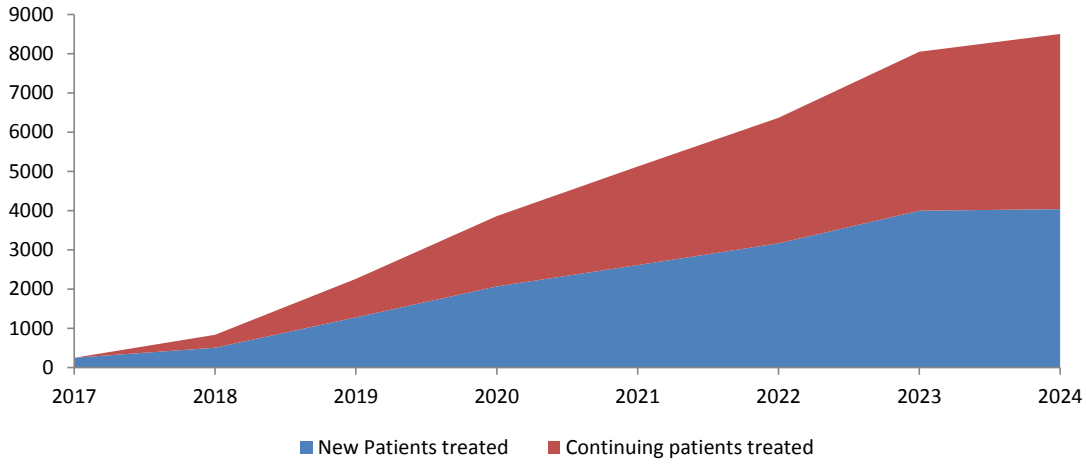
Due to the patient-tailored aspect of therapy, we believe that AGS-003 penetration into this market is linked to various factors, including clinical data, manufacturing, and physician education. We have modeled in a relatively slow ramp for AGS-003 compared to other oncology products due to the education of physicians and surgeons that will be needed on how to implement leukapheresis and the shipping of samples for manufacture. As a reminder, RCC is currently treated with small molecule oral therapies; therefore, use of AGS-003 will represent a paradigm shift. We currently estimate 15% and 10% penetration in the U.S. and EU, respectively.

In our view, there is room for significant upside to our market penetration estimates. We believe the clinical data is the key factor controlling market share. Physicians in the field believe a four+ month improvement in overall survival would be clinically meaningful; therefore, if ADAPT hits the six-month bar set forth in the SPA, we believe the therapy should be commercially viable. Greater than six months survival benefit, in our view, could be a source of significant upside

We do not see manufacturing capacity as a gating factor; at launch Argos intends to have capacity for over 2,000 patients and can increase capacity in a modular fashion over time.

We estimate a cost of $10,000 per dose, with an average of eight doses in year one and four doses in subsequent years. We estimate that at steady state, 65% of new patients to therapy will receive all doses in year one and 45% of continuing patients will receive all doses (Figure 25), although these rates may change depending upon Phase III data. Assuming 15% discounts in the U.S. and a 30% discount in price in the EU, we arrive at $463M in peak revenue for this indication in 2024.

FIGURE 25. Estimated Numbers of mRCC Patients on Therapy Over Time (U.S. + EU)



Source: JMP Securities LLC

Early RCC

Argos will initiate studies in early stage RCC this year. We assume a Phase III study and sBLA will be needed for approval in this population and model a 2019 U.S. launch with an EU launch one year behind. We estimate peak penetration of 5% into the target populations of ~22,000 and ~18,000 patients in the U.S. and EU, respectively. Our estimates are lower in this population as we believe that the higher risk subsets of this population would primarily use AGS-003; however, with no clinical data as of yet, it is hard to estimate. We keep cost and discounts the same as for mRCC; however, we have increased the percent of patients receiving all doses of drug to 70% and 50% in year one and in subsequent years, respectively, due to the earlier stage of disease. These assumptions give us $171M in peak revenue in 2024.

Commercial strategy

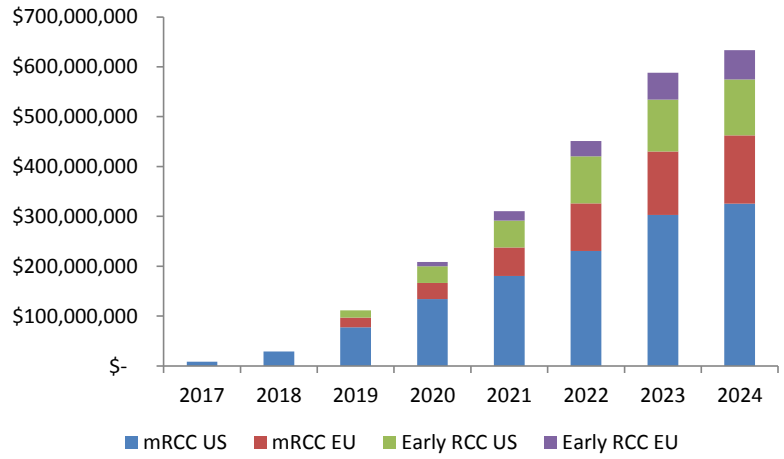
Argos plans to market AGS-003 in the U.S. and potentially in the EU. In our view, a small sales force will be needed, allowing Argos to execute without a partner. Most mRCC patients are treated in the community; Argos will target both urologic oncologists who perform surgery to collect tumor samples and also medical oncologists who treat patients post-surgery. The company believes demand will start with the 2,500-3,000 urologists who perform nephrectomies and broaden from there.

Argos plans to launch with a force of about 50-60. A large part of Argos's commercial efforts will be the logistics of tumor collection and leukapheresis as well as dose delivery. In addition to this force, 10-12 medical science liaisons will be ready for launch and the company intends to begin educational programs and market preparations in 2015. In our view, physician education of how to integrate AGS-003 into standard practice will be a key to uptake and we are encouraged that Argos will begin this process well in advance of launch.

In the EU, Argos intends to either launch on its own or to partner. In our view, there will be a lag between the U.S. and EU launches as requirements for EU approval are less clear at this time, giving Argos time to either find a partner or to prepare for commercialization and potentially start-up a European manufacturing site. Currently, all EU clinical trial sites are receiving AGS-003 product from the U.S. and in our view, it will be feasible to begin EU launch with manufacturing only in the U.S. and build out an EU facility only when demand warrants it.

Our revenue build for RCC is shown in Figure 26 with $634M estimated in 2024.

FIGURE 26. Estimated RCC Revenue Build



Source: JMP Securities LLC

HIV CONTROL – POTENTIAL FOR UPSIDE

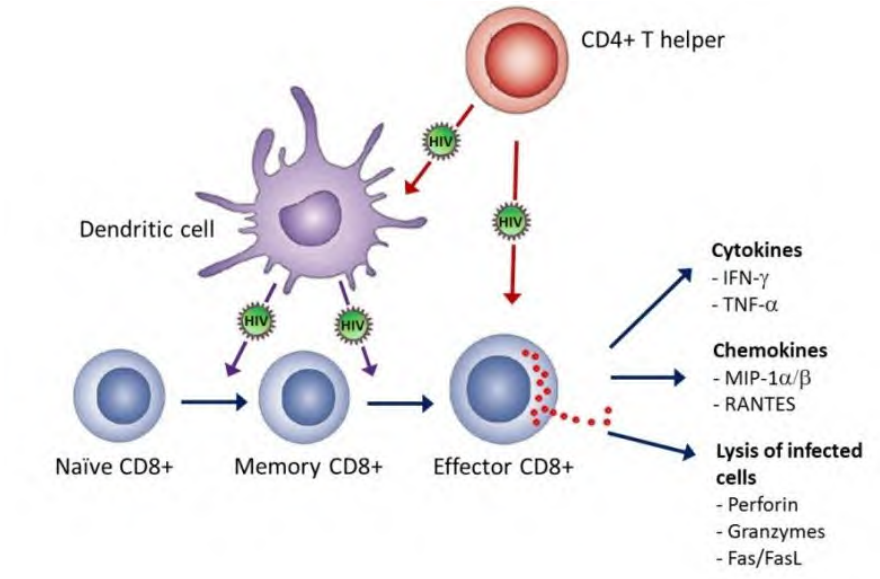
The AGS-004 program in HIV is mostly funded by grants from the government; therefore, in our view, this program represents a free flyer and potential upside to investors. We believe these data can help to validate the Arcelis platform mechanism of action and be supportive of the AGS-003 program, adding value to Argos shares.

An approach that can increase memory cells has potential to help patients control their disease in the absence of ART.

The HIV virus interferes with patients' immune systems, blocking activation of CD8+ cells to launch an attack against the virus (Figure 27). There has been a lot of research conducted on patients who are HIV positive but are long-term non-progressors, or "elite controllers". Although infected with HIV, these patients have undetectable virus without taking anti-retroviral therapy (ART) and have normal levels of CD4+ T cells. However, if the virus from these patients is studied ex-vivo, it can infect other cells, leading to the hypothesis that it is the host that makes an elite controller, not the virus itself. Various studies into the mechanism of disease control suggest that these patients have memory cells that can attack the virus. Therefore, an approach that can increase memory cells has the potential to help patients control their disease in the absence of ART.

The Arcelis platform enables Argos to create immunotherapies that can target specific mutated antigens and increase CD8+ CD28+ memory T cells by circumventing patients' CD4+ T cells. Therefore, this platform has the potential to restore immunity in patients who are HIV positive. As in cancer immunotherapy, we believe the Arcelis platform can succeed where other HIV vaccines have failed due to: 1) the use of patient specific antigens, and 2) the ability to launch an immune response without CD4+ cells.

FIGURE 27. How HIV Effects DC and T cell Signaling



Source: De Haes, Intech, http://dx.doi.org/10.5772/51583

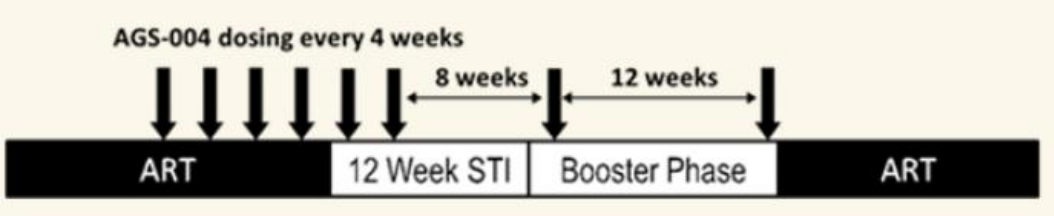
AGS-004

AGS-004 uses the same design tenets as AGS-003. The HIV virus is isolated from the archived blood of patients and mRNA encoding four HIV antigens (Gag, Nef, Revand, Vpr) is amplified for loading into DCs. Interestingly, to date, no two patients have had the same sequence for any of the four antigens amplified, underscoring the need for therapy to specifically address patient's mutated antigens. The manufacturing of the DCs is identical to the process for AGS-003; the only difference is that RNA is acquired from the blood of patients and not tumor samples.

Phase IIa established proof of concept

Study design. Argos completed a single-arm Phase IIa study in patients chronically infected with HIV, infected for an average of about two years prior to initiating ART therapy. AGS-004 was dosed every four weeks on top of background ART and after four doses of AGS-004, patients interrupted ART for 12 weeks. AGS-004 continued to be dosed every four weeks and then quarterly (Figure 28). Four patients involved in the study had received eight doses of AGS-004 prior to treatment interruption instead of four because they were rolled over from the Phase 1 tolerability study of AGS-004.

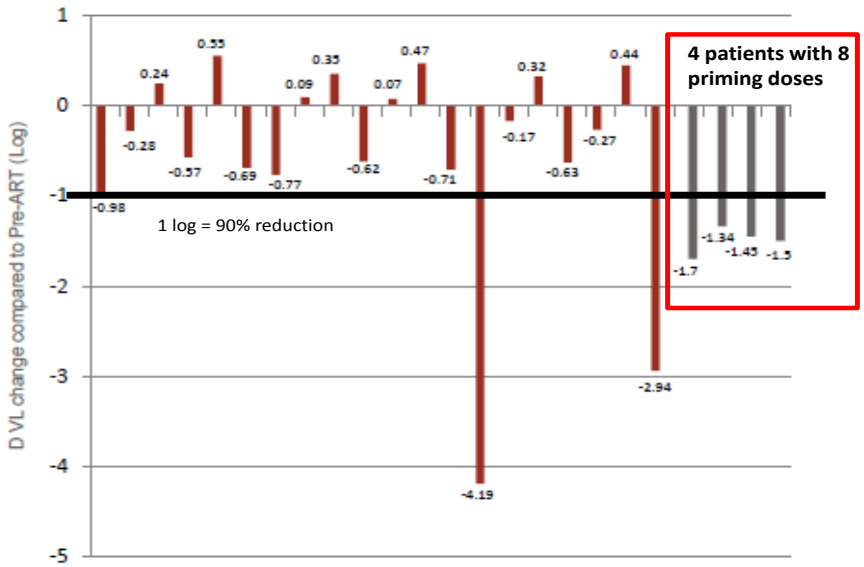
FIGURE 28. Phase IIa Trial Design



Source: Company reports

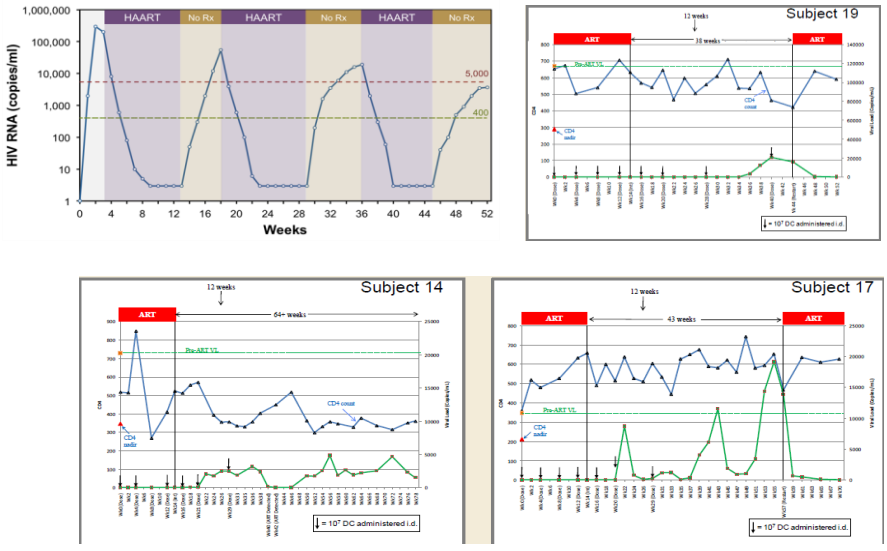
Efficacy. Patients in the Phase IIa study saw a viral load increase of 20% after ART removal and at 12 weeks after ART interruption, 81% of patients saw a decrease in viral load (Figure 29) compared to their "set point" or the steady state level of virus after HIV infection and before initiation of ART (Figure 30, upper left-hand corner). The four patients with eight priming doses of AGS-004 saw a 97% decrease in viral load compared to their set point. One patient has been off ART for four years with normal CD4+ T cell levels. In a typical HIV patient, interruption of ART will lead to the rapid increase of viral load to the patient's set point. Looking at representative patient level data (Figure 30), it appears that for most patients, T cell levels stay at/above 350 and the viral load does not rebound.

FIGURE 29. Change in Viral Load Compared to Set Point after ART Treatment Interruption



Source: Company reports

FIGURE 30. Typical Viral Load Rebound (upper left); Patient Level Data from AGS-004 Phase IIa Trial



Source: Company reports

Argos is conducting an NIH-funded, placebo-controlled, randomized Phase IIb study to confirm the AGS-004 Phase IIa results in a blinded trial and to collect immunological data looking for CD28+ T cells as a proof of mechanism. Patients are dosed continuously for 12 months with AGS-004 – four doses on top of background ART and then for 12 weeks after ART treatment interruption. After the 12-week treatment interruption, AGS-004 can still be dosed without ART if CD4+ T cell counts are above 450 and viral load is under 10,000 copies, a number considered to be correlative of a low risk of developing AIDS. The study is fully enrolled with 53 patients and we anticipate data this summer.

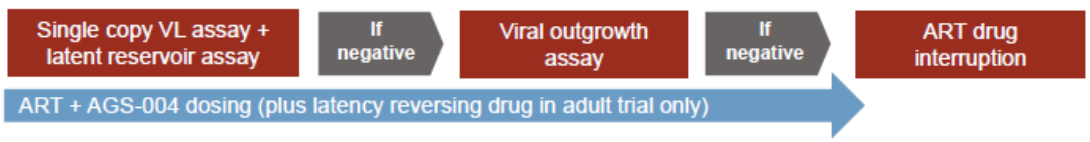
There was also an open-label acute arm of the Phase IIb study, with acute patients defined as those who initiate ART therapy within 45 days of primary HIV infection. These patients are expected to have more robust immune systems than chronic patients who waited months or years after infection to begin ART. This study did not require all patients to interrupt therapy at a set time, only if they met pre-specified criteria. Six acute patients were given AGS-004 on top of ART; immune responses were monitored after three doses of AGS-003 and a patient had to have at least four doses of AGS-004 before interruption of therapy. The criteria for treatment interruption included immune responses at pre-specified levels, viral load less than 50 copies/mL (undetectable) and CD4+ T cell counts above 350. All six patients were eligible for treatment interruption and we anticipate some of these data will be presented at CROI this week.

Phase IIb data – what it means

Read-through. In our view, the Phase IIb data should help to validate the Arcelis platform and further support proof of mechanism. Argos is looking for CD28+ memory cells in HIV patients and how the increase in these cells correlates with viral load reduction and outcomes. We believe these data, if positive, support the mechanism of both AGS-004 and AGS-003. We also note that this is the first controlled study to evaluate the Arcelis platform. Looking at differences in immune response between the AGS-004 group and placebo should help support and characterize therapy-specific immune responses.

Path forward. Even with positive Phase II data, we do not expect Argos to run a similarly designed Phase III study. Advances in technology, such as detecting single copies of the virus or the ability to measure latent reservoirs of HIV virus in organs such as the brain or bone marrow that are not depleted with ART therapy, have opened up other opportunities in trial design. These alternative designs, similar to the Phase IIb acute study, can test the efficacy of AGS-004 without having patients interrupt ART therapy until it is determined that they have undetectable viral load and that virus is not competent (by viral outgrowth assay to measure viral persistence) (Figure 31). If both tests are negative, then ART can be interrupted.

FIGURE 31. Future AGS-004 Trial Design



Source: Company reports

Argos will initiate two investigator led studies in 2014 for AGS-004 that we believe will mostly be funded by the NIH; therefore, we view the program as a free flyer with potential for significant upside for investors. The first study will be run in adults, the second in pediatrics.

- **Adult study**. The adult study will explore the potential to rid patients of reservoir HIV by using HDAC inhibitor induced expression of HIV from reservoirs. The hypothesis is that AGS-004 can help the immune system attack the virus when activated by HDACs, clearing the reservoirs, allowing for patients to then discontinue ART.
- **Pediatric study.** This study will focus on the cohort of teenagers who have been on ART since birth or shortly thereafter, who have very little latent reservoirs of the virus. The immune system of these patients has never been exposed to the virus; therefore, AGS-004 will be a surrogate immune system that can deplete the small amounts of virus that are present, in an attempt to turn these patients into elite controllers. There is likely no infectious plasma archived for these patients to extract RNA; thus, Argos can grow the ex vivo virus from reservoir samples to make product.

If either of these studies is successful in providing a mechanism to move even a subset of patients off of ART therapy as elite controllers, we believe it will help clarify a regulatory path forward and potentially catalyze partnering discussions from large pharmaceutical players in the HIV space such as Gilead, GSK, or Merck. However, due to a lack of clarity on a path to market, we see this program as upside for Argos and do not include it in our valuation at this time.

MANUFACTURING AND COLLABORATIONS

The Arcelis platform consists of a manual process to: 1) prep patients' cells obtained by leukapheresis into DCs; 2) extract and amplify RNA obtained from patients' tumors; and 3) assemble the final AGS product. The company is automating each step in order to increase capacity and lower costs (Figure 32). Argos has an agreement with the FDA that no bridging study will be needed for approval of AGS-003, only comparability data between the manual and automated processes. We believe the company has solved most of the technical risk of automation. In our view, the FDA's allowance of a switch to automated manufacturing between Phase III and commercial product suggests that the automated process is similar enough to the manual process to not derail plans for commercialization. However, we believe Argos has set aggressive time lines to have all machines and disposables validated by the end of 2015; we see some risk in the timelines for BLA filing if validation stretches into the early part of 2016.

FIGURE 32. Arcelis Manufacturing

Source: Company reports

We believe Argos will seek non-dilutive debt financing for a manufacturing facility (~$20M) with the intent to break ground this year. In our opinion, the financial risk is mitigated to some extent as Argos can build out manufacturing to meet demand in a modular fashion and will likely have to invest only 25% of costs prior to launch, with 40% of costs deferred until Phase III data. A set of three machines to complete the manufacturing steps costs about $1.5M and we estimate that Argos will have three sets on hand for launch to generate product for 2,000 patients. The company intends to ultimately have 15 sets of machines.

The manufacturing process has been designed to fit into standard current practice for RCC. Tumor sample has one week to get to Argos; it is stable for 10 days. The leukapheresis sample, usually taken from a patient two weeks after surgery, has to arrive at Argos in four days. These timelines allow for manufacturing to be centralized at one site. This is followed by six days of processing once the leukapheresis sample arrives at the site and then quality control and validation which take 2-4 weeks. Electrophoresis is used to get both patient RNA and CD40L RNA into the cells and the CD40L can be used as an internal standard for transfection efficiency.

Release criteria test both the stability and potency of the final product. The product is then frozen down into a directly injectable container. When thawed, it is tested for viability and potency, measured by the ability to secrete IL-12, and then injected into patients by intradermal injection. Argos has demonstrated no loss in potency through five years of sample storage.

Overall, we believe manufacturing should not be a limiting factor in the launch of AGS-003. The company plans to have data supporting product manufacture from tumor samples obtained from surgery or biopsy. In our view, the greater risk is commercial due to the need for a paradigm shift in RCC therapy and the burden of physician education.

Argos has granted Medinet Co, LTD an exclusive license to manufacture AGS-003 in Japan for mRCC. This was accompanied by a $1M payment from Medinet and a $9M loan that will be paid down with milestones for each interim analysis for AGS-003 and final data read-out. If the loan is not repaid, Medinet will be given a non-exclusive royalty bearing license to sell Arcelis products for cancer in Japan.

Argos has entered an exclusive agreement with Green Cross to develop and commercialize AGS-003 for mRCC in South Korea. Argos will receive royalties from mid-single to low-double-digit teens on net sales for 15 years from the first commercial sale in South Korea.

In Russia and related countries (Armenia, Azerbaijan, Belarus, Georgia, Kazakhstan, Kyrgyzstan, Moldova, Tajikistan, Turkmenistan, Ukraine, and Uzbekistan) Argos has granted exclusive license to AGS-003 to Pharmstandard. Argos has rights to clinical data generated by Pharmstandard and will receive tiered royalties from low-single digits to mid-double-digit teens.

INTELLECTUAL PROPERTY

Argos has a variety of patents and patent applications that last through to 2033 and 2027 in the U.S. and EU, respectively. In our view, the patent suite and trade secrets involved in the Arcelis manufacturing process should protect Argos from generic competition.

Argos licensed patents from Celldex claiming composition of matter for CD40L, entitling Celldex to a $1M milestone at first commercial sale of AGS-003 and a capped royalty. However, these rights terminate upon expiration of the CD40L composition of matter patents in November 2016; therefore, we believe Argos will not likely pay any milestones or royalties, as we model a 2017 AGS-003 launch. The company also licensed a patent from Duke covering the use of RNA to load dendritic cells that expires in April 2017.

We do not see expiration of these patents as a risk to Argos due to the other patents in its portfolio. Specifically, there is an issued patent covering its proprietary CD40L construct and method of use that does not expire until 2025. Therefore, even if other companies will be able to use RNA in DCs beginning in 2017, they will not have access to the CD40L or to the method of maturing DCs that is proprietary to Argos. Argos also has a patent on the use of monocytes that are at least six hours old that expires in 2029. This patent covers the technology that allows for Argos to ship monocytes to a centralized facility in lieu of culturing them right away, protecting its centralized manufacturing. Patents specifically related to AGS-004 for HIV expire in 2028, before extensions.

MANAGEMENT TEAM

Argos was founded by Ralph Steinman who won the Nobel Prize for discovering dendritic cells and its role in adaptive immunity. Management team members have been with Argos for at least six years. CSO Charles Nicolette was previously the Director of Antigen Discovery at Genzyme and Doug Plessinger worked on the Velcade launch at Millennium and was involved in immunotherapy at BMS prior to joining Argos.

FIGURE 33. Management Team

	Position	Prior Experience
Jeff Abbey, J.D., M.B.A.	President & CEO	•IAN, Eilenberg & Krause, Debevoise & Plimpton
Charles Nicolette, Ph.D.	CSO & VP Research & Dev.	•Genzyme Molecular Oncology, Cold Spring Harbor Laboratory
Fred Miesowicz, Ph.D.	COO & VP Manufacturing	•Gamida-Cell, Hybridon Specialty Products, Cellcor, a subsidiary of Cytogen, DuPont
Doug Plessinger, RPh	VP Clinical and Medical Affairs	•BMS, Millennium Pharmaceuticals, inVentiv Health
Lori Harrelson, CPA	VP Finance	•LipoScience, Ernst & Young

Source: Company reports

FIGURE 34. JMP Estimated Income Statement

	FY011A	FY012A	Mar-13A	Jun-13A	Sep-13A	Dec-13E	FY013E	Mar-14E	Jun-14E	Sep-14E	Dec-14E	FY014E	FY015E	FY016E	FY017E	FY018E	FY019E	FY20E	FY21E	FY22E	FY23E	FY24E
Revenues:																						
AGS-003		0	0	0	0	0	0	0	0	0	0	0	0	0	8,538	28,610	111,577	208,348	310,413	451,212	588,010	633,557
mRCC (clear)													0	0	8,538	28,610	96,590	166,587	237,625	326,260	430,299	462,645
mRCC (nonclear)														0	0	0	14,986	41,762	72,788	124,952	157,711	170,912
other																						
AGS-004			0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
							0					0	0	0	0	0	0	0	0	0	0	0
Total product revenue	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**8,538**	**28,610**	**111,577**	**208,348**	**310,413**	**451,212**	**588,010**	**633,557**
Total revenue	**7,643**	**7,039**	**800**	**1,000**	**1,500**	**1,700**	**5,000**	**1,200**	**1,000**	**800**	**300**	**3,300**	**200**	**0**	**8,538**	**28,610**	**111,577**	**208,348**	**310,413**	**451,212**	**588,010**	**633,557**
Operating expenses:																						
COGS	0	0	-	0	0	-	-	-	0	0	-	-	-	-	6,233	13,733	31,242	37,503	55,874	81,218	105,842	114,040
R&D	12668	17,617	5500	5650	5842	6000	22,992	8000	9000	10000	10500	37,500	40,000	40,000	40,000	40,000	40,000	40,000	40,000	40,000	40,000	40,000
G&A	3704	6,136	1100	1200	1405	1600	5,305	1800	2000	2000	2000	7,800	7,878	12,000	18,000	25,000	31,250	39,063	48,828	61,035	76,294	95,367
Total operating expenses	**16,372**	**23,752**	**6600**	**6,850**	**7,247**	**7,600**	**28,297**	**9800**	**11,000**	**12,000**	**12,500**	**45,300**	**47,878**	**52,000**	**64,238**	**78,735**	**102,492**	**116,566**	**144,703**	**182,254**	**222,136**	**249,408**
Loss from operations	**(8,729)**	**(16,713)**	**(5,800)**	**(5,850)**	**(5,747)**	**(5,900)**	**(23,297)**	**(8,600)**	**(10,000)**	**(11,200)**	**(12,200)**	**(42,000)**	**(47,678)**	**(52,000)**	**(55,700)**	**(50,125)**	**9,085**	**91,783**	**165,710**	**268,958**	**365,874**	**384,149**
Other income (expense):																						
Interest income	0	6242	2	2	3	6	12	12	10	12	10	30	6342	90	99	63	45	70	3	5	9	14
Interest expense	(11412)	0	0	0	0	0	0	(68)	(68)	(68)	(68)	(270)	(270)	(270)	(270)	(270)	0	0	0	0	0	0
Other, net	-	0		0	0				0	0												
Total other income (expense)	**(11412)**	**6242**	**2**	**2**	**3**	**6**	**12**	**12**	**(58)**	**(55)**	**(57)**	**(240)**	**6072**	**(180)**	**(171)**	**(207)**	**45**	**70**	**3**	**5**	**9**	**14**
Pretax income	**(20,141)**	**(10,471)**	**(5,798)**	**(5,848)**	**(5,744)**	**(5,894)**	**(23,285)**	**(8,588)**	**(10,058)**	**(11,255)**	**(12,257)**	**(42,240)**	**(41,606)**	**(52,180)**	**(55,871)**	**(50,332)**	**9,130**	**91,853**	**165,713**	**268,964**	**365,884**	**384,163**
	(927)	(352)																				
Income Taxes	-	-	0.06	*124*			-	0.06	*124*			-	-	-	-	-	*456*	*9,185*	*24,857*	*67,241*	*109,765*	*134,457*
Tax rate							0%					0%	0%	0%	0%	0%	5%	10%	15%	25%	30%	35%
Net loss	**(21,067)**	**(10,824)**	**(5,798)**	**(5,972)**	**(5,744)**	**(5,894)**	**(23,285)**	**(8,588)**	**(10,182)**	**(11,255)**	**(12,257)**	**(42,240)**	**(41,669)**	**(52,180)**	**(55,871)**	**(50,332)**	**8,673**	**82,668**	**140,856**	**201,723**	**256,119**	**249,706**
EPS																						
Basic	**(32.88)**	**(9.10)**	**(4.83)**	**(4.78)**	**(4.19)**	**(3.82)**	**(17.36)**	**(0.50)**	**(0.51)**	**(0.57)**	**(0.61)**	**(2.19)**	**(1.50)**	**(1.52)**	**(1.61)**	**(1.43)**	**0.24**	**2.28**	**3.83**	**5.41**	**6.78**	**6.53**
Weighted Shares Outstanding																						
Basic	639	1,190	1,200	1,250	1,369	1,545	1,341	17,281	19,869	19,919	19,969	19,260	27,760	34,260	34,760	35,260	35,760	36,260	36,760	37,260	37,760	38,260

Source: Company reports and JMP Securities LLC

JMP FACTS AND DISCLOSURES

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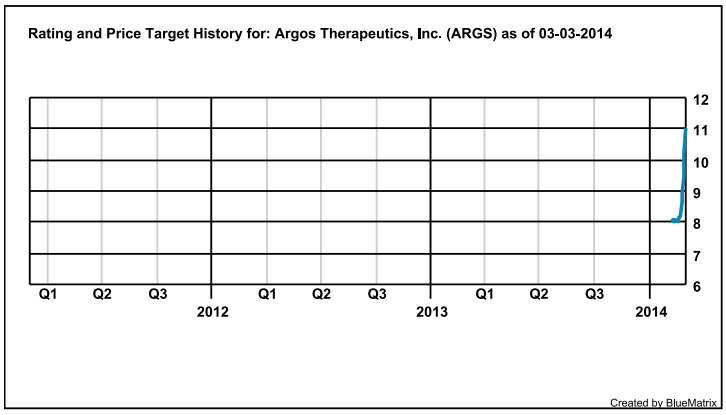
JMP Rating	Regulatory Equivalent	# Co's Under Coverage	*% of Total*	Regulatory Equivalent	# Co's Under Coverage	*% of Total*	# Co's Receiving IB Services in Past 12 Months	*% of Co's With This Rating*
MARKET OUTPERFORM	Buy	244	*56.22%*	Buy	244	*56.22%*	94	*38.52%*
MARKET PERFORM	Hold	139	*32.03%*	Hold	139	*32.03%*	18	*12.95%*
MARKET UNDERPERFORM	Sell	8	*1.84%*	Sell	8	*1.84%*	0	*0%*
COVERAGE IN TRANSITION		43	*9.91%*		43	*9.91%*	0	*0%*
TOTAL:		434	*100%*		434	*100%*	112	*25.81%*

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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