Janney CAPITAL MARKETS

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Biotechnology

October 13, 2014

Tokai Pharmaceuticals, Inc. (TKAI) - BUY

TKAI: Initiating With a Buy Rating; $21 FV

Price:	$11.82
Fair Value Estimate:	$21.00
52-Week Range:	$9.67 - $30.00
Market Cap (MM):	$258
Shr.O/S-Diluted (mm):	21.8
Average Daily Volume:	NA
Yield:	0.0%
Cash/Share:	$(3.55)
FCF Yield:	NA
Debt/Cap:	0%

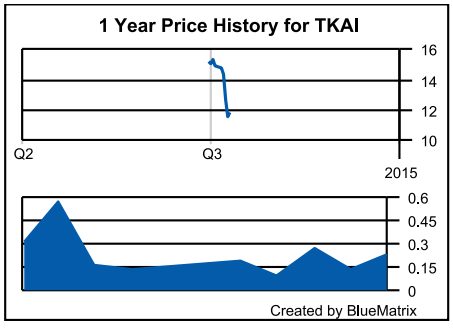
FYE: Dec	2013E	2014E	2015E
EPS:	$(1.29)E	$(1.20)E	$(1.25)E
Prior EPS:	NC	NC	NC
P/E Ratio:	NA	NA	NA

Quarterly EPS:

Q1	--	--	$(0.31)E
Q2	--	--	$(0.31)E
Q3	--	$(0.29)E	$(0.31)E
Q4	--	$(0.24)E	$(0.31)E

Quarterly Revenue (M):

Q1	$0E	$0E	$0E
Q2	$0E	$0E	$0E
Q3	$0E	$0E	$0E
Q4	$0E	$0E	$0E
Year:	$0E	$0E	$0E



PORTFOLIO MANAGER BRIEF

We view TKAI's main value driver, galeterone, as a first-in-class and best-in-class, selective and multi-targeted small molecule that should offer advantages over existing prostate cancer therapies. Galeterone's value proposition lies in its unique mechanism of action (MOA) that can confer efficacy in a resistant population and improved safety. Activity in castration-resistant prostate cancer (CRPC) and C-terminal loss should provide a quicker pathway to market and significant expansion opportunity, in our view.

ANALYST NOTES

- **Galeterone's value proposition should differentiate it from the competition.** This highly-selective oral therapy is novel based on: 1) MOA, which should allow for potential use across all stages of prostate cancer; 2) potential efficacy in metastatic CRPC patients who are resistant to other marketed hormonal therapies; and 3) safety with no need for concomitant steroid use and no seizure risks.
- **Meaningful global market opportunity with expansion from other indications.** The addressable CRPC market is approximately $9+B. We estimate galeterone could reach peak sales of $1.5B that only take into account the ARV7 mCRPC population. Upside could come from expansion in other prostate cancer populations. The CRPC market has been validated by 2013 global Xtandi and Zytiga sales of $2.1B.
- **Pipeline provides multiple shots on goal that could result in future upside.** The company's strategy of developing galeterone in multiple oncologic indications de-risks its clinical program and helps maximize the drug's commercial potential, in our view.
- **Compelling valuation ahead of de-risking milestones.** Our DCF analysis of cash flows through 2022 indicate that TKAI shares are highly undervalued, considering near-term de-risking milestones such as an FDA CMC and EMA meetings in 4Q14 and completion of assay development in 1H15 could provide share appreciation.

RESEARCH ANALYST CERTIFICATIONS AND IMPORTANT DISCLOSURES ARE ON PAGES 20 - 22 OF THIS REPORT

INVESTMENT THESIS

Tokai is developing its lead asset, galeterone, for the treatment of prostate cancers that target C-terminal loss splice variants, which can cause resistance to other marketed therapies. We view galeterone as a first-in-class and best-in-class, Phase III-ready therapy that can offer the potential for improved efficacy and safety over currently marketed products, ease of use, broad utility, and importantly, potential for lower risk of resistance. We believe galeterone's value propositions will differentiate it from its competitors to make a meaningful impact in the $9+B marketplace.

ISSUES TO CONSIDER

Key Issue	Our Position	Timing	Impact
Product differentiation from competitors	Galeterone's unique, selective and targeted MOA, including distinct androgen receptor (AR) degradation, should help differentiate it from marketed therapies that have improved outcomes.	12-24 Months	
One-product company	The company has one main drug, galeterone, as its main value driver. However, we view the drug as a pipeline within a drug as galeterone is being developed in numerous other prostate and other cancers.	12-24 Months	
Lack of major clinical data in near term	Although top-line Phase III data in ARV7 splice variants will not be available until YE:16, there are numerous other value-creating catalysts that could boost shares in the near term such as results of the FDA CMC and EMA meetings in 4Q14, completion of assay development in 1H15 and clinical data presentations at the GU ASCO and ASCO conferences in 1H15.	0-3 Months	

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Company Description:

Tokai Pharmaceuticals (TKAI) is a development stage biopharmaceutical company focused on developing and commercializing unique and proprietary therapies, utilizing galeterone for the treatment of prostate cancer and other androgen-driven diseases. Galeterone is a highly selective, multi-targeted, oral small molecule drug candidate that disrupts the androgen receptor pathway. Currently, TKAI is developing galeterone for the precision treatment of patients with castration resistant prostate cancer (CRPC) that have a specifically altered androgen receptor. Currently, galeterone has completed a Phase 1 proof-of-concept trial and has completed the first of a two part Phase 2.

We are initiating coverage of Tokai Pharmaceuticals with a Buy rating and $21 fair value estimate. We view TKAI as a reasonable investment for small-cap investors for the following reasons:

- **Novel MOA.** The benefit to outsourcing business processes is that it allows clients to focus on core offerings and it reduces costs.
- **Significant global market opportunity in lead indications.** We estimate peak worldwide sales for galeterone could reach $1.5B on fairly conservative assumptions for ARV7 splice variants.
- **Several shots on goal.** The company's strategy of developing galeterone in multiple indications de-risks the clinical program and provides the company with several potential unmet medical opportunities, in our view.
- **Value-creating catalysts.** Near-term de-risking milestones could provide share appreciation.
- **Attractive valuation.** Our DCF analysis of cash flows through 2022 suggest decent upside to current levels, without including value from other indications.

Prostate cancer is the most commonly diagnosed solid organ malignancy in the US and is the second leading cause of cancer death in American men.

What is Castration Resistant Prostate Cancer (CRPC)?

Prostate cancer is the most commonly diagnosed solid organ malignancy in the US and is the second leading cause of cancer death in American men. According to the American Cancer Society approximately 233,000 new cases of prostate cancer will be diagnosed in the US with approximately 29,000 deaths.

The growth and survival of prostate cancer tumor cells depends primarily on the functioning androgen receptor signaling pathway. Androgens such as testosterone are male sex hormones. As testosterone fuels tumor growth, first-line therapy focuses on reducing the amount of testosterone. Though many patients respond to this therapy, almost all advanced prostate cancer patients experience tumor growth despite the low levels of testosterone. These patients are considered "castration resistant".

Treatment of Prostate Cancer:

Growth and Survival of Prostate Cancer – In order to understand the treatment of prostate cancer, the mechanisms of how these tumor cells survive must be explored. As previously mentioned, the key to prostate cancer is the functioning of the androgen receptor signaling pathway. This pathway is usually activated by androgens, such as testosterone or DHT. The androgens bind to the ligand binding domain of specific androgen receptors in prostate cancer cells. Once bound, the androgen/androgen receptor complex passes into the nucleus of the cancer cell, binding to DNA and triggering abnormal cell growth and tumor progression.

First-line Therapies:
As testosterone is the initial trigger for prostate cancer growth, first-line therapies usually involve lowering the amount of testosterone in the body. This is also known as androgen deprivation therapy or ADT, which usually includes luteinizing hormone releasing hormone or LHRH. Early-stage patients who receive and respond to this treat are considered to have hormone-sensitive prostate cancer. The majority of advanced prostate cancer patients initially respond to this therapy. Sadly, after beginning ADT, almost all of these patients experience a recurrence in tumor growth even with the testosterone reduction. As previously explained, these patients are considered to now have "castration resistant" prostate cancer.

An option for these CRPC patients is an anti-androgen which blocks the binding of the androgen to the androgen receptor. Like treatment with LHRH, eventually, patients develop resistance. Once resistance develops, patients are taken off drug but remain on LHRH for the remainder of their lives.

Prior to 2010, if a patient became resistant to both of these hormonal treatments, the next line of therapy was chemotherapy. The only FDA-approved drug at that time was Taxotere (docetaxel). If that patient failed to respond to the chemotherapy, there were no other FDA-approved treatments.

Second-line Therapies:
Since 2010 the FDA has approved five new agents to treat patients with CRPC. These treatments include Zytiga, Xtandi, Jevtana, Provenge and Xofigo. Of these new treatments, Zytiga and Xtandi have the highest in worldwide sales with 2013 sales of $1.7B and $455M, respectively.

Zytiga – Approved in April of 2011, Zytiga is an oral secondary hormonal treatment for use in combination with prednisone to treat men with metastatic CRPC post- and pre-chemotherapy. Zytiga works by inhibiting CYP17, which is an enzyme required for androgen synthesis. By inhibiting CYP17, less testosterone is synthesized.

Xtandi – Approved in August 2012, Xtandi is an oral secondary hormonal treatment for use in men with pre- and post-chemotherapy metastatic CRPC. Xtandi works differently than Zytiga. Xtandi is an androgen receptor antagonist and blocks the binding of testosterone or DHT to the androgen receptor. Treatment for these advanced patients depends on the status of the disease, including whether it is metastatic and what prior treatments the patient has received. Various treatments and approved patient populations are listed in Exhibit 1.

Exhibit 1: FDA-Approved Treatment For Advanced Prostate Cancer

Patient Populations	Treatment Options	Non-Metastatic		Metastatic			
				CRPC			
		Hormone sensitive	CRPC	Pre-Chemo	First-Line Chemo	First-Line Post Chemo	Salvage
Primary Hormonal Treatment	LHRH	X	X	X	X	X	X
	Androgen Receptor Antagonists	X	X				
Secondary Hormonal Treatment	Zytiga			X		X	
	Xtandi					X	
Chemotherapy	Taxotere				X		
	Jevtana					X	X
Immunotherapy	Provenge			X		X	
Bone Targeting Agent	Xofigo			X	X	X	X

Source: Company reports and Janney reports

Why an Unmet Medical Need Still Exists: Truncated Androgen Receptors:

Truncated or shortened androgen receptors are missing the end of the receptor that contains the ligand binding domain. This is known as having C-terminal loss. Androgen receptor splice variant-7 (AR-V7) is an example of a truncated androgen and is the most prevalent of splice variants that cause C-terminal loss. The loss of the C-terminus causes continual activation of the androgen receptor pathway. This activation means that tumor growth continues even in the absence of androgens and androgen binding.

The lack of the C-terminus is believed to diminish the effectiveness of treatments like Zytiga and Xtandi. As Zytiga works by reducing the amount of testosterone or DHT produced and Xtandi works by blocking the binding of those androgens, these therapies require a functional binding site in order to be effective. However, tumors that exhibit truncated androgen receptors with C-terminus loss, including AR-V7, do not have this functional binding site and are believed to continue to grow despite the patient being on therapy. Limitations of these drugs have been supported by recent published research.

MD Anderson – At ASCO, researchers from MD Anderson presented data from a clinical study of 60 CRPC patients with bone metastases who were treated with a sequential regimen of Zytiga and Xtandi. Within the study, the primary resistance was defined as discontinuation of therapy due to symptomatic and/or imaging evidence of disease progression at least four months after treatment initiation. A subset of 15 patients was evaluated for C-terminal loss with 4 having C-terminal loss, including 2 with AR-V7. All four patients showed primary resistance. Of the 11 that did not have C-terminal loss or AR-V7, nine showed benefit. The data is summed up in Exhibit 2:

Exhibit 2: MD Anderson C-Terminal Loss and AR-V7 Findings at ASCO

	N	Primary Resistance	Benefit
AR-V7 positive	2	100% (2/2)	0% (0/2)
C-Terminal loss (excluding AR-V7)	2	100% (2/2)	0% (0/2)
Negative for AR-V7 and C-terminal loss	11	18% (2/11)	82% (9/11)

Source: Company reports

An additional study was accepted for publication by European Urology in May 2014. This study involved the evaluation of bone biopsy specimens from CRPC patients with bone metastases that had been treated with Xtandi to determine the effect of Xtandi on the cancer and to associate any effects with clinical observation. Primary resistance was defined as discontinuation of therapy due to symptomatic and/or imaging evidence of disease progression at least four months after treatment initiation; Moderate disease was considered progression within four to six months and prolonged benefit was at least six months after treatment initiation.

The study involved 23 patients. Those with AR-V7 showed 86% of primary resistance and 38% of patients that did not have AR-V7 showed primary resistance. Results are shown in Exhibit 3:

Exhibit 3: MD Anderson AR-V7 Baseline (European Urology)

Outcome	N	Primary Resistance	Moderate Benefit	Prolonged Benefit
AR-V7 Positive	7	86% (6/7)	14% (1/7)	0% (0/7)
AR-V7 Negative	16	38% (6/16)	31% (5/16)	31% (5/16)

Source: Company reports

Johns Hopkins – Hopkins conducted a clinical trial with researchers evaluating the effect of AR-V7 in patients with metastatic CRPC on tumor responsiveness to treatment with Xtandi and Zytiga. In the trial, 31 patients received Xtandi and 31 patients received Zytiga. In the Xtandi-treated group, 12 out of 31 patients were identified as having AR-V7. None of these 12 patients with AR-V7 achieved the trial's primary endpoint of maximal PSA reduction of at least 50%. Eleven of the 12 patients with AR-V7 did not achieve any PSA reduction. Ten of 19 patients who did not have AR-V7 achieved a maximal PSA reduction of at least 50% reduction. The median radiographic progression free survival of patients with AR-V7 was 2.1 months compared to 6.1 months in those without AR-V7. In the Zytiga-treated group, 6 of the 31 patients were identified as having AR-V7. None of the six with AR-V7 achieved any PSA reduction during the treatment. Seventeen of the 25 patients who did not have the AR-V7 achieved a maximal PSA reduction of at least 50%. The median radiographic progression free survival of patients with AR-V7 was 2.3 months and had not yet been reached in the patients without AR-V7 (Exhibit 4).

Exhibit 4: Johns Hopkins Data

			Results				
Treatment	N	AR-V7+	AR-V7 Status	PSA50	p-value*	rPFS	p-value*
Xtandi	31	38% (12/31)	+	0%	0.004	2.1 months	<0.001
			-	52%		6.1 months	
Zytiga	31	19% (6/31)	+	0%	0.004	2.3 months	<0.001
			-	68%		Not Reached	

* Statistically significant as p-value is 0.05 or less

Source: Company reports

Hopkins researchers also tracked the prevalence of AR-V7 based on prior treatment. Based on the data, TKAI believes that treatment with Xtandi and Zytiga may be associated with an increase in the prevalence of AR-V7, causing cross-resistance to sequential therapy and thus leaving patients who were treated with either Xtandi or Zytiga no further secondary hormonal treatment options (Exhibit 5).

Exhibit 5: Prevalence of AR-V7 in CRPC in Johns Hopkins Trial

Treatment Status Prior to Entry Into Johns Hopkins Trial	Percentage of Patients in Pre-Treatment Group who had AR-V7
Pre-enzalutamide (Xtandi) and pre-abiraterone (Zytiga)	11.6%
Post-enzalutamide *only*	25.0%
Post-abiraterone *only*	51.2%
Post-enzalutamide and post-abiraterone	66.7%

Source: Company reports

Galeterone: A Multi-Targeted Androgen Pathway Disruptor:

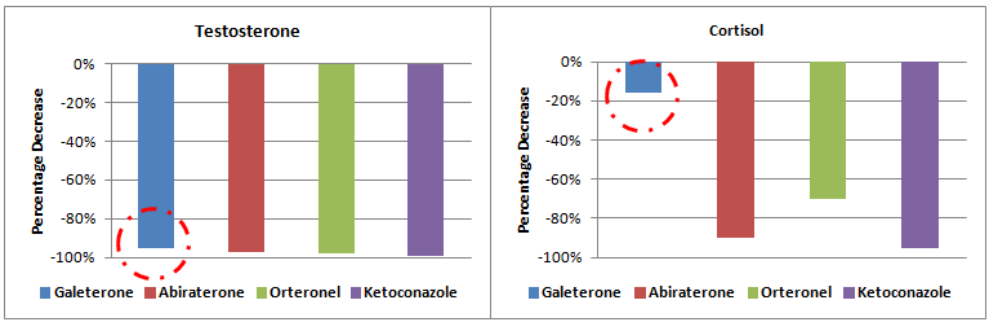
What makes galeterone a unique molecule is that it disrupts the androgen receptor signaling pathway not by one mechanism of action like Xtandi and Zytiga, but by three.

What makes galeterone a unique molecule is that it disrupts the androgen receptor signaling pathway not by one mechanism of action like Xtandi and Zytiga, but by three mechanisms. Galeterone combines CYP17 inhibition, androgen receptor antagonism and androgen receptor degradation.

CYP17 Lyase Inhibition: As with Zytiga, galeterone is an inhibitor of CYP17, a protein with two enzymatic functions: hydroxylase and lyase. As CYP17 plays a central part in synthesizing the androgens which act to fuel tumor growth, CYP17 inhibitors have been developed to treat CRPC. However, inhibition of the CYP17 hydroxylase causes the accumulation of certain steroids including progesterone and corticosterone and a reduction in cortisol. This misbalance can result in mineralocorticoid excess.

An ideal CYP17 inhibitor will selectively block the lyase function of CYP17 relative to hydroxylase so that these steroids do not accumulate to the point of mineralocorticoid excess. In preclinical studies of galeterone and Zytiga, galeterone showed selective blockage of the lyase function of CYP17 relative to the hydroxylase function. In contrast, Zytiga more selectively blocked the hydroxylase function relative to the lyase function (Exhibit 5).

Exhibit 5: CYP17 Lyase Activity



Source: Company reports

Androgen Receptor Antagonism: As with Xtandi, galeterone also blocks androgens from binding to the androgen receptor. This ultimately halts a cascade that eventually decreases the expression of androgen-responsive genes that drive tumor growth. Galeterone has shown potency of antagonism greater than or comparable to other androgen receptor antagonists, like Xtandi, in in vitro studies (Exhibit 6).

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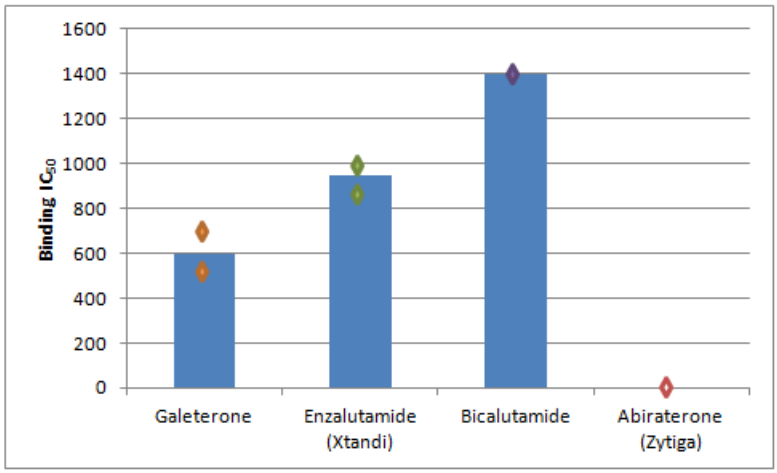
Exhibit 6: Androgen Receptor Antagonism



Source: Company reports

Androgen Receptor Degradation:

Galeterone not only has the same mechanism of action as Xtandi and Zytiga, but it also has a third that enhances the degradation of the androgen receptor itself. This degradation reduces the amount of androgen receptors in a tumor cell to which androgens can bind and decreases the sensitivity of androgen responsive cells to androgen.

The effect of galeterone to reduce androgen receptor levels has been observed in tumor cell lines and a xenograft model in mice. TKAI has observed this effect of galeterone in varying degrees in prostate cancer cell lines across a broad range of full and mutated androgen receptor length as well as other alterations, including the AR-V7 splice variance. In contrast, in both independent preclinical studies and company-run preclinical studies, reductions in androgen receptor levels have not been observed using in vivo or in vitro models of prostate cancer treated with abiraterone (active ingredient in Zytiga), bicalutamide (active ingredient in Casodex) and enzalutamide (active ingredient in Xtandi). Currently, there are no approved drugs or drugs in development other than galeterone with a mechanism of action of androgen receptor degradation.

Galeterone Preclinical Development:

Both in vitro and in vivo preclinical studies have been done to evaluate galeterone's effect on prostate cancer, including efficacy on hormone-sensitive tumor cell-lines, tumors expressing AR-V7 and other splice variances, tumors expressing androgen receptor point mutations and in combination with other novel targeted agents.

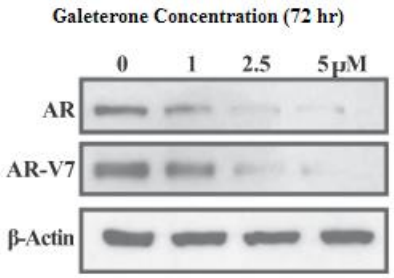
Effective Androgen Receptor Degradation Even with AR-V7 - Androgen receptor splice variants are produced in tumor cells due to an aberrant RNA splicing event. As a result, a truncated androgen receptor protein is synthesized that lacks the C-terminal end of the protein, the region of the protein responsible for androgen binding. Tumor cells that express altered androgen receptors that lack the C-terminal end of the protein are not responsive to agents whose activity requires a functional ligand binding domain.

In addition, the lack of the ligand binding domain causes the remaining splice variants to be constitutively active, or continuously signaling, meaning that

activation of the androgen receptor pathway and tumor growth occurs even in the absence of androgens and androgen binding. This indicates the importance of androgen receptor degradation on the prevention of tumor growth.

In Exhibit 7, results can be seen from a preclinical study in which androgen receptor degradation was measured using cell lines that expressed full-length and splice variant receptors. Results show that levels of both full-length and AR-V7 are reduced in a dose dependent fashion.

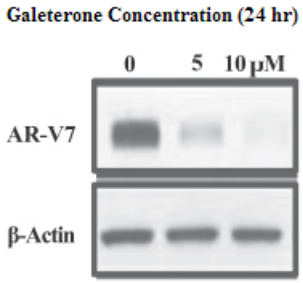
Exhibit 7: Galeterone Causes Decreased Levels of AR-V7 and AR



Source: Company reports

To demonstrate that galeterone would degrade the AR-V7 protein alone, in the absence of the full-length androgen receptor, galeterone was studied in a prostate cancer cell line that only expressed AR-V7 and not the full-length androgen receptor. As shown in Exhibit 8 below, in this study, AR-V7 protein levels were reduced in a dose dependent fashion in cells that only expressed AR-V7 and not the full-length androgen receptor, confirming that galeterone can act directly on the AR-V7.

Exhibit 8: Decreased Levels of AR-V7 Only



Source: Company reports

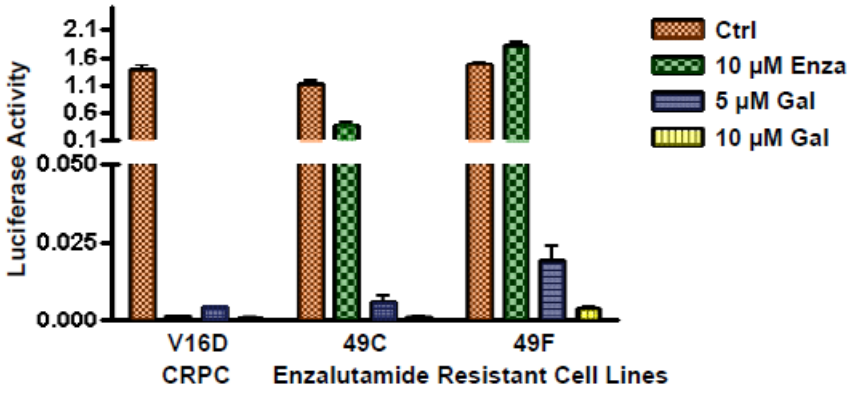
Degradation of Receptors = Reduced Signaling - In addition, with the Vancouver Prostate Center, TKAI examined whether degradation of androgen receptors translated into reduced androgen receptor signaling and reduced tumor growth in the same cells with the AR-V7 variant.

The Vancouver Prostate Centre conducted a series of studies evaluating the anti-tumor activity of galeterone and enzalutamide (active ingredient in Xtandi) in AR-V7 expressing cells. In these studies, galeterone reduced tumor cell proliferation, reduced androgen receptor levels, and decreased nuclear translocation of the androgen receptor, while enzalutamide was only weakly effective in these measures of anti-tumor activity.

In these studies, the effect of galeterone or enzalutamide on androgen responsive gene expression was also evaluated by measuring the activity of luciferase, a fluorescent marker, inserted into tumor cells, with lower luciferase activity indicating greater inhibition of androgen signaling.

As shown in Exhibit 9, in these studies, the tumor cell line that did not express AR-V7 (V16D) had reduced luciferase activity when treated with enzalutamide or galeterone. However, the enzalutamide-resistant tumor cell lines that did express AR-V7 (49C and 49F) only had reduced luciferase activity when treated with galeterone. When treated with enzalutamide, these tumor cells had increased luciferase activity or only a minimal reduction in luciferase activity, indicating a lower inhibition of androgen signaling relative to galeterone in enzalutamide-resistant tumor cells with AR-V7.

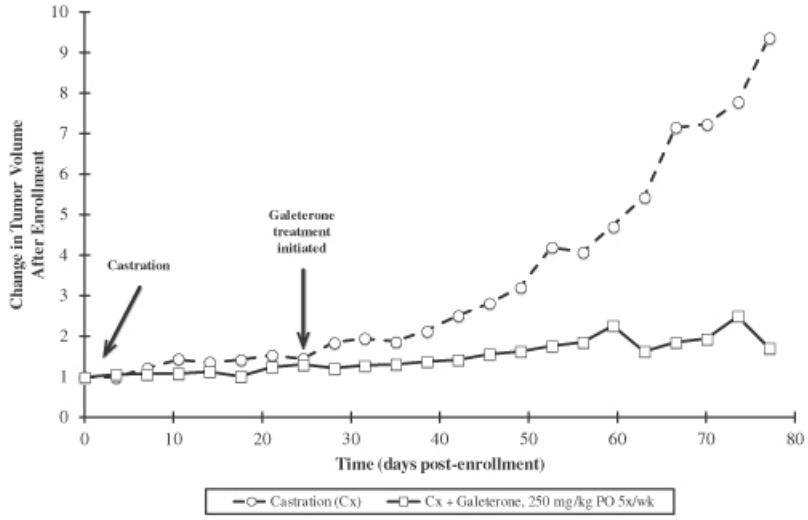
Exhibit 9: Enzalutamide-Resistant Cell Lines



Source: Company reports

Target AR-V7 and Inhibit Tumor Growth – Galeterone was also evaluated in vivo in a LuCaP136 xenograft model of human prostate cancer tumor cells grown in castrated mice. LuCaP136 is a prostate cancer cell line that expresses AR-V7. As shown in Exhibit 10 below, the tumors grew in control animals. However, castrated animals treated with galeterone showed a pronounced tumor growth inhibition.

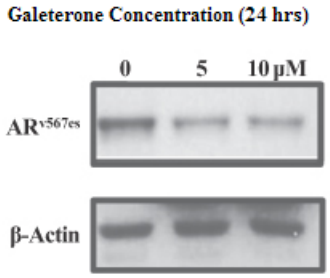
Exhibit 10: Tumor Growth Inhibition



Source: Company reports

Activity in Other Spice Variants (ARv567es) - ARv567es, like AR-V7, is a truncated androgen receptor with C-terminal loss. To demonstrate that galeterone would degrade the ARv567es protein alone, in the absence of a full-length androgen receptor, galeterone was studied in a prostate cancer cell line that only expresses ARv567es, and not the full-length androgen receptor. As shown in Exhibit 11 below, in this study, ARv567es protein levels were reduced in a dose dependent fashion in cells that only express ARv567es and not the full-length androgen receptor, confirming that galeterone can act directly on the ARv567es.

Exhibit 11: Other Splice Variants



Source: Company reports

Galeterone Clinical Development:

The IND for galeterone for the treatment of CRPC was submitted to the FDA in August 2009 and clinical trials were initiated in November 2009. As of May 12, 2014, galeterone had been administered to a total of 234 prostate cancer patients and health volunteers in Phase 1 and Phase 2 clinical trials. In the Androgen Receptor Modulation Optimized Response (ARMOR) program, galeterone has treated 101 CRPC patients in the ARMOR 2 trial and 49 in the ARMOR 1.

ARMOR2 Trial:

The ARMOR 2 trial was initiated in December 2012 as an open label Phase 2. The trial has two parts. The first part is a dose escalation phase designed to confirm the dose of galeterone to be evaluated in part two. Part two is designed to evaluate the safety and efficacy of galeterone in distinct CRPC patient populations.

The primary endpoint of ARMOR2 is based on a decrease in PSA (prostate-specific antigen) levels. This is the standard, accepted marker used to determine if a patient's prostate cancer is responding to therapy. This endpoint has also been used as a key efficacy endpoint in Phase 2 clinical trials of other prostate cancer treatments.

Part 1 of ARMOR2 Trial – Part 1 of the trial enrolled 25 CRPC treatment-naïve patients with progressive disease and three patients whose disease progressed during treatment with Zytiga, or Zytiga-refractory patients. The treatment-naïve patients were enrolled in one of three escalating dose cohorts: 1700 mg/day, 2550 mg/day and 3400 mg/day with the Zytiga-refractory patients all receiving doses of 2550 mg/day. All patients in Part 1 received treatment for up to the initial period of 12 weeks followed by the optimal continued dose for those patients that tolerated the treatment and whose cancer had not progressed.

At least 50% of patients at all dose levels achieved a 30% or greater decrease in PSA. Based on monitoring committee recommendations, the 2550 mg/day dose was chosen to advance into Part 2.

Part 2 of ARMOR2 Trial – Part 2 of the trial is currently ongoing. The 2550 mg/day dose will be evaluated in up to a total of 108 patients in several advanced prostate cancer populations. Other endpoints include safety, tumor response rate and evaluation of C-terminal androgen receptor expression in relation to N-terminal androgen expression. This is summarized in Exhibit 12. As of May 12, 2014 TKAI had enrolled 73 patients in Part 2 of the trial.

Exhibit 12: Trial Details

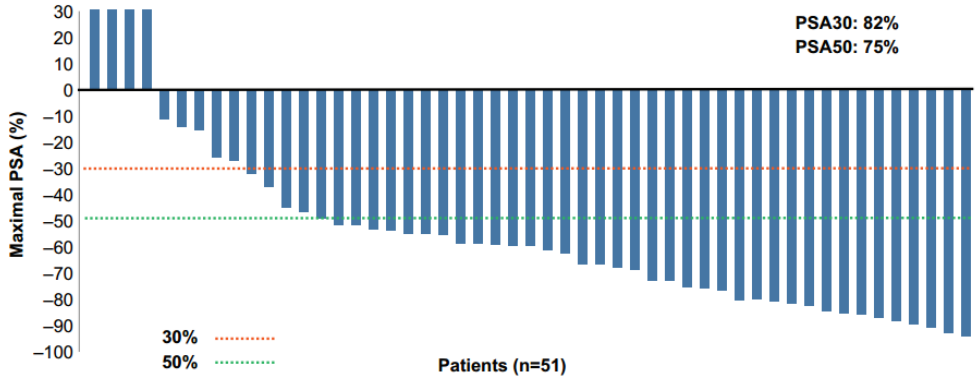
Patient Population	Number of Patient	Primary Endpoint
Non-metastatic CRPC treatment-naïve patients	Up to 48	% of patients with a maximal reduction in PSA levels of at least 30% from baseline to the end of the primary treatment phase
Metastatic CRPC treatmetn-naïve patients		
Zytiga-refractory patients	Up to 30	% of change in PSA levels from baseline to the end of the primary treatment phase
Zxtandi-refractory patients	Up to 31	

Source: Company reports

Clinical Data Presented at ASCO:

In May 2014, interim efficacy and safety data from the ARMOR2 trial were presented. The data were from patients who received the 2550 mg/day dose of galeterone. In 51 evaluable CRPC treatment-naïve patients in Part 1 and Part 2 of the trial, during the first 12 weeks of dosing, 82% had a maximal reduction in PSA levels of at least 30% and 75% had a maximal reduction in PSA levels of at least 50%. The results can be seen in the waterfall plot in Exhibit 13.

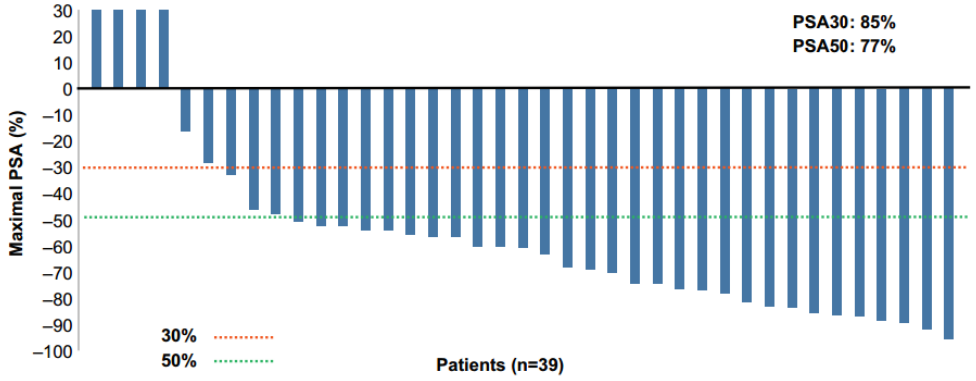
Exhibit 13: Waterfall Plot



Source: Company reports

In the 39 metastatic CRPC treatment-naïve patients who received the 2550 mg/day dose, during the first 12 weeks of dosing, 85% had a maximal reduction in PSA levels of at least 30% and 77% had a maximal reduction in PSA levels of at least 50%. These results can be seen in the waterfall plot in Exhibit 14.

Exhibit 14: Maximal PSA Response in Pre-Chemo Metastatic CRPC Treatment-Naïve Patients



Source: Company reports

Zytiga-Refractory: The 12-week data from the Zytiga-refractory patients were also reported. Two patients achieved a maximal reduction in PSA levels of at least 30% and of the eight who were evaluable as Response Evaluation Criteria in Solid Tumors (RECIST), five had stable disease and three patients had progressive disease.

C-Terminal Loss: Other data that were presented at ASCO involved a retrospective subset analysis. This analysis involved four treatment-naïve CRPC patients in ARMOR2 that were identified as having C-terminal loss as determined by the evaluation of C-terminal androgen receptor expression in relation to N-terminal androgen receptor expression. All four patients achieved maximal reductions in PSA levels of at least 50%. TKAI believes that these data support the view that androgen receptor degradation may be active in patients without the intact ligand binding domain of the C-terminal binding site.

Safety Profile: Of the 87 patients that had been treated with galeterone as of May 14th, the drug was well tolerated. Approximately 90% of all treatment-emergent adverse events reported were grade 1 or 2 in severity and were generally manageable and reversible. There were no reported cases of seizure or mineralcorticoid excess. The most common adverse events were nausea, decreased appetite, fatigue, diarrhea, pruritus and increased aminotransferase indicating elevated liver enzyme levels.

Six of these patients (7%) experienced a grade 3 or 4 treatment-emergent increase in aminotransferase. These symptoms were asymptomatic, transient and all six patients recovered following a temporary drug withdrawal. Four of the six patients were re-challenged at a reduced dose with none showing a recurrence of a grade 3 or higher adverse event.

There were three unexpected serious adverse events that were possible related to treatment with galeterone. One was a case of angioedema in a patient who was taking medications associated with angioedema. Another was an episode of dizziness and fainting in a patient with a history of nausea, diabetes and hypertension who discontinued treatment four days prior to the episode and lastly, a case of hypocalcemia and hyperparathyroidism in a patient with a history of both.

Clinical Data Presented at EMSO 2014:

At ESMO 2014, the company presented additional data in the M0/M1 treatment-naïve population. In the M0/M1 treatment naïve group at 2550 mg/day (n=60), there was an 83% PSA30 and 70% PSA50. In the M1 treatment naïve group at 2550 mg/day (n=39), there was an 85% PSA30 and a 77% PSA50. The safety profile was consistent with that seen previously (N= 107). A total of 7 naïve CRPC patients had C-terminal loss and 6 out of 7 patients had PSA50. A single non-responsive patient discontinued early for unrelated event and did not receive full dosing.

ARMOR1 Trial

The ARMOR 1 trial was initiated in November 2009 as an open label, dose escalation Phase 1 clinical trial, enrolling 49 metastatic and non-metastatic CRPC treatment-naïve patients in eight sites across the US.

Patients were enrolled in eight cohorts based on dose level and dosing schedule. Escalating doses were administered from 650 mg/day through 2600 mg/day as a single daily dose or a split dose twice daily. Based on patient preference, galeterone was taken with a meal or with a food supplement. Patients received treatment for an initial 12-week period followed by optional continued dosing. Treatment was continued until disease progression or patient withdrawal due to adverse events or other reasons.

Safety results from ARMOR1: Overall, galeterone was well tolerated. Patients, as an aggregate were dosed with galeterone, for approximately 8,000 days, with individual patients receiving galeterone for up to 20 months. Approximately 90% of treatment-emergent adverse events reported for the first 12 weeks of treatment were grade 1 or grade 2 in severity and were generally manageable and reversible. The majority were assessed as not related or unlikely related to galeterone. The most common treatment-emergent adverse events reported for the first 12 weeks of treatment were fatigue, increased aminotransferase, nausea, diarrhea and pruritus. The incidence of treatment-emergent adverse events was comparable between

cohorts and was not dose related. A total of eight patients (or 16%) experienced a grade 3 treatment-emergent increase in aminotransferase indicating elevated liver enzyme levels. Similar to the ASCO data, these events were asymptomatic and transient. Of the eight patients, two patients voluntarily withdrew from the trial, and six patients restarted at the same dose level or one dose level below with no recurrence of a grade 3 or higher adverse event. A maximum tolerated dose was not reached in the trial. In the ARMOR1 trial, there was only one unexpected serious adverse event assessed by the investigator as possibly related to treatment with galeterone: a case involving a 77-year old patient who developed rhabdomyolysis, an acute disintegration of muscle tissue, and acute renal failure that occurred while receiving simvastatin, a statin known to be associated with rhabdomyolysis. In addition, the patient had underlying chronic renal insufficiency, renal artery stenosis and hydronephrosis requiring stents and presented after a fall, all of which are risk factors for either acute renal failure or rhabdomyolysis.

Efficacy results from ARMOR1: Patients in each of the doses tested experienced reductions in PSA. In the 12 patients who received the highest dose in the study, 2600 mg/day, maximal PSA decreases of at least 30% were observed in 75% of the patients, and maximal PSA decreases of at least 50% were observed in 42% of the patients. Of the 49 patients in the trial, 22% experienced maximal PSA decreases of at least 50%, and 49% experienced maximal PSA decreases of at least 30%. While favorable, TKAI believes that they were adversely affected by the exposure variability associated with the food effect of the PIC formulation. Radiographic evidence of tumor shrinkage and overall tumor stabilization was seen in multiple patients as assessed by CT/MRI scans and bone scans as measured by RECIST. Thirty-nine patients had measurable disease at baseline, including five patients receiving the 2600 mg/day dose. Of the five patients, two had partial responses, and a third patient had a near partial response with a reduction in maximal PSA levels of 28%. Of the 39 patients, 22 had stable disease at the end of the 12-week treatment period.

Development Plans for Galeterone:

The strategic plan for galeterone is to initiate a registration study in AR splice variants and then expand the indications. In order to progress to the planned Phase 3 pivotal trials, TKAI will need to develop assays that detect C-terminal loss or AR-V7. TKAI currently plans to contract with third parties to develop these assays and to use other widely available methodologies and technologies, which should de-risk the development plan. TAKI is currently exploring developing an assay as an in vitro companion diagnostic test.

We believe galeterone is well-positioned to address the unmet medical need in CRPC patients given 1) positive Phase 2 data in C-terminal loss and supportive preclinical data; 2) significant patient exposure data in CRPC with greater than17,000 patients days; 3) completion of end-of-Phase 2 and pre-Phase 3 meetings; 4) finalization of pivotal trial details in M1 treatment naïve patients (n≤170 patients; rPFS primary endpoint); 5) CTC AR-V7 assay development underway; and 6) completion of clinical trial material production. The company intends to initiate a pivotal trial in 1H15 with top-line results available by YE:16. The company plans to expand use of galeterone into additional prostate cancer and other AR-driven indications to broaden its commercial potential.

Intellectual Property:

As of July 31, 2014, TKAI owned two issued U.S. patents, ten U.S. provisional and non-provisional patent applications, one issued foreign patent and 34 foreign applications in the galeterone patent portfolio. TAKI also has rights under a license agreement with University of Maryland, Baltimore (UMB) to five issued U.S. patents and 44 issued foreign patents as well as three U.S. patent applications and 13 foreign applications. The owned and licensed patent and patent applications, if issued, are expected to expire on various dates from 2017 through 2034, without taking into account any possible patent term extensions.

Though the initial compositions and methods of use of a class of compounds encompassing galeterone expires in 2017, TKAI has filed for or has acquired licensed patents and patent applications relating to galeterone covering methods of use, pharmaceutical compositions, combination treatments, prodrugs, metabolites, and analogs of galeterone and their use. Even though TKAI has no patent protection specifically covering the chemical structure of galeterone, these additional applications and patents should protect the asset within the CRPC therapeutic area.

Method of Use: TKAI has licensed from UMB a U.S. patent covering a method of treating prostate cancer in a human subject by administering galeterone, which is expected to expire in 2027. The license also includes granted patents in the European Patent Convention and Japan covering the use of galeterone to treat prostate disease, including prostate cancer and prostatic hyperplasia. Similar patents have been granted or allowed in Australia, Canada, Hong Kong, South Korea, Mexico, New Zealand, Singapore, South Africa, and the Eurasian Patent Organization. These patents are expected to expire in 2026. In addition, we have pending applications in Brazil, China, the European Patent Convention, India, Israel, Indonesia and Japan.

TKAI has also filed three U.S. provisional patent applications covering the use of galeterone in treating Xtandi-resistant prostate cancer mediated by androgen receptor variants, including splice variants such as AR-V7. The term of a patent, if issued, claiming priority to these provisional applications would be expected to expire in 2034.

Pharmaceutical Compositions: TKAI has filed U.S. and international patent applications relating to a galeterone formulation and its use where the galeterone is present in a spray dried dispersion. TKAI has pending applications in the United States, the European Union, Australia, Brazil, Canada, China, India and Japan. The term of any patent in this family, if issued, would be expected to expire in 2032. In addition, TKAI has licensed from UMB a U.S. patent application covering a pharmaceutical composition of galeterone. The term of any patent, if issued, claiming priority to this application would be expected to expire in 2026.

Combination Treatments: TKAI has filed patent applications or licensed from UMB patent applications covering the use of galeterone in combination with other therapeutic drugs. For example, TKAI has filed U.S. and foreign patent applications covering the use of galeterone in combination with inhibitors of the Akt/PI3K pathway. TKAI has pending applications in the United States, the European Union, Australia, Canada and Japan. The term of any patent in this family, if issued, would be expected to expire in 2032.

Prodrugs, Metabolites and Analogs: TKAI has filed patent applications or licensed from UMB patent applications directed to prodrugs, metabolites or analogs of galeterone. For example, TKAI has licensed a U.S. patent application from UMB directed to certain prodrugs of galeterone. If issued, the term of the resulting patent,

if issued, would be expected to expire in 2029. TKAI has also filed patent applications in the United States and certain other countries including Australia, Brazil, Canada, China, the European Union, India and Japan directed to other prodrugs of galeterone. If issued, the term of the resulting patents would be expected to expire in 2030. Further, TKAI has filed patent applications in the United States and certain other countries including Australia, Brazil, Canada, China, the European Union, India and Japan directed to compounds which have been identified as metabolites of galeterone and which may be biologically active. If issued, the term of the resulting patents would be expected to expire in 2030. TKAI has also obtained a license to a UMB PCT patent application directed to analogs of galeterone that disrupt androgen receptor signaling by degrading the androgen receptor. The term of any patent, if issued, claiming priority to this PCT patent application would be expected to extend to 2034.

Management:

Jodie Morrison - President & CEO: Ms. Morrison has served as president and chief executive officer at Tokai and as a member of the board of directors since March 2013. Prior to her appointment, Jodie served as both the company's chief operating officer and head of clinical affairs over the seven preceding years. Throughout her tenure at Tokai, Jodie has led the company's operational management and galeterone prostate cancer development program. Prior to joining Tokai, Jodie was director of clinical operations and medical affairs at Dyax Corporation. During her tenure at Dyax, she built and led the clinical development teams for Kalbitor (hereditary angioedema) and DX-88 (cardiothoracic surgery), and oversaw the Kalbitor clinical trials that ultimately led to its marketing approval. Prior to joining Dyax, she held clinical management positions at both Curis, Inc. and at Diacrin, Inc. She received a B.A. in neuroscience from Mount Holyoke College, her business training through the Greater Boston Executive Program at MIT Sloan School of Management and her clinical research certification from Boston University School of Medicine.

Lee Kalowski - Chief Financial Officer: Mr. Kalowski has served as chief financial officer at Tokai since September 2014. Prior to joining Tokai, he served as a vice president in global biotechnology equity research at Credit Suisse. In this role, Lee served as a senior research analyst covering the biotechnology industry, including numerous companies globally in the prostate cancer therapeutic area. Prior to Credit Suisse, Lee worked at Johnson & Johnson in mergers & acquisitions in the pharmaceutical group, where he was involved in the analysis and execution of several completed transactions, and in global pharmaceutical equity research at Sanford C. Bernstein and Prudential Equity Group. He holds a B.A., Phi Beta Kappa, in biology and economics, from Union College and an M.B.A. in finance and health care management from the Wharton School, University of Pennsylvania.

John McBride – Chief Operating Officer & Chief Financial Officer: Mr. McBride has served as chief operating officer since February 2014 and as chief financial officer at Tokai since April 2014. Prior to joining Tokai, he founded and served as president of Alliance Life Science Advisors, Inc., a consulting firm focused on assisting life science companies with strategic planning, business development and financing projects. Prior to that, John was executive vice president and chief operating officer of Gloucester Pharmaceuticals, Inc. where he was responsible for the company's business development, finance, administrative and manufacturing functions. He has also served as global head of oncology licensing at Pharmacia Corporation; executive vice president, business operations and chief financial officer at

CytoTherapeutics, Inc.; vice president, business development and treasurer at Phytera, Inc.; vice president, commercial development at Sparta Pharmaceuticals, Inc.; and vice president, business development at U.S. Bioscience, Inc. He holds a B.S. in biochemistry and an M.S. in chemical engineering from the University of Wisconsin and an M.B.A. from the Wharton School, University of Pennsylvania

Karen Ferrante, M.D., – Head of R&D and Chief Medical Officer: Dr. Ferrante has served as head of research and development and chief medical officer at Tokai since April 2014. Prior to Tokai, she served as oncology therapeutic area head for Takeda Pharmaceuticals and Takeda Cambridge, USA site head. Prior to that, Karen held senior positions at Millennium Pharmaceuticals and its parent company, Takeda Pharmaceuticals including her role as chief medical officer and a head of R&D for Millennium, The Takeda Oncology Company. From 1999 to 2007, she held positions of increasing responsibility at Pfizer Global Research & Development, culminating as vice president and therapeutic area clinical leader in oncology development. Karen began her career in the pharmaceutical industry as associate director of clinical oncology at Bristol-Myers Squibb Company. For more than a decade prior, she was at the New England Deaconess Hospital in Boston (Beth Israel Deaconess), where she completed her internship and residency in internal medicine followed by her fellowship in hematology and oncology. While at the Beth Israel Deaconess Hospital, she served as instructor, clinical instructor and clinical fellow in medicine at the Harvard Medical School. Karen has been an author of a number of peer-reviewed papers in the field of oncology, an active participant in academic and professional associations and symposia, is the holder of several patents and serves as a member of the board of Progenics Pharmaceuticals. She holds a B.S. in chemistry and biology from Providence College and an M.D. from Georgetown University.

Susan Steward– Head of Regulatory Affairs, Quality and Compliance: Ms. Steward has served as head of regulatory affairs at Tokai since 2010. Prior to joining Tokai, Sue was vice president of regulatory affairs at TransMolecular, Inc. where she also led strategic planning and IP management for its oncology programs. Prior to joining TransMolecular, Sue was vice president of regulatory affairs for Genzyme Corporation, and in parallel was a director for MG Biotherapeutics, a joint venture of Medtronic Inc. and Genzyme. While at Genzyme, Sue was the regulatory lead on novel drug development collaborations with Diacrin, Inc., Cambridge Antibody Technologies and Dyax, Inc. Prior to joining Genzyme, she served as quality assurance supervisor for Abbot Laboratories. Sue received a B.A. from Annhurst College and a J.D. from Concord Law School. She is a fellow of the Regulatory Affairs Professionals Society (FRAPS), and is both U.S. and European Union Regulatory Affairs Certified (RAC).

Tokai Pharmaceuticals, Inc. (NASDAQ: TKAI)

Income Statement

(In thousands, except per share data)

	2012 A	2013 A	6-Months Ending Q2:14 A	For the Quarter Ending Q3:14 E	For the Quarter Ending Q4:14 E	2014 E	For the Quarter Ending Q1:15 E	For the Quarter Ending Q2:15 E	For the Quarter Ending Q3:15 E	For the Quarter Ending Q4:15 E	2015 E
Revenue:											
Collaboration / Milestones	-	-	-	-	-	-	-	-	-	-	-
Product Revenue	-	-	-	-	-	-	-	-	-	-	-
Total Revenue	-	-	-	-	-	-	-	-	-	-	-
Cost of Product Sales	-	-	-	-	-	-	-	-	-	-	-
Gross Profit	-	-	-	-	-	-	-	-	-	-	-
Cost and Expenses:											
Research and Development	7,370	12,201	7,948	3,974	3,974	15,896	6,165	6,165	6,165	6,165	24,661
Sales & Marketing	-	-	-	-	-	-	-	-	-	-	-
General and Administrative	2,279	3,548	2,829	1,415	1,415	5,658	812	812	812	812	3,249
Total Costs and Expenses	9,649	15,749	10,777	5,389	5,389	21,554	6,978	6,978	6,978	6,978	27,910
Operating Income (Loss)	(9,649)	(15,749)	(10,777)	(5,389)	(5,389)	(21,554)	(6,978)	(6,978)	(6,978)	(6,978)	(27,910)
Other Income (Expense):											
Interest Income	-	-	-	44	72	116	68	64	59	54	245
Change in Fair Value of Warrants	-	-	-	-	-	-	-	-	-	-	-
Other	(34)	(70)	79	-	-	79	-	-	-	-	-
Loss Before Income Taxes	(9,683)	(15,819)	(10,698)	(5,344)	(5,316)	(21,359)	(6,909)	(6,914)	(6,919)	(6,924)	(27,665)
Income Taxes	-	-	-	-	-	-	-	-	-	-	-
Net Income	$ (9,683)	$ (15,819)	$ (10,698)	$ (5,344)	$ (5,316)	$ (21,359)	$ (6,909)	$ (6,914)	$ (6,919)	$ (6,924)	$ (27,665)
Basic Earnings Per Share	$ (31.09)	$ (1.29)	$ (0.70)	$ (0.29)	$ (0.24)	$ (1.20)	$ (0.31)	$ (0.31)	$ (0.31)	$ (0.31)	$ (1.25)
Diluted Earnings Per Share	$ (31.09)	$ (1.29)	$ (0.70)	$ (0.29)	$ (0.24)	$ (1.20)	$ (0.31)	$ (0.31)	$ (0.31)	$ (0.31)	$ (1.25)
Basic Shares Outstanding	311	12,230	15,358	18,600	21,842	17,790	21,992	22,142	22,292	22,442	22,217
Diluted Shares Outstanding	311	12,230	15,358	18,600	21,842	17,790	21,992	22,142	22,292	22,442	22,217
Effective Tax Rate	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
EBITDA Calculation:											
Loss Before Income Taxes	$ (9,683)	$ (15,819)	$ (10,698)	$ (5,344)	$ (5,316)	$ (21,359)	$ (6,909)	$ (6,914)	$ (6,919)	$ (6,924)	$ (27,665)
Less: Interest Income	-	-	-	(44)	(72)	(116)	(68)	(64)	(59)	(54)	(245)
Plus: Depreciation	-	-	-	-	-	-	4	11	19	23	56
EBITDA	$ (9,683)	$ (15,819)	$ (10,698)	$ (5,389)	$ (5,389)	$ (21,475)	$ (6,974)	$ (6,966)	$ (6,959)	$ (6,955)	$ (27,854)
Margins:											
Gross	N/M	N/M	N/M	N/M	N/M	N/M	N/M	N/M	N/M	N/M	N/M
Operating	N/M	N/M	N/M	N/M	N/M	N/M	N/M	N/M	N/M	N/M	N/M
Net Income (Loss)	N/M	N/M	N/M	N/M	N/M	N/M	N/M	N/M	N/M	N/M	N/M
EBITDA	N/M	N/M	N/M	N/M	N/M	N/M	N/M	N/M	N/M	N/M	N/M
Growth Rates:											
Total Revenue		N/M	N/M	N/M	N/M	N/M	N/M	N/M	N/M	N/M	N/M
Operating Income		N/M	N/M	N/M	N/M	N/M	N/M	N/M	N/M	N/M	N/M
Net Income (Loss)		N/M	N/M	N/M	N/M	N/M	N/M	N/M	N/M	N/M	N/M
Research and Development Expense		65.5%	N/M	N/M	N/M	30.3%	N/M	-22.4%	55.1%	55.1%	55.1%
Selling, General and Administrative Expense		55.7%	N/M	N/M	N/M	59.5%	N/M	-71.3%	-42.6%	-42.6%	-42.6%

Source - Company reports and Janney Montgomery Scott LLC estimates

RESEARCH ANALYST CERTIFICATIONS AND IMPORTANT DISCLOSURES ARE ON PAGES 20 - 22 OF THIS REPORT

IMPORTANT DISCLOSURES

Research Analyst Certification

I, Kimberly Lee, the Primarily Responsible Analyst for this research report, hereby certify that all of the views expressed in this research report accurately reflect my personal views about any and all of the subject securities or issuers. No part of my compensation was, is, or will be, directly or indirectly, related to the specific recommendations or views I expressed in this research report.

Janney Montgomery Scott LLC ("Janney") Equity Research Disclosure Legend

Tokai Pharmaceuticals, Inc. currently is, or during the past 12 months was, a Janney Montgomery Scott LLC client. Janney Montgomery Scott LLC, provided investment banking related services.

Janney Montgomery Scott LLC managed or co-managed a public offering of securities for Tokai Pharmaceuticals, Inc. in the past 12 months.

Janney Montgomery Scott LLC received compensation for investment banking services from Tokai Pharmaceuticals, Inc. in the past 12 months.

Janney Montgomery Scott LLC intends to seek or expects to receive compensation for investment banking services from Tokai Pharmaceuticals, Inc. in the next three months.

The research analyst is compensated based on, in part, Janney Montgomery Scott's profitability, which includes its investment banking revenues.

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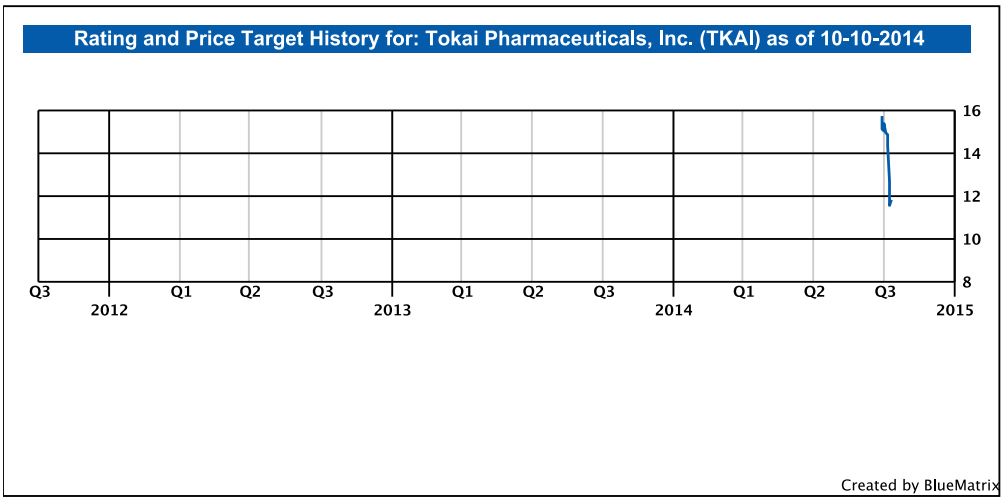
BUY: Janney expects that the subject company will appreciate in value. Additionally, we expect that the subject company will outperform comparable companies within its sector.

NEUTRAL: Janney believes that the subject company is fairly valued and will perform in line with comparable companies within its sector. Investors may add to current positions on short-term weakness and sell on strength as the valuations or fundamentals become more or less attractive.

SELL: Janney expects that the subject company will likely decline in value and will underperform comparable companies within its sector.

Price Charts



Janney Montgomery Scott Ratings Distribution as of 6/30/14

			IB Serv./Past 12 Mos.	
Rating	**Count**	**Percent**	**Count**	**Percent**
BUY [B]	207	53.80	53	25.60
NEUTRAL [N]	176	45.70	28	15.90
SELL [S]	2	0.50	0	0.00

***Percentages of each rating category where Janney has performed Investment Banking services over the past 12 months.**

Other Disclosures

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