William Blair

Tokai Pharmaceuticals, Inc.

A Novel Prostate Cancer Agent Heading Down an Efficient Path to Approval: Initiating Coverage With an Outperform Rating and $44 Price Target

We are initiating coverage of Tokai Pharmaceuticals with an Outperform rating and a price target of $44, based on our belief that the lead asset galeterone will play an important role in the prostate cancer treatment paradigm and achieve peak worldwide sales of $1.8 billion from the first indication. Galeterone encompasses the mechanisms of action of both current blockbuster Zytiga from Johnson & Johnson and blockbuster-to-be Xtandi from Medivation and Astellas, and goes one step further to actively degrade the androgen receptor (AR). This leads to its effectiveness in a subset of chemo-naïve mCRPC (metastatic castration-resistant prostate cancer) patients, likely 20%, who do not respond to Zytiga or Xtandi. On the safety side, galeterone has demonstrated a benign profile to date, without the need for concomitant prednisone administration like Zytiga, and is devoid of theoretical seizure potential as compared with Xtandi.

We believe that Tokai has identified a highly efficient path to market for galeterone and could create substantial value in a relatively short time. Galeterone is poised to enter Phase III development in chemo-naïve mCRPC (or M1) patients who harbor the AR-V7 splice variant in their AR expression that renders these patients unresponsive to Zytiga or Xtandi. We estimate the study to begin in early 2015, with top-line data released in late 2016 and potential approval and launch in late 2017 or early 2018.

We value Tokai's stock at $44 per share for the AR-V7 indication alone, based on our probability-adjusted net present value (NPV) model. We estimate worldwide sales for galeterone will reach $1.8 billion in the United States and Europe in 2027 in the AR-V7 variant population alone. Assuming an 85% probability of success, our probability-adjusted NPV model suggests a fair value for Tokai shares at $44 at the end of 2015.

Substantial upside exists to our valuation. Further label extension and geographical expansion of galeterone are upside to our valuation. We believe that galeterone could challenge the leadership position of Xtandi based on its potential superior resistance profile and comparable safety profile; we currently project Xtandi as the largest share taker in the future prostate cancer treatment landscape with $7.6 billion in worldwide peak sales. Further, Tokai's AR-degradation discovery platform could produce more clinical candidates with superior resistance profiles that follow galeterone.

Tokai Pharmaceuticals, Inc. is a biopharmaceutical company based in Cambridge, Massachusetts, focused on the development of galeterone and an androgen receptor-degradation platform to address prostate cancer and potentially other hormone-driven cancers.

October 13, 2014
Basic Report (14-127)

Stock Rating:	**Outperform**
Company Profile:	**Aggressive Growth**
Price Target:	$44.00
Symbol:	TKAI (Nasdaq)
Price:	$11.55 (52-Wk.: $11.24–$30.00)
Market Value (mil.):	$252
Fiscal Year End:	December

Estimates	**2013A**	**2014E**	**2015E**
EPS FY	-$3.61	-$1.30	-$1.47
Sales (mil.)	$0	$0	$0
Valuation			
P/E	NM	NM	NM

Trading Data	
Shares Outstanding (mil.)	21.8
Float (mil.)	6.5
Average Daily Volume	166,357
Financial Data	
Total Debt/Total Capital	0
Enterprise Value (mil.)	$148
Price/Book	NM

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Investment Overview

Prostate Cancer Is the Most Commonly Diagnosed Cancer in Men in the United States
According to the National Cancer Institute (NCI), nearly 2.5 million men in the United States are living with prostate cancer, with about 215,000 new cases diagnosed each year. About one in six men will be diagnosed with prostate cancer in their lifetime. Of the newly diagnosed cases, about 80% are found localized to the prostate and relatively benign, whereas 20% are advanced, including 5% that are already metastatic. Although localized prostate cancer has a 99% five-year survival rate, once the disease reaches the metastatic stage, the survival rate falls to 30%. In 2014, it is expected that prostate cancer will claim the lives of nearly 30,000 men in the United States.

Targeting the AR Axis Is the Foundation of Prostate Cancer Treatment; We Ascribe One-Half of the Future Prostate Cancer Market to Agents Inhibiting the AR Axis
Prostate cancer is an androgen (mostly testosterone and dihydrotestosterone or DHT)-dependent disease, and activation of the androgen-androgen receptor (AR) pathway is the key mechanism for prostate cancer growth at all stages of the disease. Suppressing the AR axis is therefore the foundation of prostate cancer treatment.

- ***Two major ways of inhibiting the AR signaling pathway: lowering androgen levels, and blocking the AR. Zytiga and Xtandi are representative new blockbuster agents.*** The first-line therapy to lower androgen levels includes androgen-depravation therapy, such as Lupron, that reduces testosterone levels comparable to that of surgical castration. Zytiga is a second-line therapy, serving to further knock down testosterone levels by blocking a key enzyme in the androgen synthesis process. Launched in the United States in April 2011 and with a current run-rate of over $2.2 billion in worldwide sales, Zytiga had the top oral oncology drug launch in history.

  Agents that directly target the AR are termed anti-androgens. Anti-androgens bind to AR thereby blocking the ability of androgens to do so; this reduces subsequent AR translocation into the nucleus and AR-dependent gene transcription. The first-generation anti-androgen is Casodex; Xtandi is a second-generation anti-androgen that has much improved efficacy and safety profile over Casodex, and we believe Xtandi will start to replace Casodex in the near future. We note that Casodex is the most-prescribed drug for prostate cancer with 600,000 scripts annually in the United States. Xtandi was launched in the United States in September 2012 and booked second quarter 2014 sales of $227 million for a run-rate of more than $900 million. We expect Xtandi to achieve worldwide sales of $1.6 billion in 2015.

- ***We believe that targeting the AR axis will continue to be the cornerstone of prostate cancer treatment, and we project Xtandi, galeterone, and other agents targeting the AR axis to take one-half of the future prostate cancer market.*** In our prostate cancer market model (as illustrated in exhibit 11, on page 22), we have the new hormonal agents that target the AR axis, including Xtandi and galeterone, projected at one-half of the total prostate cancer market that peaks at $17 billion in 2025-2030.

A Three-Pronged Attack May Position Galeterone as Best-in-Class Agent Targeting the AR Axis
Galeterone (formerly known as TOK-001) is a first-in-class, oral small molecule drug that encompasses the mechanisms of action of both Zytiga and Xtandi, and goes one step beyond to actively degrade the AR. This last characteristic leads to galeterone's effectiveness in a subset of patients who do not respond to Zytiga or Xtandi.

- ***Similar to Zytiga, galeterone inhibits androgen synthesis.*** CYP17 is a key enzyme that plays multiple roles during the androgen production process. Galeterone functions by specifically blocking CYP17 lyase to reduce androgen synthesis.

- ***Like Xtandi, galeterone competitively binds to AR to block androgen-mediated signaling.*** Galeterone blocks androgen-AR interaction and thus inhibits androgen-mediated signaling. This reduces subsequent AR translocation into the nucleus and AR-dependent gene transcription, without which the downstream effects associated with prostate cell growth, proliferation, and survival cannot occur.

- ***Galeterone goes one step further to degrade AR so as to overcome certain resistance to Zytiga and Xtandi.*** A number of resistant point mutations and splice variants in the AR have been identified, which Zytiga and/or Xtandi have little or no activity against. Preclinical studies have shown that galeterone treatment leads to degradation of wild-type as well as Zytiga or Xtandi-resistant AR, demonstrating a unique mechanism to overcome this category of resistance. Currently, no agents on the market or in development can degrade AR, making galeterone a first-in-class therapeutic.

Galeterone's Advantages Over Zytiga and Xtandi

Based on its differentiated mechanisms of action, superior resistance profile and potentially satisfactory safety profile, galeterone could become a best-in-class treatment targeting the AR axis, in our opinion.

- ***Galeterone more selectively inhibits the CYP17 enzyme in the testosterone synthesis pathway as compared with Zytiga, eliminating the need for prednisone co-administration and the requirement of concomitant food intake.*** Zytiga is the only therapy on the market that inhibits CYP17, an enzyme that plays key roles in androgen production and possesses two enzymatic functions: hydroxylase and lyase. Zytiga is a more-specific inhibitor of the CYP17 hydroxylase, which leads to accumulation of a number of steroids such as progesterone, deoxycorticosterone and corticosterone, and a reduction in cortisol, which results in mineralcorticoid excess (ME), a syndrome characterized by hypertension, hypokalemia, fluid retention, and edema. As a result, Zytiga has to be co-administered with prednisone to mitigate these side effects; despite the use of prednisone, 30% of patients still develop some level of ME. In contrast, galeterone specifically inhibits CYP17 lyase activity, which does not lead to ME, negating the need for concomitant prednisone therapy.

  In addition, Zytiga should be taken on an empty stomach and must be followed by an additional hour of fasting to avoid over-absorption and toxicity. Galeterone was specifically formulated to avoid this inconvenience and consequently does not have a food effect.

- ***Galeterone is also an AR antagonist with similar potency as Xtandi, but does not have seizure potential.*** Galeterone has similar potency as Xtandi in vitro. On the safety side, it does not have any theoretical seizure potential, as it does not interact with the $GABA_A$ receptor in the brain, which is the main mechanism leading to seizures. Xtandi interacts with $GABA_A$ receptors in the brain, and in clinical studies there has been a low incidence of seizures observed—0.9% in the post-chemo mCRPC setting and 0.1% in the chemo-naïve mCRPC setting. Since the seizure incidence is very low, it has not become a problem for Xtandi's adoption in the marketplace. By eliminating the theoretical risk of seizures, galeterone will likely have 0% seizure rate, a potential advantage.

- ***Galeterone has been shown to be effective in a subset of patients with AR mutations or splice variants who are unresponsive to both Zytiga and Xtandi, owing to its ability to actively degrade AR.*** Both Zytiga and Xtandi require the binding of the androgen to the AR to accomplish their anti-oncogenic effects. However, certain point mutations and splice variants of AR have mutated or truncated ligand-binding domain, thereby acting in a constitutively active way without the need of androgen binding. For example, the T878A point mutation in the AR allows for progesterone to bind and activate AR, giving rise to Zytiga resistance. In addition, the

F876L mutation, which changes antagonistic binding to agonist signaling, was recently linked to Xtandi resistance. The most-common AR splice variant is AR-V7, which lacks the ligand binding domain and remains constitutively active; cells harboring this splice variant are therefore both Zytiga-resistant and Xtandi-resistant.

In contrast, galeterone's mechanism of action does not solely depend on disrupting receptor-ligand interaction; galeterone can degrade AR by targeting the N-terminus and DNA binding domain, which remain well-conserved throughout the various mutated and spliced forms of AR. By degrading not only the wild-type but also the mutated forms of AR, galeterone can overcome the above-mentioned resistance to Zytiga and Xtandi, paving the way for a best-in-class treatment.

Exhibit 1
Tokai Pharmaceuticals, Inc.
Mechanisms of Action

	CYP17 Lyase Inhibitor	AR Antagonist	AR Degrader
	 Inhibits androgen synthesis	 Blocks androgen binding	 Decreases AR levels
Zytiga $1.7B 2013 sales	✓		
Xtandi $445M 2013 sales *ARN509 / ODM-201*		✓	
GALETERONE Differentiated Selective Multi-targeted	✓	✓	✓
	• *No mandatory steroids* • *Fasting not required* • *Preclinical activity in mutation T878A*	• *Not a $GABA_A$ antagonist* • *No seizures* • *Preclinical activity in mutation F876L*	• *Blocks refractory mechanisms* • *Active in C-terminal loss AR splice variants*

Source: Tokai Pharmaceuticals, Inc.

An Efficient Clinical Development Strategy: Targeting the AR-V7 population

- ***Galeterone activity observed in C-terminal loss patients to date forms the basis of the Phase III design.*** To date, seven chemo-naïve mCRPC (or M1) patients in galeterone Phase II studies have been identified as having C-terminal loss in their AR expression, and six of them responded to galeterone with a maximal PSA decline of at least 50% (PSA50 response). The seventh patient who did not demonstrate a PSA decline dropped out of the study, due to an adverse event unrelated to galeterone after six weeks of treatment. We note that in such patients with C-terminal loss, Zytiga or Xtandi would demonstrate no PSA response.

- ***C-terminal loss and AR-V7 splice variants: current unmet need presents lower-hanging fruit and opportunity for fast track to approval.*** As galeterone can target and degrade AR with C-terminal loss that Zytiga or Xtandi does not have activity against, it can address an important unmet medical need. The dominant form of C-terminal loss of the AR is the AR-V7 splice variant. It is unclear what the percentage is of patients at the chemo-naïve mCRPC setting who harbor the AR-V7 variant; literature suggests a range from 12% to over 30%. We assume 20% in our model.

- ***Phase III ARMOR3-SV is designed to target chemo-naïve mCRPC patients who harbor the AR-V7 splice variant.*** Tokai has reached an agreement with the FDA on the design of a single pivotal study that targets chemo-naïve mCRPC (or M1) patients who harbor the AR-V7 splice variant. To conduct such a trial, Tokai needs to first develop a companion diagnostic test to screen patients with the AR-V7 splice variant, which should be developed, validated, and approved by the FDA by early 2015. The pivotal ARMOR3-SV study intends to enroll 170 patients who harbor the AR-V7 splice variant and randomize them to either galeterone or Xtandi. The primary endpoint is radiographic progression-free survival (rPFS), and secondary endpoints include overall survival (OS), PSA response and safety.

- ***Top-line data from ARMOR3-SV could come as early as year-end 2016; we believe Tokai has identified a highly time-efficient and capital-efficient path to approval.*** Assuming that the study is initiated in early 2015, Tokai guides top-line data release by year-end 2016, a relatively short period to conduct any Phase III oncology program, in our opinion. A new drug application (NDA) could be submitted in early 2017 and galeterone could hit the market in late 2017/early 2018, which represents a quick route to market. Furthermore, the 170-patient single pivotal study required for approval is also highly capital-efficient, as compared with most other Phase III oncology programs.

We Assign an 85% Chance of Success to the Phase III ARMOR3-SV Study

We assign the galeterone Phase III program an 85% probability of success, based on the following arguments and rationales.

- ***Targeting the AR axis is the major approach to treat prostate cancer, and galeterone has a three-pronged approach to inhibit the AR axis.*** Targeting the AR axis is the most relevant and the dominating approach to treat prostate cancer. The first-line androgen depravation therapy Lupron and the first-generation anti-androgen Casodex have been the mainstay of prostate cancer therapy for decades. Second-generation hormonal agents Zytiga and Xtandi became successful blockbusters shortly after their respective launches. We believe galeterone, which also targets the AR axis with three distinct mechanisms, should have a high probability of success.

- ***Activity of galeterone in the AR-V7 splice variants is supported by preclinical and clinical observations.*** The activity of galeterone in vitro to reduce AR levels, including wild type AR, AR with point mutations, and AR with various truncated forms including C-terminal loss and AR-V7, has been observed. Its activity in AR variants was also observed in an in vivo xenograft mouse model. Besides the preclinical evidence, clinical data to date also demonstrated that six out of seven chemo-naïve mCRPC patients with C-terminal loss have responded to galeterone.

- ***Favorable and efficient study design agreed upon with the FDA: the first mCRPC pivotal study with rPFS as primary endpoint.*** The two recently conducted Phase III pivotal studies in the chemo-naïve mCRPC (or M1) settings, COU-AA-302 for Zytiga and PREVAIL for Xtandi, enrolled more than 1,000 and 1,700 patients, respectively, and had rPFS and OS as co-primary endpoints. Both studies took three to four years from start of enrollment to top-line data. In contrast, the FDA has approved Tokai to conduct a single pivotal study for galeterone in the same chemo-naïve mCRPC setting, but specifically in the AR-V7 population and with rPFS as the primary endpoint; as a result, the target enrollment required is only 170. Such a design significantly reduces time and capital required for galeterone approval, as compared to the paths to approval taken by Zytiga and Xtandi.

- ***We believe the ARMOR3-SV study is well powered to be successful on the primary endpoint of rPFS.*** ARMOR3-SV targets to enroll 170 chemo-naïve mCRPC patients harboring the AR-V7 variant; the patients will be randomize 1:1 to galeterone and Xtandi. The study is 90% powered to demonstrate an 82% improvement in median rPFS, galeterone over Xtandi.

  We believe the powering assumption for the study is conservative. In the PREVAIL study in the chemo-naïve mCRPC (or M1) setting where Xtandi was evaluated against placebo, the PSA50 response was 78% for Xtandi and 3% for placebo, which corresponded to an rPFS of 16 months for Xtandi and only 4 months for placebo. In M1 patients with AR-V7 variants, to date we have observed 86% (six out of seven) PSA50 response for galeterone, versus 0% for Xtandi. Further, the rPFS of Xtandi in the AR-V7 population is observed at 2-3 months; in contrast, among the six AR-V7 positive patients who achieved PSA50 on 12 weeks of galeterone treatment, four have gone on to the extension phase of the study and have demonstrated rPFS of 5.2 months to greater than 9 months to date. As a result, we believe the 82% improvement in rPFS assumed for the ARMOR3-SV study is likely conservative.

- ***Development of a companion diagnostic to prospectively identify AR-V7 for study enrollment is prerequisite for the Phase III study start in early 2015, and approval is required for commercialization.*** Tokai has contracted third parties to finalize and validate an assay to prospectively identify the AR-V7 variant from patient's circulating tumor cells (CTC). Tokai needs to submit an investigational device exemption application (IDE) for the assay to the FDA before start of the Phase III study. This companion diagnostic test will need to be approved by the FDA through its premarket approval or PMA process before commercialization. During the Phase III study, the AR-V7 identification will be conducted by a central laboratory. Tokai expects to screen 1,000 chemo-naïve mCRPC (or M1) patients to identify the target enrollment of 170 patients positive for AR-V7, assuming an approximate 20% prevalence.

Worldwide Commercialization Strategy
Tokai holds the worldwide commercialization rights to galeterone and intends to build a urology- and oncology-focused sales-and-marketing organization in the United States. Tokai intends to partner with other organizations to commercialize galeterone outside the United States.

We assume 100% ownership of galeterone by Tokai in the United States and that Tokai will receive 25% royalties on sales in Europe. Sales in other geographical territories are upside to our model.

Revenue Model for Galeterone in the United States and Europe
Exhibit 2, on the following page, includes our galeterone revenue model in the United States and Europe. We assume 20% of chemo-naïve mCRPC patients harbor the AR-V7 variant.

- ***Targeted patient population should ensure high penetration.*** We assume peak penetration for galeterone in the AR-V7 population at 90%.

- ***Pricing and reimbursement.*** We estimate a 2018 launch net price for galeterone at $90,000 in the United States, after a gross-to-net discount rate of 13%. Such pricing is at a slight premium of 5% to our Xtandi net price assumption in 2018. In addition, we forecast an annual 2% price increase in the United States. In Europe, we model galeterone launch pricing at 70% of that in the United States and 0% price increase going forward. We believe such assumptions might be conservative, as galeterone could be priced at a higher premium to Xtandi, due to its capability of overcoming Xtandi resistance and addressing an unmet need.

- ***Our revenue projection.*** We derive peak sales of galeterone of $1.8 billion in 2027, with $1.08 billion in the United States and $760 million in Europe.

Exhibit 2
Tokai Pharmaceuticals, Inc.
William Blair Revenue Model for Agents in M1 Setting, United States and Europe
(dollars in thousands)

		2014	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024	2025	2026	2027
Total # of U.S. patients w/ mCRPC		121.9	126.9	132.0	137.4	142.9	148.7	154.8	161.1	167.6	174.4	181.5	188.9	196.5	204.5
US - Chemo-naive mCRPC															
# of patients eligible, seeking treatment		37.3	38.8	40.4	42.0	43.7	45.5	47.4	49.3	51.3	53.4	55.5	57.8	60.1	62.6
Total Patients Treated %		43%	67%	82%	89%	97%	114%	131%	137%	141%	145%	147%	146%	145%	144%
Zytiga (abiraterone) from JNJ		33.8%	28.5%	26.8%	25.4%	23.9%	15.5%	7.8%	1.9%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Abiraterone (generic)		0.0%	0.0%	0.0%	0.0%	0.0%	10.9%	23.5%	28.6%	33.2%	37.1%	40.0%	42.9%	45.9%	48.8%
Xtandi (enzalutamide) from MDVN / Astellas		8.9%	37.5%	49.3%	51.7%	54.7%	57.6%	58.5%	58.5%	56.6%	53.6%	50.7%	47.8%	44.9%	41.9%
Galeterone from Tokai		0.0%	0.0%	0.0%	0.0%	2.0%	6.5%	11.2%	14.7%	16.0%	17.2%	17.6%	17.6%	17.6%	17.6%
ARN-509 from JNJ -or- ODM-201 from Bayer		0.0%	0.0%	0.0%	0.0%	2.0%	7.1%	11.1%	11.7%	11.7%	11.7%	11.7%	11.7%	11.7%	11.7%
US - Chemo-naive mCRPC, list price per quarter															
Zytiga (abiraterone) from JNJ	19,500	17,650	18,003	18,363	18,731	19,105	19,487	19,877	20,076	20,277	20,480	20,684	20,891	21,100	21,311
Abiraterone (generic)	2,500	2,308	2,354	2,401	2,449	2,498	2,548	2,599	2,625	2,652	2,678	2,705	2,732	2,759	2,787
Xtandi (enzalutamide) from MDVN / Astellas	22,350	19,605	19,998	20,397	20,805	21,222	21,646	22,079	22,300	22,523	22,748	22,975	23,205	23,437	23,672
Galeterone from Tokai	22,350	-	-	-	-	22,336	22,782	23,238	23,470	23,705	23,942	24,182	24,423	24,668	24,914
ARN-509 from JNJ -or- ODM-201 from Bayer	18,750	17,311	17,657	18,010	18,371	18,738	19,113	19,495	19,690	19,887	20,086	20,287	20,489	20,694	20,901
US - Chemo-naive mCRPC, sales	Peak Sales														
Zytiga (abiraterone) from JNJ	800,729	852,174	831,380	796,300	799,801	800,729	646,583	359,448	140,390	9,604	(0)	(0)	(0)	(0)	(0)
Abiraterone (generic)	329,597	-	-	-	-	-	28,484	95,236	138,590	169,875	203,516	231,918	261,959	294,461	329,597
Xtandi (enzalutamide) from MDVN / Astellas	2,608,092	111,554	809,009	1,538,288	1,757,608	1,968,329	2,202,854	2,407,502	2,535,384	2,608,092	2,606,705	2,592,498	2,570,058	2,538,584	2,497,212
Galeterone from Tokai	1,079,015	-	-	-	-	38,501	193,348	408,559	620,182	750,790	848,041	926,928	976,818	1,026,645	1,079,015
ARN-509 from JNJ -or- ODM-201 from Bayer	603,476	-	-	-	-	32,299	172,507	361,733	445,940	470,571	494,575	519,803	546,319	574,186	603,476
Total # of EU patients w/ mCRPC		145	151	157	164	170	177	184	192	200	208	216	225	234	243
EU - Chemo-naive mCRPC															
# of patients eligible, seeking treatment		44	46	48	50	52	54	56	59	61	64	66	69	72	75
Total Patients Treated %		30%	42%	61%	80%	92%	105%	112%	123%	130%	130%	127%	125%	122%	117%
Zytiga (abiraterone) from JNJ		27.9%	34.4%	34.1%	32.7%	31.2%	23.8%	4.8%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Abiraterone (generic)		0.0%	0.0%	0.0%	0.0%	0.0%	9.9%	27.4%	35.4%	37.5%	40.0%	41.0%	42.9%	43.9%	43.9%
Xtandi (enzalutamide) from MDVN / Astellas		2.1%	7.7%	26.5%	42.0%	45.9%	48.8%	51.7%	54.7%	57.6%	54.6%	50.7%	47.8%	44.9%	41.9%
Galeterone from Tokai		0.0%	0.0%	0.0%	0.0%	2.0%	6.5%	11.2%	15.6%	17.6%	17.6%	17.6%	17.2%	16.2%	15.0%
ARN-509 from JNJ -or- ODM-201 from Bayer		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%
EU - Chemo-naive mCRPC, list price per quarter															
Zytiga (abiraterone) from JNJ	14,625	13,455	13,455	13,455	13,455	13,455	13,455	13,455	13,455	13,455	13,455	13,455	13,455	13,455	13,455
Abiraterone (generic)	1,875	1,725	1,725	1,725	1,725	1,725	1,725	1,725	1,725	1,725	1,725	1,725	1,725	1,725	1,725
Xtandi (enzalutamide) from MDVN / Astellas	15,645	14,393	14,393	14,393	14,393	14,393	14,393	14,393	14,393	14,393	14,393	14,393	14,393	14,393	14,393
Galeterone from Tokai	16,721	-	-	-	-	15,383	15,383	15,383	15,383	15,383	15,383	15,383	15,383	15,383	15,383
ARN-509 from JNJ -or- ODM-201 from Bayer	14,063	12,938	12,938	12,938	12,938	12,938	12,938	12,938	12,938	12,938	12,938	12,938	12,938	12,938	12,938
EU - Chemo-naive mCRPC, sales	Peak Sales														
Zytiga (abiraterone) from JNJ	878,165	540,539	806,506	878,165	878,103	873,352	794,582	338,253	14,581	(0)	(0)	(0)	(0)	(0)	(0)
Abiraterone (generic)	222,346	-	-	-	-	-	18,418	81,096	132,586	153,540	169,407	183,639	197,461	213,100	222,346
Xtandi (enzalutamide) from MDVN / Astellas	2,015,725	32,473	124,398	504,870	1,063,287	1,329,960	1,473,917	1,627,373	1,790,859	1,964,940	2,015,725	1,937,875	1,897,768	1,855,967	1,807,643
Galeterone from Tokai	759,511	-	-	-	-	31,589	155,524	322,018	499,969	632,907	676,850	704,333	722,549	759,511	735,400
ARN-509 from JNJ -or- ODM-201 from Bayer	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Total Worldwide Sales in M1 Setting	Peak Sales														
Zytiga (abiraterone) from JNJ	1,677,904	1,392,713	1,637,886	1,674,465	1,677,904	1,674,081	1,441,165	697,701	154,972	9,604	(0)	(0)	(0)	(0)	(0)
Abiraterone (generic)	551,943	-	-	-	-	-	46,902	176,333	271,176	323,415	372,923	415,557	459,420	507,561	551,943
Xtandi (enzalutamide) from MDVN / Astellas	4,622,430	144,027	933,406	2,043,158	2,820,895	3,298,289	3,676,771	4,034,875	4,326,243	4,573,032	4,622,430	4,530,372	4,467,826	4,394,552	4,304,855
Galeterone from Tokai	1,814,414	-	-	-	-	70,089	348,872	730,576	1,120,151	1,383,696	1,524,891	1,631,261	1,699,367	1,786,156	1,814,414
ARN-509 from JNJ -or- ODM-201 from Bayer	603,476	-	-	-	-	32,299	172,507	361,733	445,940	470,571	494,575	519,803	546,319	574,186	603,476

Sources: William Blair & Company, L.L.C. estimates

Valuation: 12-Month Price Target of $44

In building a probability-adjusted NPV model, we estimate the peak sales of a given drug candidate, its probability of advancing to the next stage of development and eventually reaching the market, and a company's share of revenue and expenses depending on the commercialization plan and/or structure of partnerships, if any. We then calculate the cash flows after adjusting all revenues and expenses with respective cumulative probabilities for each stage. The cash flows are then discounted back using an industry-specific weighted average cost of capital (WACC) of 12% to arrive at a probability-adjusted NPV for each drug candidate. Once we determine the NPV for each candidate, we add net cash and other costs, which include expenses not directly associated with the development of the clinical candidates, to arrive at a fair value estimate for a stock.

In exhibit 3, we summarize our sum-of-the-parts valuation for Tokai Pharmaceuticals. As discussed above, we assign an 85% probability for galeterone to reach the market and estimate peak sales for galeterone in the United States and Europe at $1.8 billion. We value galeterone at about $42 per share. Adding the net cash at the end of 2015 and subtracting net present value of estimated expenses not directly associated with galeterone development, we derive our price target of $44.

Exhibit 3
Tokai Pharmaceuticals, Inc.
Sum-of-the-Parts Fair Value
(dollars in thousands, except shares)

Drug	Peak Sales	Stage of Development	Estimated Launch Date	Probability of Commercialization	Percentage of Sales to Company	Probability-Adjusted NPV	Value per Share	Percentage of Fair Value
Galeterone	$ 1,814,414	Pre-Phase III	H1:2018	85%	100% US; 25% ex-US	$936,181	$42.36	95.6%
Subtotal						**$936,181**	**$42.36**	**95.6%**
Net Cash at year-end 2015						$51,424	$2.33	5.2%
Net Present Value of additional Gain (Loss)*						($8,000)	($0.36)	(0.8%)
Sum-of-Parts Fair Value						**$979,605**	**$44.33**	**100.0%**

Sources: William Blair & Company, L.L.C. estimates

Intellectual Property for Galeterone

In 2006, Tokai entered into an exclusive master licensing agreement with the University of Maryland, Baltimore for the exclusive worldwide rights to make, use, and sell galeterone and related compounds.

The composition of matter patent covering galeterone is set to expire in 2017. The issued method-of-use patent goes to 2027, with potential extension of up to five years under Hatch-Waxman. The pending formulation patent could also extend the patent protection to 2032.

Large Commercial Potential with Considerable Upside to Our Estimates and Valuation

- ***Five new prostate cancer therapies approved in the past four years are expected to generate estimated worldwide sales of nearly $4 billion in 2014. We project novel prostate cancer agents to reach $17 billion in worldwide sales in 2025-2030.*** Before 2010, only Taxotere had demonstrated survival advantage in prostate cancer. From 2010 to 2014, five new products have demonstrated significant OS benefit, and we estimate worldwide sales of the newly approved therapies—Zytiga, Xtandi, Jevtana, Xofigo, and Provenge—at nearly $4 billion in 2014. We expect the sales for all novel agents in prostate cancer to reach nearly $17 billion in 2025-2030 (see exhibit 11, on page 22).

- ***Galeterone's first indication targets about 20% of chemo-naïve mCRPC patients, fulfilling an unmet need in this setting. We project $1.8 billion in peak sales for this indication alone.*** In the chemo-naïve mCRPC (or M1) setting, there are an estimated 50,000-70,000 patients

in the United States and more in Europe. Currently, the Provenge immunotherapy and the second-generation hormonal agents Zytiga and Xtandi are approved in this setting. Galeterone is targeting 20% of the patients in this setting who do not respond to Zytiga or Xtandi. Zytiga, Xtandi, or galeterone could be used in combination or in sequence with Provenge. We estimate galeterone to achieve sales of $1.8 billion in 2027 in the AR-V7 indication alone.

- ***Significant upside to this estimate exists should galeterone manage to be approved earlier in the treatment continuum or gain more market share from the other agents than we project.*** Potential sources of major upside to our revenue estimate include going into earlier urology settings in prostate cancer, combination with currently approved drugs such as Xofigo, and opportunity in treating a subset of patients who failed Zytiga or Xtandi.

- ***A blue-sky scenario could be that galeterone, based on its superior resistance profile and comparable safety profile, will challenge Xtandi's foundational role in prostate cancer. We currently estimate peak sales for Xtandi at $7.6 billion.*** We believe that Xtandi is poised to start replacing Casodex, the most-prescribed prostate cancer drug with 600,000 annual scripts written in the United States. We believe that Xtandi could become a foundational therapy for prostate cancer based on its strong efficacy and benign safety profile. We project peak sales for Xtandi at $7.6 billion. Given its superior resistance profile, galeterone could have an advantage over Xtandi in the early settings, in our opinion, if galeterone demonstrates a comparable safety profile. Launching in early 2018, more than five years after Xtandi, galeterone could further move into earlier settings to challenge Xtandi's dominating position in the market, rivaling Xtandi as the next standard of care for prostate cancer.

- ***Geographic expansion is another source of upside to our estimates.*** Our estimates only take into account the first indication for galeterone and only in the United States and Europe. Geographic expansion of sales by partners would generate further upside to our estimates.

- ***AR degradation platform could produce more candidates in the pipeline. Tokai in-licensed galeterone as well as the AR degradation platform from the University of Maryland, Baltimore.*** These compounds could have improved properties over galeterone in AR inhibition and degradation and could generate value in the long term.

Key Catalysts Driving Value in the Next 12-24 Months

In exhibit 4, we summarize key upcoming events for Tokai; highlighted rows indicate potential stock-moving catalysts. We believe a number of catalysts will drive value in the next 12-24 months, including: 1) further data presentation of galeterone activity in AR mutations and variants at the European Organisation for Research and Treatment of Cancer (EORTC) symposium (Barcelona, November 18-21); 2) initiation of the pivotal ARMOR3-SV study in chemo-naïve mCRPC patients with AR-V7 in early 2015; and 3) top-line data from ARMOR3-SV by the end of 2016.

Exhibit 4
Tokai Pharmaceuticals, Inc.
Key Events

Date	Event
Q1:12	
Q2:12	Positive ARMOR1 data presented at ASCO; fast track designation received from the FDA
Q3:12	Begin ARMOR2 part 1
↓ **Q1:14**	ARMOR2 part 1 results presented at ASCO-GU
Q2:14	Amend ARMOR2 protocol to include AR mutation screeing, combination with Xofigo, and use in late-stage, post-chemo cohorts
Q3:14	Further ARMOR2 updates; pre-Phase III meeting with the FDA
Q4:14	Clinical AR mutation data analysis; additional preclinical data with novel and approved therapies
Q1:15	Companion diagnostic validated; Phase III ARMOR3-SV study initiation
↓ **Q4:16**	Top-line data from ARMOR3-SV study
H1:17	NDA submission

Sources: Company reports and William Blair & Company, L.L.C. estimates

Experienced Management Team

Tokai Pharmaceuticals is led by an experienced management team, whose executives have successful track records. In exhibit 5, we summarize the key experience of the top executives.

Exhibit 5
Tokai Pharmaceuticals, Inc.
Management Team

Management	Position	Previous Experience
Jodie Morrison	President and Chief Executive Officer	Former COO and head of clinical affairs; former director of clinical operations and medical affairs at Dyax Corporation; clinical management at Curis, Inc. and Diacrin, Inc.
John McBride	Chief Operating Officer	Founded and served as president of Alliance Life Science Advisors, Inc.; executive vice president and COO of Gloucester Pharmaceuticals, Inc.; global head of oncology licensing at Pharmacia Corporation; executive vice president of business operations and CFO at CytoTherapeutics, Inc.; vice president of business development and Treasurer at Phytera, Inc.; vice president of commercial development at Sparta Pharmaceuticals, Inc.; vice president of business development at U.S. Bioscience, Inc.
Lee Kalowski, MBA	Chief Financial Officer	Former vice president of Global Biotechnology Equity Research at Credit Suisse; prior roles at Johnson & Johnson, Sanford C. Bernstein, and Prudential Equity Group; Involved in healthcare equity research including companies with prostate cancer therapeutics.
Karen Ferrante, M.D.	Head of R&D and Chief Medical Officer	Oncology therapeutic area head for Takeda Pharmaceuticals Takeda Cambridge, USA site head; CMO and head of R&D for Millennium, The Takeda Oncology Company; vice president and therapeutic area clinical leader in oncology development at Pfizer Global R&D; associate director of clinical oncology at Bristol-Myers Squibb Company.
Susan Stewart	Head of Regulatory Affairs, Quality and Compliance	Vice president of regulatory affairs at TransMolecular, Inc.; vice president of regulatory affairs for Genzyme Corporation; QA supervisor for Abbott Laboratories.
Douglas Jacoby, Ph.D.	Head of Research	Lead scientist at TransMolecular and Diacrin.
Khalid Mamlouk, Pharm.D.	Vice President, Head of Medical Affairs	Head of medical affairs at Algeta; senior director of scientific relations and marketing at Dendreon Corporation; senior MSL in the Medical Affairs Group at Genentech Bio-Oncology.
Cindy Driscoll	Head of Finance and Administration	Finance and operations leadership positions at TransMolecular, Inc. and Gloucester Pharmaceuticals; corporate controller positions with software companies including m-Qube, Channelwave and Novasoft.

Sources: Tokai Pharmaceuticals, Inc.

Key Risks to Our Outperform Rating and Price Target

Key risks to our Outperform rating and price target include: 1) clinical risk of the Phase III program; 2) regulatory risk related to receiving approval for galeterone in the United States and Europe; 3) development and approval of the companion diagnostic; 4) reimbursement risk; and 5) financing risk.

Prostate Cancer and Its Treatment: An Overview

Prostate Cancer Is the Most Commonly Diagnosed Cancer in Men in the United States

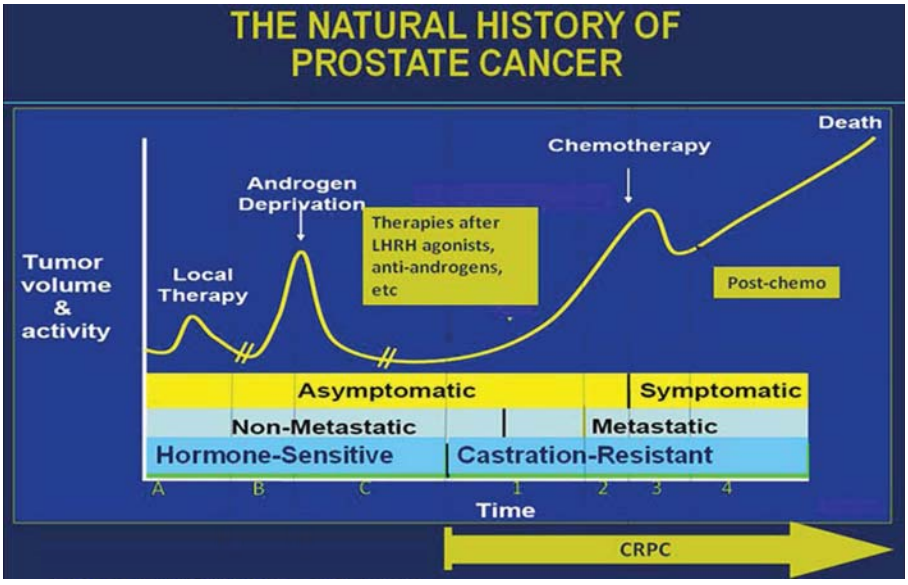
Epidemiology. According to the National Cancer Institute (NCI), nearly 2.5 million men in the United States are living with prostate cancer, with about 215,000 new cases diagnosed each year. About one in six men will be diagnosed with prostate cancer in their lifetime. Of the newly diagnosed cases, about 80% are found localized to the prostate and relatively benign, whereas 20% are advanced, including 5% that are already metastatic. Localized prostate cancer, if properly treated, has a five-year survival rate of close to 100%; however, 20%-30% of patients eventually relapse after local therapy. When metastatic, the disease invariably becomes fatal, with a five-year survival rate of about 30%. In 2014, it is expected that prostate cancer will claim the lives of nearly 30,000 men in the United States.

Detection and staging. Prostate cancer is detected during a medical examination and/or during PSA (prostate-specific antigen) screening. The majority of patients are asymptomatic at the time of diagnosis; symptoms are usually associated with a more-advanced stage of the disease. Prostate cancer is staged from I to IV, with stages I and II classified as localized disease, stage III as locally advanced, and stage IV as metastatic disease (when cancer has already spread to other tissues such as the lymph nodes, bones, and visceral organs, such as liver and lung).

When prostate cancer is suspected, a biopsy is performed and a Gleason score read by a pathologist. The Gleason system is used to grade prostate tumors from 2 to 10, where 10 indicates the most abnormalities.

Natural history. The prostate cancer disease-spectrum can be described in a series of states, as illustrated in exhibit 6, defined by the presence or absence of detectable metastases, whether testosterone levels are in the castrate or noncastrate range, and whether the patient is asymptomatic or symptomatic. Each state represents a clinically significant milestone for disease progression and choice of therapies.

Exhibit 6
Tokai Pharmaceuticals, Inc.
Natural History of Prostate Cancer



Source: Drake C., "Novel Immune-based Therapies for Castrate-resistant Prostate Cancer: Implications for Patients and Practices," 2010, Medscape

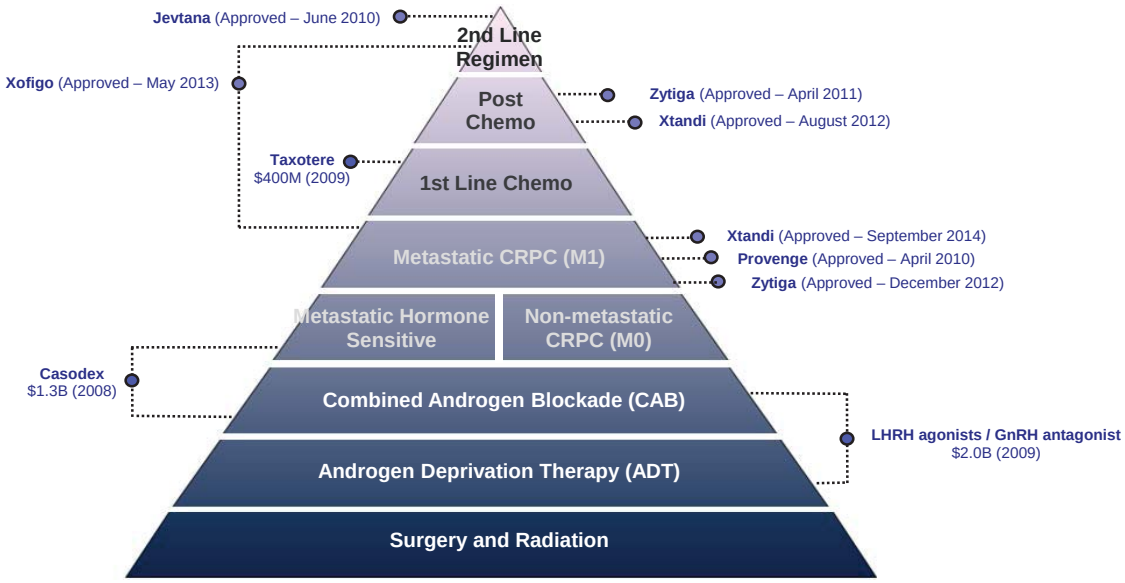
Six Drugs Have Demonstrated OS Benefit in Prostate Cancer, With Five Approvals in the Past Four Years

Until 2010, Taxotere (docetaxel, Sanofi) was the only agent that demonstrated OS benefit in prostate cancer. It is remarkable that in the past four years, five agents gained approval by demonstrating OS benefit in metastatic castration-resistant prostate cancer (mCRPC): Provenge (sipuleucel-T, Dendreon) for chemo-naïve setting; Jevtana (cabazitaxel, Sanofi) for the second-line chemo setting; Zytiga (abiraterone acetate, Johnson & Johnson) and Xtandi (enzalutamide, Medivation and Astellas) for both chemo-naïve and post-chemo settings; and Xofigo (Alpharadin, or radium-223 chloride, Bayer) for mCRPC patients with symptomatic bone metastases.

Current Treatment Sequence for Prostate Cancer

We summarize the current treatment sequence for prostate cancer in exhibit 7. After local therapy, hormonal agents such as LHRH (luteinizing hormone-releasing hormone) agonists and Casodex are used to inhibit the AR pathway. If the disease progresses, secondary hormonal treatments such as ketoconazole and estrogen are used off-label to further stifle the AR pathway. In a subset of patients, the disease becomes refractory and progresses to the metastatic stage (mCRPC); at present, Provenge, Zytiga, and Xtandi are approved in the chemo-naïve mCRPC setting, or the M1 setting. Once patients progress further, they are administered Taxotere, the first-line chemotherapy. Failing Taxotere, patients could go on to Zytiga or Xtandi if they were not exposed to these agents before chemotherapy, followed by second-line chemotherapy Jevtana, if they are fit enough. Xofigo was recently approved for mCRPC patients with symptomatic bone metastases.

Exhibit 7
Tokai Pharmaceuticals, Inc.
Prostate Cancer: Current Treatment Paradigm



Source: William Blair & Company, L.L.C.

Comparing Launches of the Five Recent Approvals: Provenge, Jevtana, Zytiga, Xtandi, and Xofigo
We illustrate in exhibit 8, on the following page, quarterly U.S. sales of the five latest approved drugs in mCRPC for since their launches.

Provenge was launched in April 2010 in the United States as the first-ever immunotherapy to demonstrate OS benefit in oncology. Despite this exciting scientific achievement, Provenge sales since launch have fallen short of expectations. Sales improved in second quarter 2014 to about $82 million; however, Provenge and its developer, Dendreon, face serious difficulties, including: stiff competition from other agents both available and in development, high cost of goods sold due to an involved manufacturing process, and onerous debt burden that brings into question Dendreon's viability as a company.

After its approval in June 2010, Jevtana sales started strongly in patients who failed the first-line chemo therapy Taxotere. However, the use of Zytiga (after its April 2011 launch) has depressed Jevtana sales, as Zytiga has been sequenced in front of Jevtana in patients who failed Taxotere and created a "delay-to-peak" effect for Jevtana. As illustrated in exhibit 8, Zytiga has eaten into Jevtana's sales significantly.

Zytiga has become the best oral oncology drug launch in history. It was launched in the United States in April 2011 and in Europe in the fall of 2011, after receiving approval in the post-chemo mCRPC setting. The chemo-naïve mCRPC approval came in December 2012 in the United States and Europe. As of July 2014, Zytiga has been approved in more than 90 countries for the post-chemo setting and more than 40 countries in the chemo-naïve M1 setting. Zytiga generated $966 million in revenue in 2012, the first full year on the market, with $463 million in the United States. Zytiga revenue for second quarter 2014 was $562 million worldwide, representing an annual run-rate of $2.2 billion.

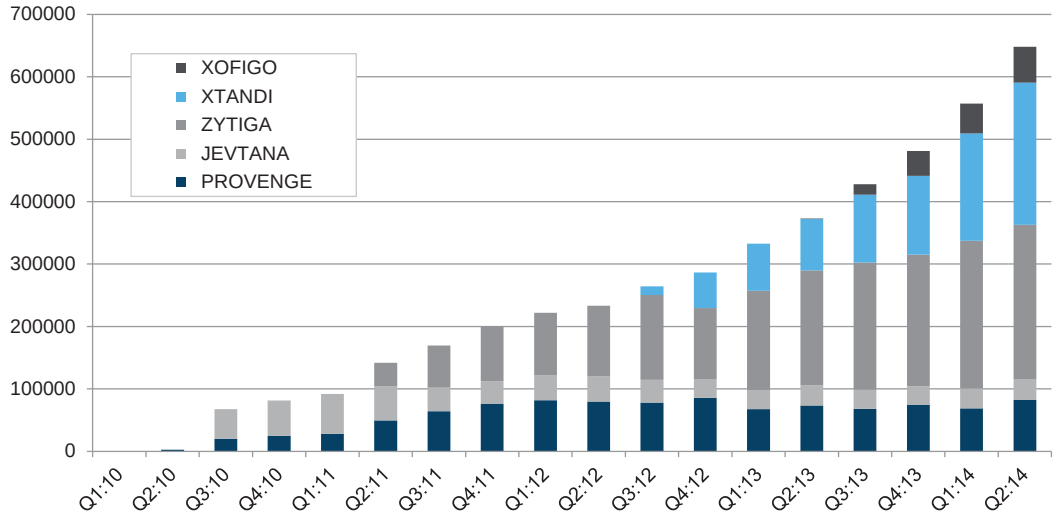
Xtandi was approved in the United States for the post-chemo CRPC setting in August 2012 and launched in September 2012. As the second drug following Zytiga in the same indication, total prescriptions (TRx) of Xtandi as of August 2014 are tracking at about 85% of Zytiga's at the corresponding quarter during Zytiga's launch. Xtandi was approved for the chemo-naïve M1 setting in September 2014. A registration-enabling study in the non-metastatic CRPC setting, or the M0 setting, is ongoing. We expect Xtandi sales to eventually overtake Zytiga, and we believe this move will be catalyzed by Xtandi's recent approval in the M1 setting as well as the upcoming data from TERRAIN and STRIVE studies that evaluate Xtandi head-to-head against Casodex; we expect Xtandi to demonstrate superiority in efficacy to Casodex. We project Xtandi worldwide sales of $1.6 billion in 2015.

Xofigo was approved in May 2013 in the United States. It is a bone-seeking radionuclide of alpha-emitter radium-223. After intravenous administration, Xofigo travels to the lesions in the bone and kills cancer cells via radiation, which reaches 2 to 8 cells in distance. The Phase III ALSYMPCA study demonstrated significantly improved OS in mCRPC patients with multiple skeletal metastases and significant bone pain. This study provided direct proof for the first time that targeting bone metastases in prostate cancer can prolong survival. It is unclear to us how big the Xofigo opportunity could be, and the general sentiment has been that adoption might be more difficult in the United States, where radio-oncology is less integrated with medical oncology as compared with Europe. As of second quarter 2014, Xofigo's annual sales run-rate in the United States is $230 million.

Pricing. Provenge is priced at $93,000, and patients only receive one course of therapy in their lifetime. Jevtana is priced at $8,000 per three-week cycle and an average treatment course lasts six cycles, resulting in a total cost of $48,000. Zytiga was priced at $5,000 per month at launch in April 2011, and a number of price increases have been instituted; as of August 2014, the price per month is about $6,837, or about $82,000 per year. Given the treatment duration of about five to eight months in the post-chemo setting, the average cost per course of Zytiga is roughly $44,000. In the chemo-naïve setting, the treatment duration is roughly doubled at 11-16 months, leading to an

average total cost of about $90,000 per patient for Zytiga. Xtandi was priced at $7,440 per month at launch; with a treatment duration of eight months in the post-chemo setting, the total cost per patient is almost $60,000. The treatment duration in the chemo-naïve setting, based on the PREVAIL data, could be as long as 16 months, which could lead to an annual cost of up to $120,000 per patient for Xtandi. We note that Xofigo is priced at $69,000 for a course of six injections.

Exhibit 8
Tokai Pharmaceuticals, Inc.
Quarterly U.S. Sales of Recently Launched Therapies for mCRPC
(dollars in thousands)



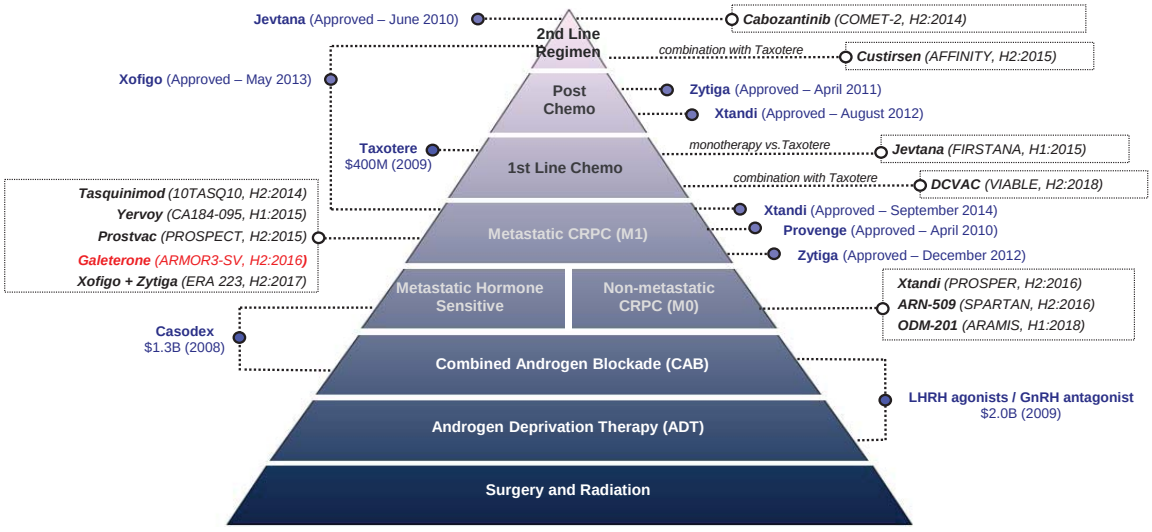
Sources: Company reports; IMS Health; and William Blair & Company L.L.C. estimates

Major Treatment Options in Phase III Development

Current Development Landscape in Prostate Cancer

As illustrated in exhibit 9, there are two major areas of concentrated clinical activities: the non-metastatic CRPC setting (M0), and the chemo-naïve front-line mCRPC setting (M1). We illustrate the Phase III agents and our estimates of the timing of their Phase III data releases in exhibit 9. Key agents that will report Phase III data in the next 3-18 months include cabozantinib (Exelixis), Yervoy (Bristol-Myers Squibb), custirsen (OncoGenex and Teva), tasquinimod (Ipsen and Active Biotech), and Prostvac (Bavarian Nordic). We believe as the data evolves and the armamentarium is enriched, the practice pattern in prostate cancer will change profoundly.

Exhibit 9
Tokai Pharmaceuticals, Inc.
Prostate Cancer Competitive Landschape



Source: William Blair & Company, L.L.C.

Targeting the AR Axis Is Foundation of Prostate Cancer Therapy

Androgen-androgen receptor axis. Prostate cancer is an androgen (mostly testosterone and dihydrotestosterone, or DHT)-dependent disease, and activation of the androgen-androgen receptor (AR) pathway is the key mechanism for prostate cancer growth at all stages of the disease. Suppressing the AR axis is therefore the foundation of prostate cancer treatment.

Inhibiting androgen (testosterone) synthesis—Zytiga. The first-line therapy to lower androgen levels includes androgen-depravation therapy such as Lupron, which reduces testosterone levels comparable to those of surgical castration. Zytiga is a second-line therapy, serving to further knock down testosterone levels by blocking a key enzyme in the androgen synthesis process. Launched in the United States in April 2011 and with a current run-rate of over $2.2 billion in worldwide sales, Zytiga had the top oral oncology drug launch in history.

Inhibiting androgen receptor—Xtandi, ARN-509, ODM-201. Agents that target the AR are termed anti-androgens. Differing from Zytiga, anti-androgens bind to AR thereby blocking the ability of androgens to do so; this reduces subsequent AR translocation into the nucleus and AR-dependent gene transcription. The first-generation anti-androgen is Casodex, while Xtandi is a second-generation anti-androgen that has much improved efficacy and safety profile over Casodex, and we believe Xtandi will start to replace Casodex starting in the near future. We note that Casodex is the most-prescribed drug for prostate cancer with 600,000 scripts annually in the United States. Xtandi was launched in the United States in September 2012, and booked second quarter 2014 sales of $227 million, for a run-rate of more than $900 million. We expect Xtandi to achieve worldwide sales of $1.6 billion in 2015.

Two follow-on AR antagonists, ARN-509 and ODM-201, have advanced into Phase III development, and could become competitors to Xtandi if they make it to the market. ARN-509, the lead candidate for Aragon (private, San Diego) was acquired by Johnson & Johnson for $650 million up front and $350 million in future milestones in 2013, while ODM-201 is being developed by Orion in collaboration with Bayer. Both share the same mechanism as Xtandi as true AR antagonists, and both are

superior in efficacy to the first-generation anti-androgen Casodex. However, based on the preclinical and clinical data to date, we believe ARN-509 and ODM-201 appear undifferentiated from Xtandi and are roughly four to five years behind Xtandi in market entry.

Currently, all three second-generation anti-androgens are in Phase III trials for use in the M0 setting: PROSPER trial for Xtandi, SPARTAN trial for ARN-509, and ARAMIS trial for ODM-201. If ARN-509 or ODM-201 were to go into the post-chemo or the chemo-naïve M1 setting, they would have to be compared against Xtandi, as Xtandi is already approved for those settings. By going into the M0 setting directly at roughly the same time as Xtandi, these agents will be compared with placebo. All three studies are using metastasis-free survival as the primary endpoint and will release top-line results in 2016-2017 timeframe.

The earliest time for the ARN-509 or ODM-201 to enter the market would be four to five years after Xtandi, by our estimation. Once on the market, these follow-on agents might need to compete on price, if there are no major differentiating features from Xtandi, in our opinion.

Galeterone is the truly differentiated next-generation AR axis inhibitor. Galeterone is a first-in-class, oral small molecule drug that encompasses the mechanisms of action for both Zytiga and Xtandi, and goes one step beyond to actively degrade the AR so as to overcome certain resistance that Zytiga and Xtandi have exhibited no activity against. Based on its differentiated mechanisms of action, superior resistance profile and potentially satisfactory safety profile, galeterone could become a best-in-class treatment targeting the AR axis, in our opinion.

Immunotherapy: Synergistic With AR Axis Inhibitors

Synergy observed between anti-androgen Xtandi and immunotherapy Provenge. The AR antagonists promote thymopoesis, or maturation of thymocytes, which differentiate into T cells. Therefore the combination of an anti-androgen and immunotherapy should theoretically be synergistic. An intriguing case that was published in 2013 documented an mCRPC patient who achieved a complete and durable PSA response treated with the combination of Xtandi and Provenge, providing anecdotal support of the combination. The patient progressed on LHRH agonists, and participated in the Phase I study of Xtandi while continuing his LHRH agonist therapy. The response on Xtandi therapy lasted 14 months before PSA rose again. The patient then received on-label Provenge while continuing on Xtandi, and six months later the PSA level declined to an undetectable level. His PSA level remained undetectable one year later; he did not have any metastatic disease, and his bone scan did not show any disease progression. As Provenge typically does not lead to PSA response, it is plausible that the combination of Xtandi and Provenge has led to such dramatic and durable response.

The dream combo could be Xtandi+Prostvac if Prostvac is successful in the pivotal PROSPECT study. The mechanism of synergy between Xtandi and Provenge theoretically should be translatable to Xtandi and Prostvac of Bavarian Nordic as well. If the Phase III study PROSPECT is successful—we might see interim analysis of PROSPECT during 2015—then Prostvac might be the better choice than Provenge for combination studies, as it is an off-the-shelf product. Further, as COGS for Prostvac is significantly lower than Provenge, it can be priced more attractively, especially when moving to the earlier stage disease setting.

The National Cancer Institute is conducting a Phase II study of Xtandi versus Xtandi+Prostvac, which should demonstrate some proof-of-concept data in 2015. Such a combination could be used in either local disease or metastatic disease, and could represent a possible regimen for longer-term remission or cure of prostate cancer, in our opinion.

Yervoy might be used in combination as well, and might be limited by its side effects. Yervoy belongs to another category of immunotherapy from Provenge and Prostvac in that it is a general immune checkpoint inhibitor instead of a disease-specific vaccine. Yervoy failed a Phase III study in the post-chemo mCRPC setting. Another Phase III study in the chemo-naïve M1 setting is ongoing, with top-line data expected in early 2015. Prostate cancer is more responsive to immunotherapy than most other cancers, and there is a good chance for the study to be successful. Combination possibilities exist for Yervoy as well; it could be combined with anti-androgens such as Xtandi or the vaccine Prostvac. However, due to its many side effects of the autoimmune nature, it is unclear how it would be adopted in the M1 setting, as patients are relatively healthy and have other options.

Chemotherapy: Might Be Used in Combo With AR Inhibitors
Taxotere and Jevtana are approved chemotherapies that demonstrated OS benefit in prostate cancer. Combination studies of chemo and Xtandi are ongoing and might achieve additive effect.

Other Pathway Inhibitors: Should Also Be Used on Top of AR Inhibitors
Agents with other mechanisms in Phase III studies include tasquinimod, an anti-angiogenesis agent, with top-line data expected around the end of 2014. We believe eventually these agents are likely to be used in combination with AR axis inhibitors as well as opposed to monotherapy.

Bone Targeted Agents: Complementary to AR Inhibitors
Xofigo targets bone metastases in mCRPC patients but does not have any effect in prostate cancer in other parts of the body, including visceral metastases. It is therefore plausible to combine Xofigo with an agent such as Zytiga, Xtandi or galeterone to broadly treat the metastasized prostate cancer.

Beyond 2016: Our View on the Future Treatment Paradigm

We illustrate our thoughts on the present and future prostate cancer treatment paradigms in exhibit 10, on page 21. We believe Xtandi could be used following local therapy to replace Casodex. Zytiga could be used as an add-on to Xtandi for a combined AR axis blockade. The use of these two agents in early-stage prostate cancer might significantly prolong the time to progression and enhance survival. Other modes of therapies such as immunotherapy could be used in combination to potentially achieve substantial disease control or even cure.

Xtandi Appears Poised to Become the Foundation of Prostate Cancer Therapy
The Xtandi development program spans the full spectrum of prostate cancer. It has been approved in the post-chemo mCRPC and the chemo-naïve M1 settings; TERRAIN and STRIVE studies comparing Xtandi against Casodex in the earlier-stage CRPC settings (both metastatic and non-metastatic) are to report data within the next three to nine months; PROSPER is underway targeting the non-metastatic CRPC or M0 population; and Phase II studies examining Xtandi in hormone-naïve patients as frontline therapy and in the neoadjuvant setting have been completed as well. The aspiration for Xtandi is be the foundation of prostate cancer therapy, starting a patient on the medication, and throughout the development of the disease adding on other therapies such as Zytiga, immunotherapy or chemotherapy, until the end-stage of the patient. Should this ultimate goal be achieved, we believe likely in 2015 and beyond, the addressable population for Xtandi would be much larger, and the treatment duration per patient would be many years. We believe in such a scenario the monthly price for Xtandi will likely need to come down; as stated above, the price of $7,440 per month might be prohibitive for adoption of Xtandi in earlier-line use. Despite potential price reductions as Xtandi reaches earlier-stage patients, the total revenue potential for Xtandi should increase due to larger patient population and much longer duration of therapy. As a result, we remain comfortable with our updated peak worldwide sales estimate of $7.6 billion.

Zytiga Should Be Relegated to Add-On Therapy to Xtandi
As Zytiga's safety profile is suboptimal and has to be taken with prednisone, it will be relegated to be used after Xtandi, in our opinion. As switching from Xtandi to Zytiga does not generate satisfactory responses, it might be best to add on Zytiga to Xtandi. The use of these two agents in early-stage prostate cancer might significantly prolong the time to progression and enhance survival.

In Parallel, Immunotherapy Should Flourish as Well
If Prostvac can demonstrate significant OS benefit in 2015-2016 from its pivotal Phase III PROSPECT study in the M1 setting and if the multiple Phase II studies that are in progress in earlier disease settings also demonstrate benefit in delay of disease progression at that time, Prostvac could be used in front of, or in combination with novel hormonal agents such as Xtandi, in our opinion. Provenge could move here as well, but it might be more challenging because of its cumbersome production and administration, as well as the fact that few Phase II studies are being conducted in earlier settings with Provenge. Similarly, Yervoy is being studied in the chemo-naïve mCRPC M1 setting as well, with top-line data expected in early 2015. If positive, it will be an interesting addition to the armamentarium as well.

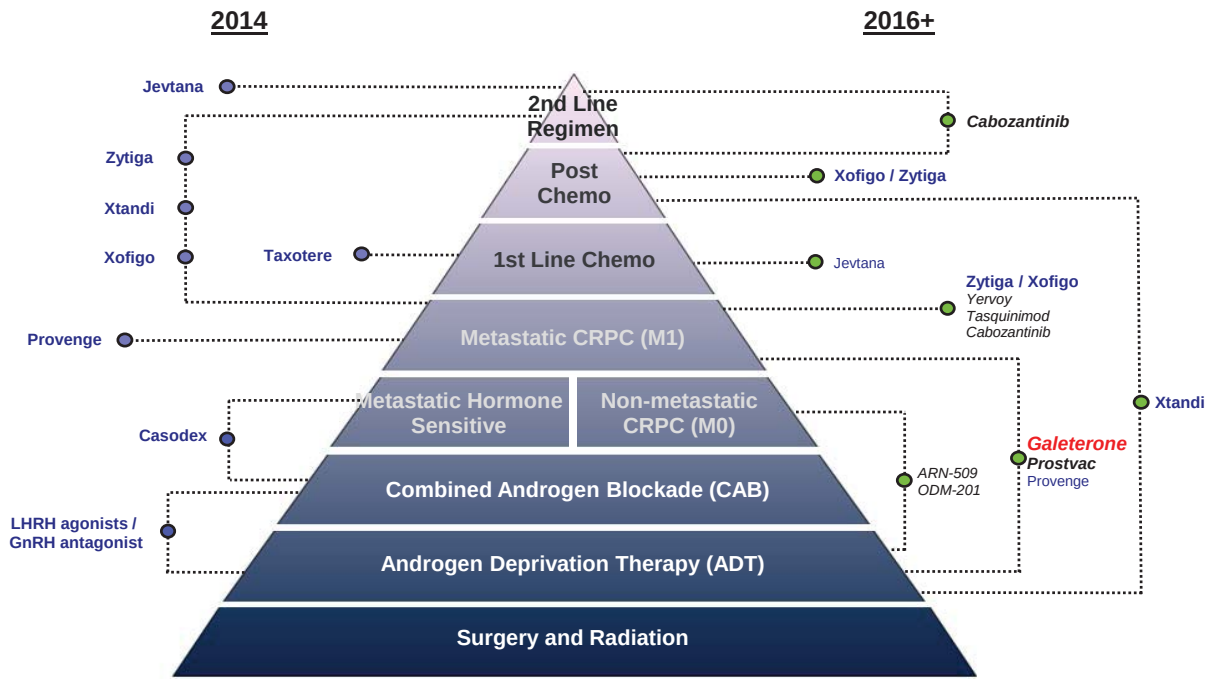
The New M1 Setting—What Options Do We Have?
After failing Xtandi, Zytiga, and/or Prostvac, patients progress to the mCRPC stage. At this point, the disease state will be different from the mCRPC seen today; it will be much more advanced, as it has failed much better hormonal agents. Xofigo in combination with other agents could be used as the next options. Xofigo specifically targets bone metastases.

At this stage of the disease, other biological pathways might overtake the AR pathway to be the main drivers to promote tumor growth. Pathways involving angiogenesis, c-Met, mTOR/PI3K, TGF-beta, integrin, and cell survival, among others, are known to be activated when the AR pathway is inhibited. We envision agents such as tasquinomod to play roles in this setting, providing alternative mechanisms of action to treat the disease with a different mechanism.

Chemotherapy Likely Becomes Last Line of Therapy
Chemotherapies such as Taxotere and Jevtana will likely become the last line of therapy for prostate cancer, in our opinion.

Galeterone Appears to Be the Only Differentiated Agent to Rival Xtandi
We believe that Xtandi soon will be poised to start replacing Casodex, the most-prescribed prostate cancer drug with 600,000 annual scripts written in the United States. We believe that Xtandi could become a foundational therapy for prostate cancer based on its strong efficacy and benign safety profile, and we project $7.6 billion in peak sales for Xtandi. A blue-sky scenario could be that galeterone, based on its superior resistance profile and potentially comparable safety profile, will challenge Xtandi's foundational role in prostate cancer. Launching in early 2018, more than five years after Xtandi, galeterone could further move into earlier settings to challenge Xtandi's dominating position in the market, rivaling Xtandi as the next standard of care for prostate cancer.

Exhibit 10
Tokai Pharmaceuticals, Inc.
William Blair View on Future Prostate Cancer Treatment Paradigm



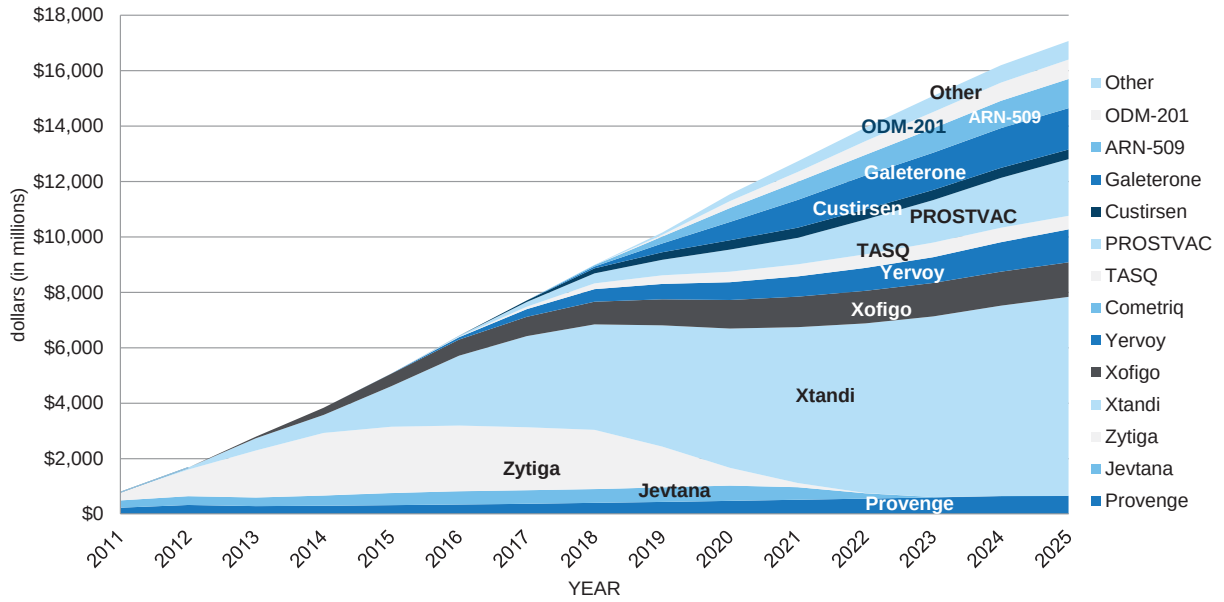
Source: William Blair & Company, L.L.C.

Prostate Cancer Market Model

We present our mCRPC market model based on the development in the prostate cancer space to date in exhibit 11, on the following page. The relative market shares from different companies and their marketed or Phase III drug candidates are based on clinical data available to date, which dictates the product profile and their relative positioning and strength in the marketplace.

We acknowledge that forecasting the market for the next 15 years based on clinical data available to date might be overly ambitious and flawed—and as a result inaccurate; however, such efforts illustrate well our thought process on the big picture. We expect to update this graph periodically, as the medicines evolve and as mergers, acquisitions, and partnership deals are struck in the space.

Exhibit 11
Tokai Pharmaceuticals, Inc.
mCRPC Market Model



Sources: William Blair & Company, L.L.C. estimates

We Project Prostate Cancer Market to Grow to $17 Billion Over the Next 10-15 Years

Taxotere was the only agent that demonstrated OS benefit in prostate cancer before 2010. It is remarkable that in the past four years, five agents gained approval by demonstrating OS benefit in mCRPC; they are Provenge (Dendreon) for the chemo-naïve setting, Jevtana (Sanofi) for the second-line chemo setting, Zytiga (Johnson & Johnson) and Xtandi (Medivation and Astellas) for both the chemo-naïve and post-chemo settings, and Xofigo (Bayer) for mCRPC patients with symptomatic bone metastases. Key agents that will report Phase III data in the next 3-18 months include cabozantinib (Exelixis), Yervoy (Bristol-Myers Squibb), tasquinimod (Ipsen and Active Biotech), custirsen (OncoGenex and Teva), and Prostvac (Bavarian Nordic). We believe as the data evolves and the armamentarium is enriched, the practice pattern in prostate cancer will change profoundly.

Agents Inhibiting AR Axis Will Likely Dominate

We continue to believe that among the myriad therapies available and in development, the AR antagonist class will dominate and become the foundation of prostate cancer treatment in the future, and we project them to occupy one-half of the future prostate cancer market due to their effectiveness, very manageable side effects, ease of use, and the spot-on mechanism of action to address the major pathway that drives prostate cancer growth. The second next-generation AR antagonist Xtandi, the follow-on compounds ARN-509 and ODM-201, as well as the next-generation galeterone constitute the market share.

Immunotherapy Could Play an Important Role

Data from the Yervoy Phase III study in the M1 setting is expected in early 2015. The Prostvac Phase III PROSPECT study, also in the M1 setting, could read out in 2015-2016. The combination of immunotherapies themselves or with anti-androgens such as Xtandi or galeterone could be the dream combination to potentially achieve longer-term remission or even a cure, in our opinion.

Galeterone Is the Truly Differentiated Next-Generation AR Axis Inhibitor

Galeterone (formerly known as TOK-001), is a first-in-class, oral small molecule drug that encompasses the mechanisms of both Zytiga and Xtandi, and goes one step beyond to actively degrade the AR so as to overcome certain resistance that Zytiga and Xtandi have exhibited no activity against. Based on its differentiated mechanisms of action, superior resistance profile, and potentially satisfactory safety profile, galeterone could become a best-in-class treatment targeting the AR axis, in our opinion.

Galeterone More Selectively Inhibits the CYP17 Enzyme

Zytiga is the only therapy on the market that inhibits CYP17, an enzyme that plays key roles in androgen production and possesses two enzymatic functions: hydroxylase and lyase. Zytiga is a more-specific inhibitor of the CYP17 hydroxylase, which leads to accumulation of a number of steroids such as progesterone, deoxycorticosterone, and corticosterone, and a reduction in cortisol, which results in mineralcorticoid excess (ME), a syndrome characterized by hypertension, hypokalemia, fluid retention, and edema. As a result, Zytiga has to be co-administered with prednisone to mitigate these side effects; despite the use of prednisone, 30% of patients still develop some level of ME. In contrast, galeterone specifically inhibits CYP17 lyase activity, which does not lead to ME, negating the need for concomitant prednisone therapy.

In addition, Zytiga should be taken on an empty stomach and must be followed by an additional hour of fasting to avoid over-absorption and toxicity. Galeterone was specifically formulated to avoid this inconvenience and consequently does not have a food effect.

Galeterone Is an AR Antagonist With No Seizure Potential

Galeterone has similar potency as Xtandi in vitro. On the safety side, it does not have any theoretical seizure potential as it does not interact with the $GABA_A$ receptor in the brain, which is the main mechanism leading to seizures. We note that Xtandi interacts with $GABA_A$ receptors in the brain and in clinical studies there has been a low incidence of seizures observed, with 0.9% in the post-chemo mCRPC setting and 0.1% in the chemo-naïve mCRPC setting. Since the seizure incidence is very low, it has not become a problem for Xtandi's adoption in the marketplace. By eliminating the theoretical risk of seizures, galeterone will likely have 0% seizure rate—a potential advantage.

Galeterone's Ability to Degrade AR Renders It Effective in a Subset of Patients Who Are Unresponsive to Zytiga or Xtandi

Both Zytiga and Xtandi require the binding of the androgen to the AR to accomplish their anti-oncogenic effects. However, certain point mutations and splice variants of AR have mutated or truncated ligand-binding domain, thereby acting in a constitutively active way without the need of androgen binding. For example, the T878A point mutation in the AR allows for progesterone to bind and activate AR, giving rise to Zytiga resistance. In addition, the F876L mutation, which changes antagonistic binding to agonist signaling, was recently linked to Xtandi resistance. The most-common AR splice variant is AR-V7, which lacks the ligand binding domain and remains constitutively active; cells harboring this splice variant are therefore both Zytiga-resistant and Xtandi-resistant.

In contrast, galeterone's mechanism of action does not solely depend on disrupting receptor-ligand interaction; galeterone can degrade AR by targeting the N-terminus and DNA binding domain, which remain well-conserved throughout the various mutated and spliced forms of AR. By degrading not only the wild-type but also the mutated forms of AR, galeterone can overcome the above-mentioned resistance to Zytiga and Xtandi, paving the way for a best-in-class treatment.

Galeterone's Activity in Alternative Splice Variants

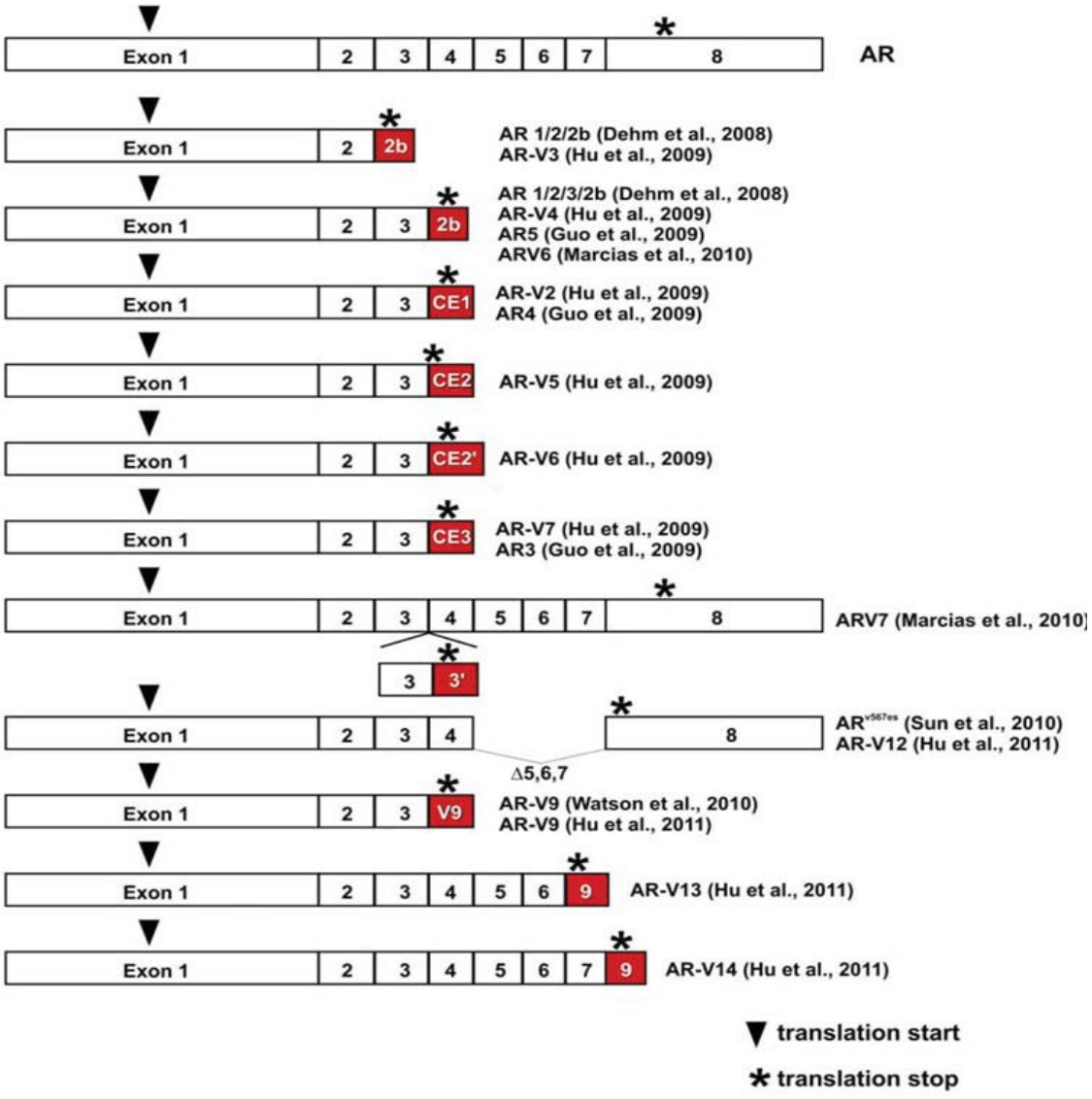
AR Mutations and Splice Variants Are Constitutively Active

The emergence of resistance could be due to many factors, including the development of point mutations and splice variants in the AR. Thus, while blocking androgen production and receptor stimulation remain crucial to slowing CRPC progression, these strategies are not sufficient to completely halt its advance.

The androgen receptor is composed of 8 exons: exon 1 encodes the N-terminal domain; exons 2-4 encode the DNA-binding domain; and exons 5-8 encode the ligand-binding domain, which includes the C-terminal domain. In prostate cancer, alternative exon splicing or exon skipping give rise to AR truncations that can affect exons 2-8, resulting in a constitutively active AR. Exhibit 12 illustrates several of the most-well-characterized AR slice variants in prostate cancer.

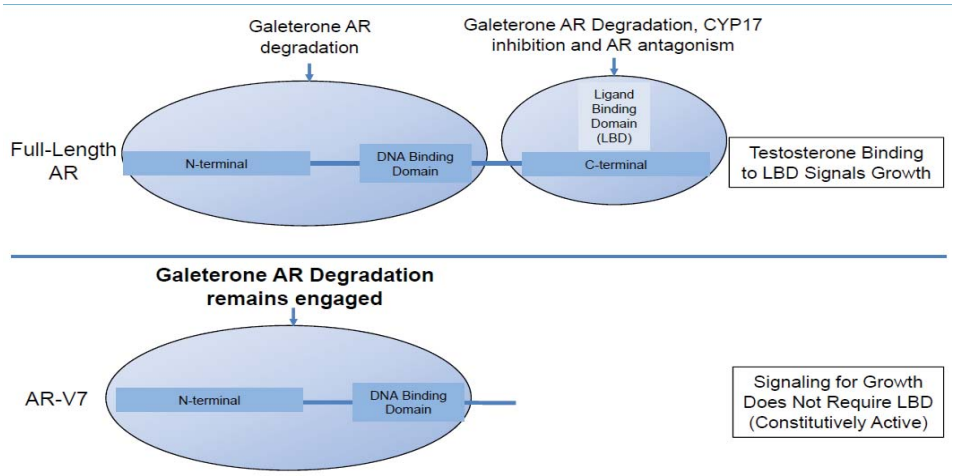
The most-common splice variant is AR-V7, which has been shown to be more highly expressed in CRPC versus hormone-naïve prostate cancer, possibly due to a treatment effect; its presence is directly associated with tumor recurrence following surgery. Moreover, because AR-V7 is truncated after exon 3, it completely lacks a ligand-binding domain, and cells with this splice variant are therefore both Zytiga-refractory and Xtandi-refractory. Exhibit 13, on page 26, represents a schematic diagram on C-terminal loss, in particular, AR-V7 splice variant.

Exhibit 12
Tokai Pharmaceuticals, Inc.
Alternative Splice Variants of the Androgen Receptor (AR)



Source: Dehm SM et al. Endocr Relat Cancer 18:R183, 2011.

Exhibit 13
Tokai Pharmaceuticals, Inc.
Androgen Receptor With C-Terminal Loss Including AR-V7 Is Constitutively Active



Source: Tokai Pharmaceuticals, Inc.

C-terminal Loss Including the AR-V7 Splice Variant Are Resistant to Zytiga and Xtandi
The C-terminus of AR contains the ligand-binding domain; Zytiga and Xtandi require this domain to exert their inhibitory functions. Without this domain the truncated form of AR becomes constitutively active, driving further prostate cancer growth and progression. Recently, new sets of data independently developed from the MD Anderson Cancer Center and The Johns Hopkins University demonstrated little or no activity by Zytiga or Xtandi in AR-V7 splice variants.

We summarize the MD Anderson data in exhibit 14; of the patients who had C-terminal loss or were AR-V7 positive, almost all demonstrated primary resistance, and 0% had derived prolonged benefit from treatment of Xtandi or Zytiga or their combination.

Exhibit 14
Tokai Pharmaceuticals, Inc.
Data from MD Anderson Linking Xtandi and Zytiga Non-Responsiveness to C-Terminal Loss/AR-47

European Urology 2014 Xtandi treatment in CRPC				
	N	Primary Resistance	Benefit	
			Moderate	Prolonged
AR-V7 Positive	7	86%	14%	0%
AR-V7 Negative	16	38%	31%	31%

ASCO 2014 Sequential Combination of Zytiga/Xtandi			
	N	Primary Resistance	Benefit
AR-V7 Positive	2	100%	0%
C-terminal loss	2	100%	0%
No AR-V7 or C-terminal loss	11	18%	82%

Source: Efstathiou et. al, European Urology, 2014, Efstathiou et. al, 2014 ASCO Annual Meeting

In the Johns Hopkins study, 31 mCRPC patients were treated with Zytiga and another 31 patients with Xtandi. Similar to the findings at MD Anderson, 0% of the AR-V7 positive patients achieved a PSA50 response, and the rPFS in these patients were only 2.1-2.3 months (exhibit 15). In contrast, for patients who were AR-V7 negative, 52%-68% PSA50 response was achieved and the rPFS ranged from 6.1 months to not-yet-reached.

Further, it appears that the prevalence of AR-V7 rises with more prior treatments. As illustrated in exhibit 15, for the naïve M1 patients, The Johns Hopkins cohorts recorded a 12% AR-V7 prevalence. However, after Xtandi treatment, the AR-V7 prevalence increased to 25%. Similarly, after Zytiga treatment, 51% of patients harbored AR-V7; and after the combination treatment of Zytiga and Xtandi, as high as 67% of patients started to harbor AR-V7.

Such dramatic differences in PSA response and rPFS also translated into statistically significant difference in overall survival. As illustrated in exhibit 16, on the following page, AR-V7 negative patients lived significantly longer than AR-V7 positive patients (p<0.001).

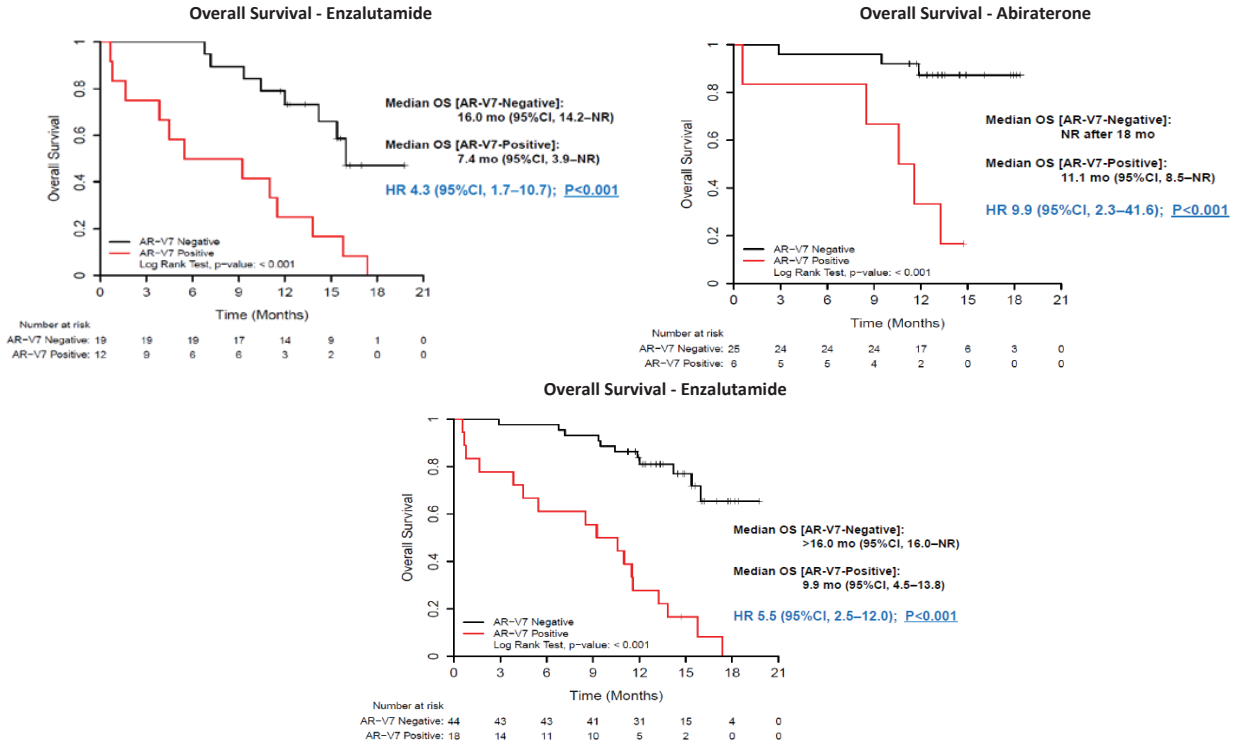
Exhibit 15
Tokai Pharmaceuticals, Inc.
Data From Johns Hopkins Study Linking Xtandi and Zytiga Non-responsiveness to AR-V7

Treatment	N	AR-V7+	Hopkins AACR/ASCO Results				
			AR-V7 Status	PSA-50	P-value	rPFS	P-Value
Zytiga	31	19%	+	0%	p<0.004	2.3 mo	p<0.001
			-	68%		Not Reached	
Xtandi	31	38%	+	0%	p<0.004	2.1 mo	p<0.001
			-	52%		6.1 mo	

CRPC Population	Prevalence
Treatment Naïve CRPC	12%
Post Xtandi	25%
Post Zytiga	51%
Post Zytiga and Xtandi	67%

Source: Antonarakis E, AACR 2014, Antonarakis E, ASCO 2014

Exhibit 16
Tokai Pharmaceuticals, Inc.
Data From Johns Hopkins Study Comparing OS in Enzalutamide and Abiraterone Treated Patients

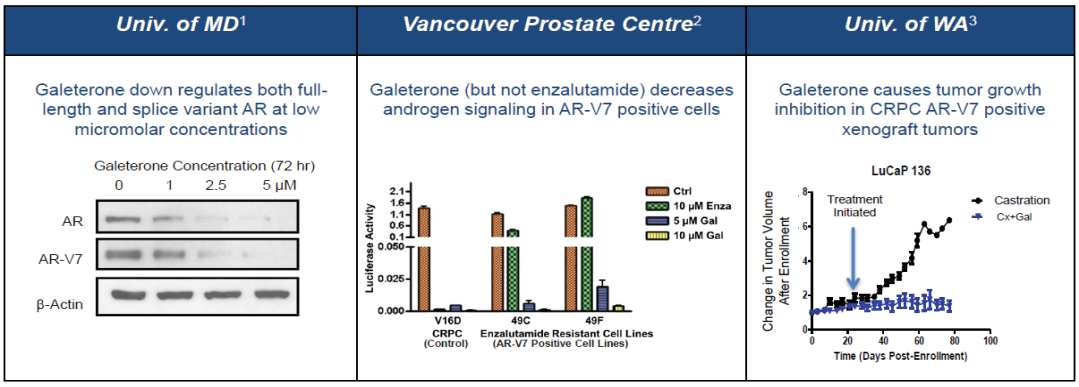


Source: Tokai Pharmaceuticals, Antonarakis et al., ESMO, September 26-30, 2014, Madrid, Spain

Galeterone Preclinical Data Demonstrates Evidence to Degrade Multiple AR Isoforms to Inhibit AR Signaling, Circumventing Drug Resistance

A number of in vitro studies demonstrated that galeterone treatment of prostate cancer cell lines leads to degradation of the AR, not only the full length, but also the truncated forms of AR and AR with point mutations. An in vivo xenograft experiment in mice also demonstrated robust activity of galeterone in AR-V7 positive tumors (exhibit 17).

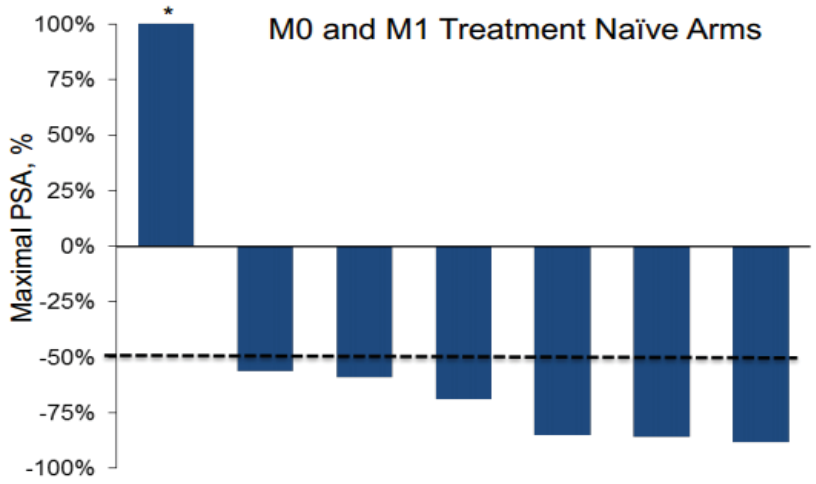
Exhibit 17
Tokai Pharmaceuticals, Inc.
Galeterone Preclinical Data: Effect on AR-V7 In Vitro



Source: Tokai Pharmaceuticals, V. Njar, 2014 (submitted), Nakouzi NA et al, AACR NCI EORTC 2013, Corey E, 2011 (unpub)

Galeterone Produced 86% PSA50 Response in Patients With C-terminal Loss
In Phase II studies, it was found that galeterone therapy was able to induce PSA50 response in 6 out of 7 treatment-naïve mCRPC patients with C-terminal loss, suggesting the potential for galeterone to address an unmet need in treating hormone-refractory prostate cancer. The seventh patient who did not demonstrate a PSA decline dropped out of the study due to an adverse event unrelated to galeterone after six weeks of treatment. This compares favorably with the 0% PSA50 response demonstrated by Zytiga and Xtandi to date.

Exhibit 18
Tokai Pharmaceuticals, Inc.
PSA Levels Post-Galeterone Treatment in Patients with AR C-Terminal Loss



Sources: Tokai Pharmaceuticals, Inc. and Taplin et al., EMSO 2014

Galeterone Clinical Program

Tokai's clinical program for its lead candidate, galeterone, has encompassed two series of studies so far, ARMOR1 (Androgen Receptor Modulation Optimized for Response) and ARMOR2. ARMOR1 was a Phase I clinical trial completed in 2012, with positive results reported at the 2012 ASCO (American Society of Clinical Oncology) meeting (June 1-June 5, Chicago). ARMOR2 is an ongoing, two-part Phase II trial evaluating galeterone in several prostate cancer populations. The two studies are summarized in detail below.

ARMOR1 Demonstrates Initial Efficacy and Safety
ARMOR1 was initiated in 2009 to investigate the safety and biological effects of galeterone in patients with CRPC. The patients were all chemo-naïve and either metastatic (M1) or non-metastatic (M0) at the time of enrollment. The open-label study ultimately enrolled 49 patients into 8 different treatment arms, testing different doses and regimens of galeterone ranging from 650 mg QD (milligrams once daily) to 2,600 mg QD. ARMOR1 used the original formulation of galeterone, an encapsulated powder.

On the efficacy side, galeterone showed strong results at its highest dose in ARMOR1. At 2,600 mg QD, 75% (9/12) of patients achieved PSA30 and 42% (5/12) achieved PSA50. In addition, at the top dose, 60% (3/5) of patients saw a tumor reduction of 25% or more on CT scans.

On the safety side, galeterone was well tolerated at all doses; 90% of the adverse events that occurred in the study were grade 1 or 2. Grade 3 and 4 adverse events included liver enzyme elevations (AST 6%, ALT 16%, and total bilirubin 2%). There were 10 serious adverse events (SAEs) in ARMOR1, and one was deemed related to galeterone, which was a grade 4 rhabdomyolysis and acute renal failure in an elderly patient. He was on simvastatin with chronic renal insufficiency, renal artery stenosis, hydronephrosis with stents, and sustained a fall.

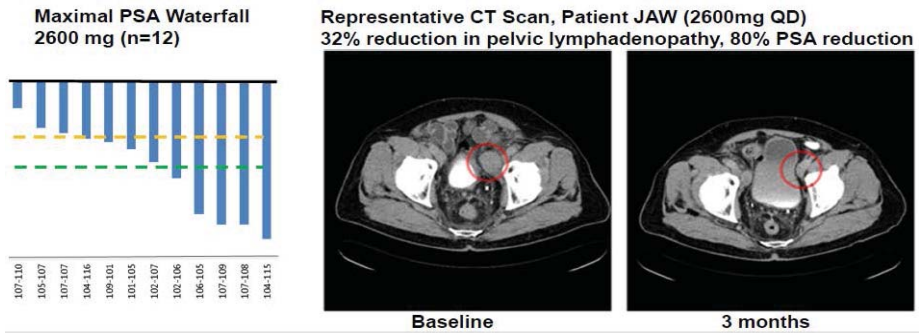
Unlike Zytiga, galeterone caused no mineralcorticoid excess, thus not requiring co-administration with prednisone.

Exhibit 19
Tokai Pharmaceuticals, Inc.
Clinical Development Summary
ARMOR (Androgen Receptor Modulation Program Optimized for Response) Program

Trial	Goal	Population	N	Forumulation	Status
ARMOR1 (Phase I)	Dose Ranging Safety and Efficacy	Treatment Naïve M0, M1	49	PIC	Complete
ARMOR2 part 1 (Phase II)	Dose Confirmation Safety and Efficacy	Treatment Naïve M0, M1	25	SDD	Complete
		Zytiga-refractory	3	SDD	Complete
ARMOR2 part 2 (Phase II)	Safety and Efficacy	Treatment Naïve M0	24	SDD	Ongoing
		Treatment Naïve M1	24	SDD	Ongoing
		Zytiga-refractory	30	SDD	Ongoing
		Xtandi-refractory	30	SDD	Ongoing
ARMOR3- SV (Phase III)	Efficacy in AR-V7 subpopulation	chemo-naïve mCRCP with AR-V7+	170	SDD	Scheduled to begin early 2015

PIC: power in capsule; SDD: spray-dried dispersion
Sources: Company reports and William Blair & Company, L.L.C. estimates

Exhibit 20
Tokai Pharmaceuticals, Inc.
ARMOR1 Proof-of-Concept Study



Source: Tokai Pharmaceuticals, Inc.

Reformulation of Galeterone Dramatically Increases Efficacy in ARMOR2

Reformulation leads to better solubility, no food effect, and better efficacy. Before the initiation of the Phase II ARMOR2 trial, Tokai reformulated galeterone from an encapsulated powder to a spray-dried dispersion tablet. This new formation allows for better solubility of the product and eliminates the modulation of its effects with food. Further, as reviewed below, the new formulation also improved efficacy.

Part 1 determined final dose at 2,550 mg QD. The ARMOR2 trial consists of two parts; the first part, now completed, was a dose-confirmation study that tested the safety and efficacy of galeterone at three doses: 1,700 mg QD, 2,550 mg QD, and 3,400 mg QD. This part of the study began enrolling in 2012 and completed enrollment in 2013. Five out of six patients receiving 2,550 mg QD achieved a PSA30 response within three months, and four out of six achieved a PSA50 response. Galeterone again showed a favorable safety profile. The 2,550 mg QD dose was selected for part two of the ARMOR2 trial.

Part 2 is ongoing with multiple cohorts of patients; PSA response comparable with those seen with Zytiga and Xtandi. The second part, which is ongoing, is testing galeterone in four different populations: M0, M1, Zytiga-refractory M1 patients, and Xtandi-refractory M1 patients with the finalized dose of 2,550 mg QD (six tablets once daily).

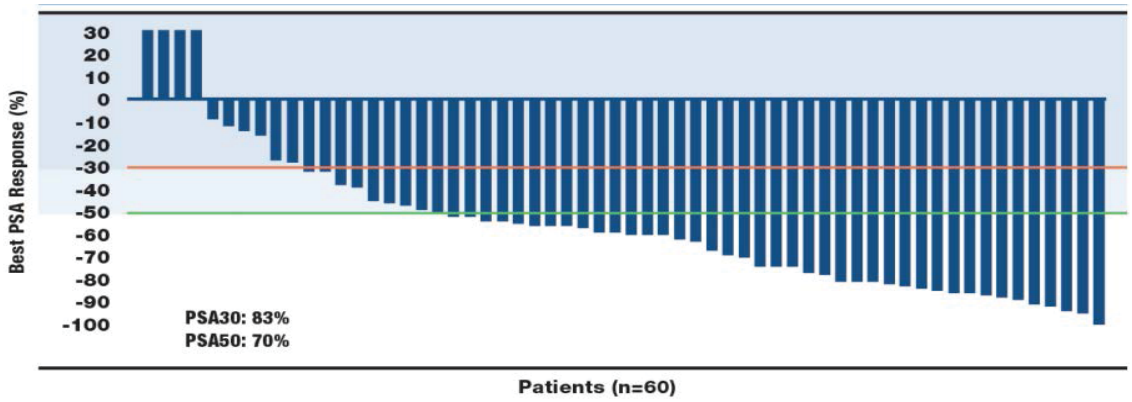
For the combined M0 and M1 patients, 83% of the 60 patients achieved a PSA30, while 70% of patients achieved a PSA50 response. Particularly among M1 patients, 85% of the 30 patients achieved PSA30 and 77% achieved PSA50 (exhibit 21, on the following page). These results are comparable to those generated by both Zytiga and Xtandi. We note that Zytiga showed a 67% PSA50, while Xtandi showed a 62% PSA50 in M1 patients in its previous Phase I/II studies.

To date, galeterone has also shown some activity in Zytiga-refractory and Xtandi-refractory patients. Eleven out of 30 Zytiga-refractory patients had a decline in their PSA on galeterone at 12 weeks of treatment, and three of them (10%) achieved a PSA30. Four out of 9 Xtandi-refractory patients so far had demonstrated a decline in their PSA on galeterone treatment; data is still immature. AR expression analysis in these patients are ongoing.

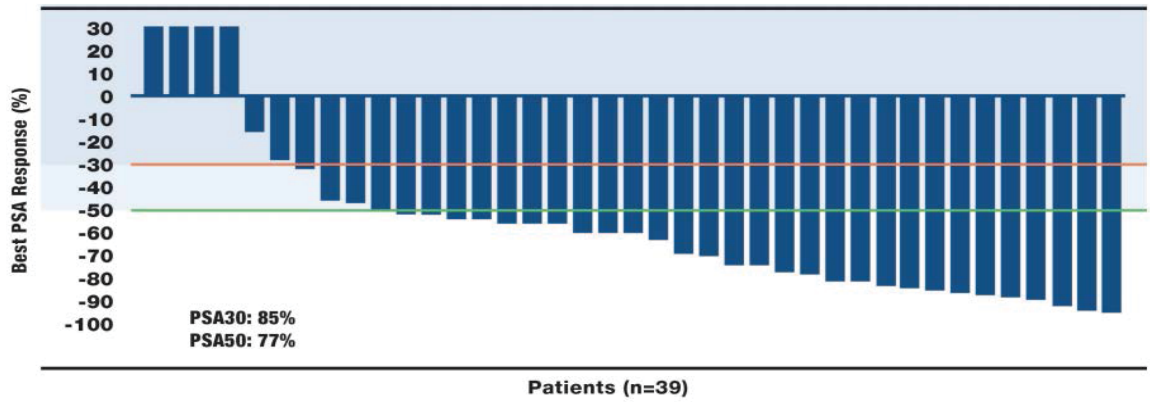
On the safety side, 76% of patients treated with 2,550 mg QD in ARMOR2 experienced at least one adverse event; 74% of those patients reported a maximum adverse event severity of grade 1 or 2. The most-common adverse events were nausea, diarrhea, decreased appetite, fatigue, increased aminotransferase levels, hypokalemia and pruritus. As found in ARMOR1, there were no reports of seizures or ME related to taking galeterone; the galeterone reformulation used in ARMOR2 eliminated the food effect observed in ARMOR1.

Exhibit 21
Tokai Pharmaceuticals, Inc.
Results From ARMOR2 Phase II Study: Effect of galeterone on PSA levels

a) Maximal PSA response at 12 weeks in treatment-naïve M0 and M1 patients



b) Maximal PSA response at 12 weeks in treatment-naïve M1 patients



C) Efficacy of the four cohorts in ARMOR2

Cohort	No.	Any PSA Decline n (%)	Best Response by RECIST 1.1 (Soft Tissue/Visceral) n (%)	Best Response by PCWG2 (Bone) n (%)
M0, TN	21	21 (100)	No evidence of M1 at 12 weeks	No evidence of M1 at 12 weeks
M1, TN	39	35 (90)	PR 3/18 (17) SD 13/18 (72)	SD 27/36 (75)
Abi-R	30	11 (37)	SD 4/11 (36)	SD 13/28 (47)
Enz-R	9	4 (44)	NA	SD 4/7 (57)

PCWG2=Prostate Cancer Working Group 2, RECIST=Response Evaluation Criteria in Solid Tumors, PR=partial response, SD=stable disease

Sources: Tokai Pharmaceuticals, Inc. and Taplin et al., ESMO 2014

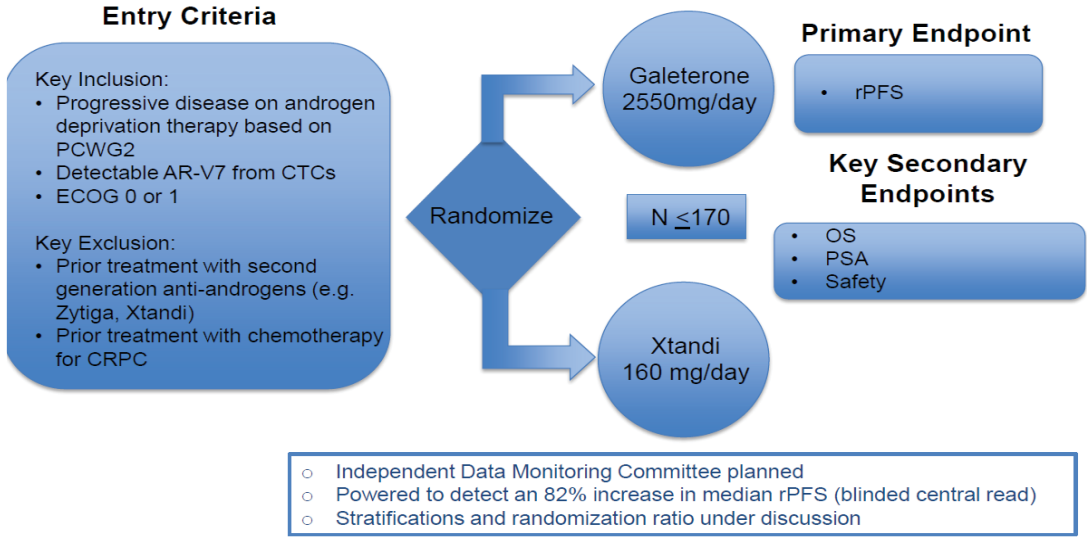
Galeterone Phase III Clinical Development Strategy

Phase III ARMOR3-SV design to target AR-V7 splice variant. Tokai has reached agreement with the FDA on the design of a single pivotal study that targets chemo-naïve mCRPC (M1) patients who harbor the AR-V7 splice variant. It is unclear what the percentage of patients at the M1 setting harbor the AR-V7 variant; literature suggests a range from 12% to over 30%. We assume 20% in our model.

To conduct such a trial, Tokai needs to develop a companion diagnostic test to prospectively screen patients with the AR-V7 splice variant, which should be developed, validated, and approved by the FDA by early 2015.

The pivotal ARMOR3-SV study intends to enroll 170 patients who harbor the AR-V7 splice variant. The patients will be randomized 1:1 to either galeterone or Xtandi. The primary endpoint is radiographic progression-free survival (rPFS), and secondary endpoints include OS, PSA response, and safety.

Exhibit 22
Tokai Pharmaceuticals, Inc.
Phase III ARMOR3-SV Clinical Trial Schematic



Source: Tokai Pharmaceuticals, Inc.

Top-line data from ARMOR3-SV could come as early as the end of 2016; we believe Tokai has identified a time- and capital-efficient path to approval. Assuming that the study is initiated in early 2015, Tokai guides top-line data release by the end of 2016—a relatively short time to conduct any Phase III oncology program, in our opinion. An NDA could be submitted in early 2017 and galeterone could hit the market in late 2017/early 2018, which represents a quick route to market. Further, the 170-patient single pivotal study required for approval is also highly capital-efficient, as compared to most other Phase III oncology programs.

A chemistry, manufacturing, and controls (CMC) information meeting with FDA is planned for first half 2015. Phase III recruitment is also scheduled to begin in 2015, with top-line data expected in 2016, and an NDA submission to follow in 2017.

We Assign an 85% Chance of Success to the Phase III ARMOR3-SV Study
We assign the galeterone Phase III program an 85% probability of success, based on the following arguments and rationales.

Targeting the AR axis is the major approach to treat prostate cancer, and galeterone has a three-pronged approach to inhibit the AR axis. Targeting the AR axis is the most relevant and the dominating approach to treat prostate cancer. The first-line androgen depravation therapy Lupron and the first-generation anti-androgen Casodex have been the mainstay of prostate cancer therapy for decades. Second-generation hormonal agents Zytiga and Xtandi became successful blockbusters shortly after their respective launches. We believe galeterone, which also targets the AR axis with three distinct mechanisms, should have a high probability of success.

Activity of galeterone in the AR-V7 splice variants is supported by preclinical and clinical observations. The activity of galeterone in vitro to reduce AR levels, including wild type AR, AR with point mutations, and AR with various truncated forms including C-terminal loss and AR-V7, has been observed. Its activity in AR variants was also observed in an in vivo xenograft mouse model. Besides the preclinical evidence, clinical data to date also demonstrated that six out of seven chemo-naïve mCRPC patients with C-terminal loss have responded to galeterone.

Favorable and efficient study design agreed on with the FDA: the first mCRPC pivotal study with rPFS as primary endpoint. The two recently conducted Phase III pivotal studies in the chemo-naïve mCRPC settings, COU-AA-302 for Zytiga and PREVAIL for Xtandi, enrolled more than 1,000 and 1,700 patients, respectively, and had rPFS and OS as co-primary endpoints. Both studies took three to four years from start of enrollment to top-line data. In contrast, the FDA has approved Tokai to conduct a single pivotal study for galeterone in the same chemo-naïve mCRPC setting, but specifically in the AR-V7 population and with rPFS as the primary endpoint; as a result, the target enrollment required is only 170. Such a design significantly reduces time and capital required for galeterone approval, as compared to the paths to approval taken by Zytiga and Xtandi.

We believe the ARMOR3-SV study is well powered to be successful on the primary endpoint of rPFS. ARMOR3-SV targets to enroll 170 chemo-naïve mCRPC patients harboring the AR-V7 variant; the patients will be randomize 1:1 to galeterone and Xtandi. The study is 90% powered to demonstrate an 82% improvement in median rPFS, galeterone over Xtandi.

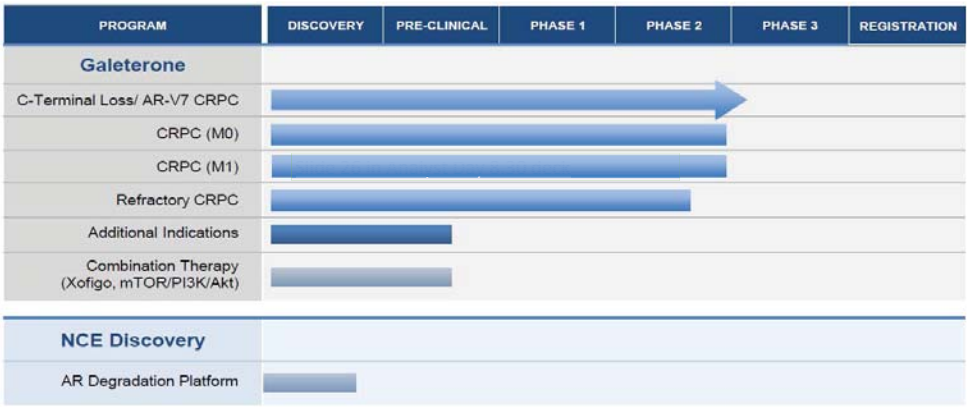
We believe the powering assumption for the study is conservative. In the PREVAIL study in the chemo-naïve mCRPC M1 setting where Xtandi was evaluated against placebo, the PSA50 response was 78% for Xtandi and 3% for placebo, which corresponded to an rPFS of 16 months for Xtandi and only 4 months for placebo. In M1 patients with AR-V7 variants, to date we have observed 86% (six out of seven) PSA50 response for galeterone, versus 0% for Xtandi. Further, the rPFS of Xtandi in the AR-V7 population is observed at 2-3 months; in contrast, among the six AR-V7 positive patients who achieved PSA50 on 12 weeks of galeterone treatment, four have gone on to the extension phase of the study and have demonstrated rPFS of 5.2 months to greater than 9 months to date. As a result, we believe the 82% improvement in rPFS assumed for the ARMOR3-SV study is likely conservative.

Development of a companion diagnostic to prospectively identify AR-V7 for study enrollment is prerequisite for the Phase III study start in early 2015, and approval is required for commercialization. Tokai has contracted third parties to finalize and validate an assay to prospectively identify the AR-V7 variant from patient's circulating tumor cells (CTC). Tokai needs to submit an investigational device exemption application (IDE) for the assay to the FDA before start of the Phase III study. This companion diagnostic test will need to be approved by the FDA through its premarket approval or PMA process before commercialization. During the Phase III study, the AR-V7 identification will be conducted by a central laboratory. Tokai expects to screen 1,000 chemo-naïve mCRPC patients to identify the target enrollment of 170 patients positive for AR-V7, assuming an approximate 20% incidence.

Other Pipeline Indications
Besides the first indication in M1 AR-V7 patients, galeterone could be developed for a number of other settings in prostate cancer, including going into earlier urology settings in prostate cancer, combination studies with currently approved drugs such as Xofigo, and potentially in Zytiga and/or Xtandi failures. Combinations of galeterone with other small molecules such as mTOR/PI3K/Akt inhibitors have also demonstrated interesting preclinical activity.

The AR degradation platform could produce more candidates in the pipeline. Tokai in-licensed galeterone as well as the AR degradation platform from the University of Maryland, Baltimore. These compounds could have improved properties over galeterone in AR inhibition and degradation and could generate value in the long term.

Exhibit 23
Tokai Pharmaceuticals, Inc.
Clinical Pipeline



Source: Tokai Pharmaceuticals, Inc.

Intellectual Property

License Agreement With University of Maryland, Baltimore
Tokai in-licensed exclusive worldwide rights to galeterone and the AR degradation platform from the University of Maryland, Baltimore. The agreement includes exclusive rights to 5 issued U.S. patents, 44 issued foreign patents, and 16 U.S. and foreign applications. So far, Tokai has paid the university $220,000 in licensing fees and milestone payments. The University of Maryland, Baltimore is entitled to future milestone payments and royalties arising from the development and commercialization of galeterone and related compounds covered under their IP. These include:

- Maintenance fees of $10,000 each year until the first commercial sale of one of the products arising from the licensed technologies;
- A milestone payment of $50,000 for each NDA submission;
- A milestone payment of $100,000 for each FDA approval;
- Low-single-digit royalties—with a minimum of $50,000 annually—on aggregate worldwide net sales of licensed technologies, including sales by sub-licensees. These payments will continue until 10 years after the first commercial sale of the product or following expiration of the last-to-expire applicable patent, whichever is later;
- 10% of all non-royalty sublicense income.

The Expanding Patent Portfolio

It is important to note that the patent covering galeterone's composition of matter is set to expire in 2017. Methods-of-use patents licensed from the University of Maryland, Baltimore, which cover prostate cancer and prostate disease, are set to expire in 2026-2027. An extension may be obtainable under the Hatch-Waxman Act.

Because of the short remaining lifespan on the patent covering composition of matter, Tokai has aggressively sought to strengthen its IP security with patents comprising methods of production, mechanisms of action, modified formulations, and use in combination therapies. Tokai owns 3 issued patents, including 1 foreign patent, and has submitted an additional 10 U.S. and 34 foreign applications. This growing portfolio covers:

- Formulation patents for galeterone in spray dried dispersion; if issued, these patients are expected to expire in 2032;
- Galeterone and related compounds for the treatment of prostate cancer mediated by AR variants including AR-V7; if issued, these patients would expire in 2034;
- Use of galeterone as a concomitant or adjuvant therapy with Akt/PI3K inhibitors and others; if issued, these patents are expected to expire in 2032;
- Prodrugs, metabolites, and analogs of galeterone;
- Novel prodrugs that inhibit CYP17 enzyme;
- Methods of synthesizing mammalian steroid metabolites and their uses in treating AR-mediated pathologies.

Exhibit 24
Tokai Pharmaceuticals, Inc.
Income Statement
(dollars in thousands)

	2012	2013	2014					2015	2016
Income Statement	FY:12A	FY:13A	Q1A	Q2A	Q3E	Q4E	FY:14E	FY:15E	FY:16E
Revenues									
Galeterone	$0	$0	$0	$0	$0	$0	$0	$0	$0
License revenue			-	-	-	-	-	-	-
Collaboration revenue				-	-	-	-	-	-
Total Revenues	$0	$0	-	-	-	-	-	-	-
Expenses									
COGS			-	-	-	-	-	-	-
% of product sales	0%	0%	0%	0%	0%	0%	0%	0%	0%
R&D expense	7,370	12,201	3,748	4,200	4,242	4,284	16,474	24,434	40,129
SG&A expense	2,279	3,548	1,129	1,700	1,717	1,734	6,280	8,222	10,853
Total Operating Expenses	**9,649**	**15,749**	**4,877**	**5,900**	**5,959**	**6,019**	**22,755**	**32,655**	**50,982**
Operating income	(9,649)	($15,749)	(4,877)	(5,900)	(5,959)	(6,019)	(22,755)	(32,655)	(50,982)
Finance income		-	-	-	-	-	-	348	199
Finance costs			-	-	-	-	-	-	-
Other (expense) income, net		24		79	-	-	79	-	-
Revaluation of preferred stock warrant liabilities	-	-	-	-	-	-	-	-	-
Revaluation of future purchase rights liabilities	-	-	-	-	-	-	-	-	-
Total other Income (expense)	**(9,649)**	**(15,725)**	**(4,877)**	**(5,821)**	**(5,959)**	**(6,019)**	**(22,676)**	**(32,308)**	**(50,783)**
Pretax income/(loss)	(9,649)	(15,725)	(4,877)	(5,821)	(5,959)	(6,019)	(22,676)	(32,308)	(50,783)
Other comprehensive gain/(loss)	-	-	-	-	-	-	-	-	-
Amortization of deemed dividend	-	-	-	-	-	-	-	-	-
Accretion to redemption value of redeemable convertible preferred stock	(34)	-	-	-	-	-	-	-	-
Provision for income taxes/(income)			-	-	-	-	-	-	-
Tax rate	0%	0%	0%	0%	0%	0%	0%	0%	0%
Net Income/(Loss)	**($9,683)**	**($15,725)**	**($4,877)**	**($5,821)**	**($5,959)**	**($6,019)**	**($22,676)**	**($32,308)**	**($50,783)**
Fair value adjustment		-	-	-	-	-	-	-	-
Cumulative translation adjustment			-	-	-	-	-	-	-
Total comprehensive loss	**($9,683)**	**($15,725)**	**($4,877)**	**($5,821)**	**($5,959)**	**($6,019)**	**($22,676)**	**($32,308)**	**($50,783)**
Pro forma EPS, continuing operations									
GAAP EPS	**($2.97)**	**($3.61)**	**($0.32)**	**($0.38)**	**($0.34)**	**($0.28)**	**($1.30)**	**($1.47)**	**($2.27)**
Weighted average shares outstanding, diluted	3,261	4,356	15,312	15,312	17,300	21,800	17,431	21,988	22,350

Sources: Company reports and William Blair & Company, L.L.C. estimates

Exhibit 25
Tokai Pharmaceuticals, Inc.
Balance Sheet
(dollars in thousands)

	2012	2013	2014	2015	2016
Balance Sheet Data	FY:12A	FY:13A	FY:14E	FY:15E	FY:16E
Current assets					
Cash and cash equivalents	11,691	31,753	97,697	66,589	16,306
			-	-	-
Short-term investments, available-for-sale	-	-	-	-	-
Trade receivables	-	-	-	-	-
Other receivables	-	-	-	-	-
Inventories, net	-	-	-	-	-
Prepaid expenses	235	425	589	589	589
Deferred financing costs, current portion	-	-	-	-	-
Other current assets	-	-	-	-	-
Total current assets	11,962	32,178	98,286	67,178	16,895
Property, plant and equipment	16	29	36	136	236
Deferred offering costs	-	-	1,524	1,524	1,524
Restricted cash	20	50	50	50	50
Other assets	-	-	71	71	71
Total assets	**$11,962**	**$32,287**	**$99,967**	**$68,959**	**$18,776**
Current liabilities					
Accounts payable	764	5	1,716	2,716	2,916
Other current liabilities	-	-	-	-	-
Convertible debenture	-	-	-	-	-
Deferred revenue	-	-	-	-	-
Accrued expenses	1,254	2,204	-	-	-
Related-party convertible notes payable	-	-	-	-	-
Future purchase rights liabilities	-	-	-	-	-
Preferred stock warrants	-	-	-	-	-
Common stock warrants	-	-	-	-	-
Total current liabilities	2,018	2,209	1,716	2,716	2,916
Deferred revenue, net of current			-	-	-
Other long-term liabilities	-	-	-	-	-
Total liabilities	**$2,018**	**$2,209**	**$1,716**	**$2,716**	**$2,916**
Stockholders' equity	(39,901)	(55,267)	12,905	(19,102)	(69,485)
Total liabilities, convertible preferred and stockholders' equity	**$11,962**	**$32,287**	**$99,967**	**$68,959**	**$18,776**

Sources: Company reports and William Blair & Company, L.L.C. estimates

Exhibit 26
Tokai Pharmaceuticals, Inc.
Cash Flows

Tokai Pharmaceuticals, Inc. Cash Flow Data	**2012** FY:12A	**2013** FY:13A	**2014** FY:14E	**2015** FY:15E	**2016** FY:16E
Net cash from operating activities					
Net Income (Loss)	(9,649)	(15,725)	(22,676)	(32,308)	(50,783)
Adjustments					
Depreciation and amortization	9	10	557	612	673
Stock-based compensation expense	210	238	1,158	1,274	1,402
Loss on asset disposition	-	-	-	-	-
Non-cash interest expense relating to notes payable	-	-	-	-	-
Non-cash interest expense relating to related-party convertible loan payable	-	-	-	-	-
Non-cash restructuring charges	-	-	-	-	-
Change in Operating Assets and Liabilities					
Contracts receivable	-	-	-	-	-
Prepaid and other assets	139	(190)	(164)	-	-
Accounts payable and accrued liabilities	(42)	191	(493)	1,000	200
Other liabilities	-	-	-	-	-
Net cash used in operating activities	**(9,333)**	**(15,476)**	**(21,618)**	**(29,421)**	**(48,508)**
Cash flows from investing activities					
Purchase of property and equipment	(8)	(23)	(121)	(133)	(146)
Change in restricted cash	-	(30)	1,524	-	-
Purchase of short-term investments	-	-	(25,000)	-	-
Sales of short-term investments	-	-	-	10,000	10,000
Maturities of short-term investments	-	-	-	-	-
Net cash used in (provided by) investing activities	**(8)**	**(53)**	**(23,597)**	**9,867**	**9,854**
Cash flows from financing activities					
Proceeds from issuance of convertible preferred stock, net of issuance costs	18,775	35,406	-	-	-
Proceeds from issuance of related-party convertible notes payable	-	-	-	-	-
Proceeds from the exercise of stock options, net of repurchases	4	215	400	1,200	2,400
Proceeds from issuance of related-party convertible loan payable	-	-	-	-	-
Proceeds from issuance of notes payable	-	-	-	-	-
Proceeds from initial public offering, net of offering costs	-	-	90,396	-	-
Payments of initial public offering costs	-	(30)	(5,325)	(1,878)	(1,878)
Repayment of notes payable	-	-	-	-	-
Net cash provided by financing activities	**18,779**	**35,591**	**85,471**	**(678)**	**522**
Cash balance (Beginning of Period)	**2,253**	**11,691**	**16,807**	**57,063**	**36,831**
Difference	9,438	20,062	40,256	(20,232)	(38,132)
Cash balance (End of Period)	**11,691**	**31,753**	**57,063**	**36,831**	**(1,301)**
Marketable securites	**-**	**-**	**40,633**	**29,758**	**17,607**
Cash balance plus marketable securities (end of period)	**11,691**	**31,753**	**97,697**	**66,589**	**16,306**

Sources: Company reports and William Blair & Company, L.L.C. estimates

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DJIA:	16,659.25
S&P 500:	1,928.21
NASDAQ:	4,378.34

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

Bristol-Myers Squibb Company (Outperform)	$49.50
Dendreon Corporation (Underperform)	$1.02
Exelixis Inc. (Outperform)	$1.55
Johnson & Johnson	$102.08
Medivation, Inc. (Outperform)	$95.01
OncoGenex Pharmaceuticals, Inc. (Outperform)	$2.28
Sanofi	$53.08
Teva Pharmaceutical Industries Ltd.	$52.82

Current Ratings Distribution (as of 9/30/14)

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Outperform (Buy)	65%	Outperform (Buy)	16%
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Underperform (Sell)	1%	Underperform (Sell)	0%

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