CANACCORD Adams

Equity Research

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BUY

AVEO : NASDAQ : US$9.39

TARGET PRICE: US$14.00

Inside

Life Sciences -- Biotechnology

AVEO Pharmaceuticals

Best-in-class cancer drug moves forward; initiating coverage with a BUY

Tivozanib could be top in RCC. Driving most of AVEO's value, in our view, is its lead product, tivozanib, a small molecule inhibitor of a critical signaling pathway implicated in tumor growth. Results from a randomized discontinuation Phase II trial released last year indicate that the clinical activity of tivozanib in renal cell carcinoma (RCC) appears to be at least equivalent to that of the market-leading therapeutic in this space, Sutent (sunitinib; Pfizer).

Phase III results in 2012. Going head-to-head against another approved RCC therapeutic, Nexavar, in a pivotal Phase III registration trial could be sufficient to convince practitioners to use tivozanib as drug of choice in the front-line RCC treatment setting. Pharmacokinetic and pharmacodynamic advantages of tivozanib (highlighted in our report) support this claim. We expect results in 2012.

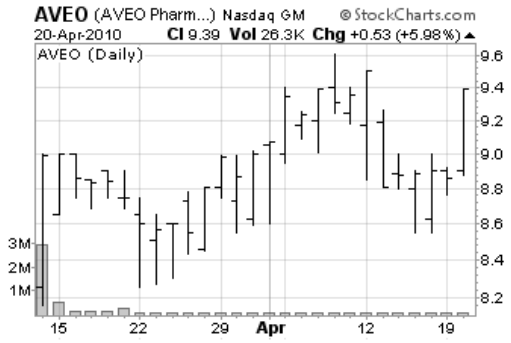
Pipeline and platform. Mitigating investment risk, other targeted therapies in AVEO's pipeline have potential given their genesis based on established platforms, but we believe no meaningful data supports clinical success at this time. AVEO has also teamed up with other players in the oncology development space that we believe are attracted to the sophisticated murine cancer model to which AVEO owns rights for drug discovery efforts. This model could prove to be an extremely valuable asset.

$14 price target. In our view, the $1.0B in 2009 global Sutent sales represents a reasonable standard for tivozanib to potentially attain. Moreover, ownership of all worldwide rights (ex-Japan) makes eventual partnering prospects very attractive. Modeling for what we expect would be an aggressive capture of Sutent market share in RCC beginning in 2013 if data from TIVO-1 recapitulates what was observed in Phase II, we arrive at a $14 price target based on a risk-adjusted DCF model. We initiate coverage of AVEO with a BUY rating.

Company Statistics

Shares Out (M):	27.6
Market Cap (M):	US$259.2
52-week Range:	8.16 - 9.61
Avg. Daily Vol. (000s):	182.2

Price Chart



Earnings Summary

FYE Dec		2009A	2010E	2011E
Revenue (M):		20.7	33.6	22.5
EPS:		(27.43)	(1.44)	(1.42)
Revenue (M):	Q1	-	5.0	-
	Q2	-	18.5	-
	Q3	-	5.0	-
	Q4	-	5.0	-
Total		20.7	33.6	22.5
EPS:	Q1	-	(0.47)	-
	Q2	-	(0.00)	-
	Q3	-	(0.47)	-
	Q4	-	(0.49)	-
Total		(27.43)	(1.44)	(1.42)

Company Description

Aveo Pharmaceuticals is a biotechnology company devoted to the discovery and clinical development of oncologic therapeutics.

INVESTMENT SUMMARY

AVEO Pharmaceuticals is a biotechnology company focused on the discovery and development of agents for treatment of oncologic indications. Driving most of the company's value, in our view, is its lead product, tivozanib, a small molecule inhibitor of a critical signaling pathway implicated in tumor growth. Results from a randomized discontinuation Phase II trial released last year indicate that tivozanib has potent clinical activity in a renal cell carcinoma (RCC) treatment setting that appears to be at least equivalent to that of the market-leading small molecule therapeutic in this space, Sutent (sunitinib; Pfizer).

Once regarded as a tumor type unresponsive to conventional therapeutics, advanced RCC can now be treated with relative success although no therapy can be regarded as curative. Supplanting traditional cytokine therapeutics, small molecule targeted therapeutics falling into two distinct classes – VEGF receptor (VEGR) and mTOR inhibitors – dominate as treatments of choice. Tivozanib belongs in the former class. Differences among class members rely primarily on relative potency (based upon improvements over placebo or prior standard of care) and tolerability.

Although it is a crowded market, we believe the combined efficacy and safety profile of tivozanib highly supports its potential as the eventual market leader in the RCC treatment setting. However, in our view, tivozanib faces a higher regulatory hurdle than its established competitors by virtue of being evaluated against an active comparator in a pivotal trial (the three other drugs, Sutent, Nexavar, and Votrient, were all evaluated compared to placebo or cytokine therapy). This presents a unique challenge for AVEO, in our view, since powering assumptions for a Phase III trial already under way (TIVO-1) are based upon historical data collected at a time when much less was understood about VEGFR inhibitor therapy. Nevertheless, we believe the chance of failure is significantly mitigated by 1) the highly convincing results from the randomized discontinuation Phase II trial confirming tivozanib activity, 2) the pharmacokinetic and pharmacodynamic advantages of tivozanib, and 3) a clear appreciation of what to expect from the Phase III comparator (Nexavar).

Mitigating investment risk, other targeted therapies in AVEO's pipeline have potential given their genesis based on established platforms, but for now no meaningful data supports clinical success at this time, in our view. AVEO has also teamed up with other players in the oncology development space, including Merck, Biogen IDEC, and OSI Pharma, which have made equity investments in the company and/or provide research funding. We believe these partners are attracted to the sophisticated murine cancer model to which AVEO owns rights for drug discovery efforts and which could prove to be an extremely valuable asset.

In our view, the $1.0B in 2009 global Sutent sales represents a reasonable standard for tivozanib to potentially attain. Moreover, AVEO owns 100% of the worldwide rights (ex-Japan) to this drug, which makes eventual partnering prospects very attractive. Modeling for what we expect would be an aggressive capture of Sutent market share in RCC beginning in 2013 if data from TIVO-1 recapitulates what was observed in Phase II, we arrive at a $14 price target based on a risk-adjusted DCF model. We initiate coverage of AVEO with a BUY rating on shares.

UPCOMING EVENTS

Figure 1: AVEO expected upcoming events

Event	Expected timing
Follow-up data from clinical pipeline programs	Q2/10
Overall survival results from the Phase II RDT tivozanib trial	Q1/11
Completion of TIVO-1 enrollment	2011
TIVO-1 results	2012

Source: Company reports and Canaccord Adams

PIPELINE

Figure 2: AVEO pipeline

Drug	Target	Partner	indication	Stage
Tivozanib	VEGFRs	None	RCC	Phase III
			Solid tumors	Phase I
AV-299	HGF-1	Merck	NSCLC	Phase II
			Solid tumors	Phase I
AV-203	ErbB3	Biogen IDEC	Solid tumors	Phase I

Source: Company reports

BUSINESS MODEL

AVEO Pharmaceuticals is devoted to the discovery and development of small molecule and biologic therapeutics for the treatment of oncology indications. Molecules are either procured from other parities (e.g., tivozanib was discovered at Kirin Pharmaceuticals) or discovered in-house. AVEO also fosters relationships with other life science companies to collaborate on the discovery and development of other product candidates and is entitled to future economics on sales and payments upon reaching specific development milestones.

TARGETED THERAPEUTICS

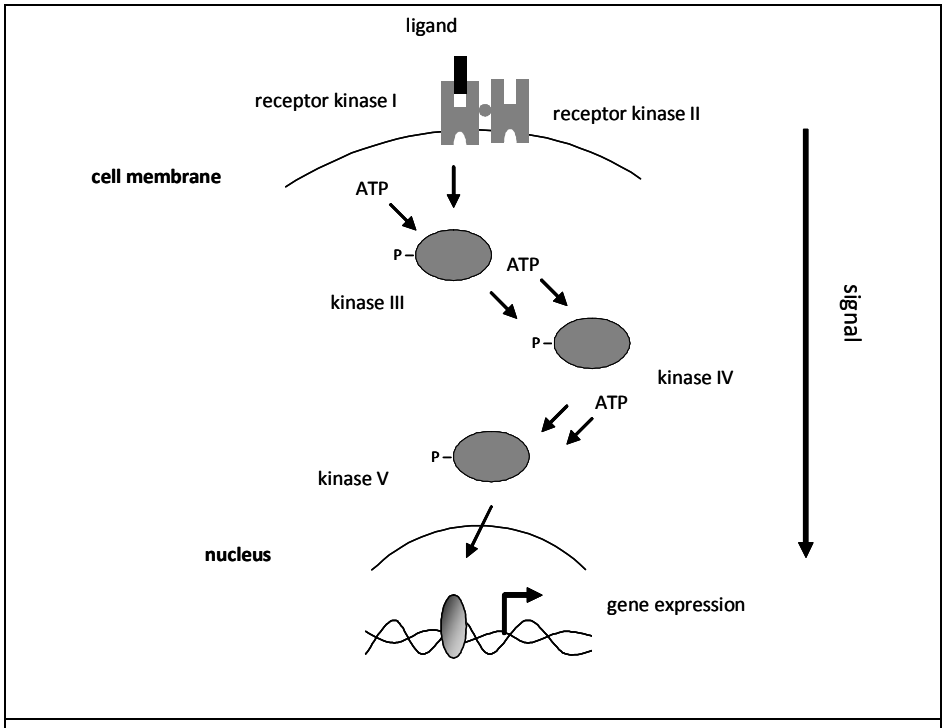
All of AVEO's products fall into the category of targeted oncology therapeutics designed to neutralize specific molecules either overexpressed in various tumors and/or implicated in aberrant cell growth. By virtue of their selectivity, targeted therapeutics theoretically provide efficacy while sparing normal tissue from the heavy collateral damage associated with older treatment modalities.

Falling into three main classes, targeted therapeutics can be defined as follows.

Synthetic small molecule kinase inhibitors designed to block signaling pathways involved in cell growth regulation

Regulation of cell growth and metabolism is controlled in large part by specialized enzymes known as kinases that "communicate" cell growth signals through the sequential chemical modification of pathway partners. For example, as illustrated in Figure 3, receptor kinase I, which may be a ligand receptor residing at the cell surface, will pair up with receptor kinase II, which "activates" kinase III lying deeper in the cell, which activates kinase IV, then V, etc., until the last protein in the chain activates relevant gene expression. Pharmacologic inhibition of any kinase on a given signal pathway, therefore, would theoretically block communication along the entire chain.

Figure 3: Simplified model of kinase-mediated signal transduction



Kinases work along a signal cascade via sequential modification of pathway members using adenosine triphosphate (ATP) as a substrate followed by activation of gene expression leading to cell growth. Inhibition of any kinase along a given pathway should theoretically block signaling along the entire pathway and block cell growth.

Source: Canaccord Adams

Biologics (often monoclonal antibodies) prepared from cell culture that bind targets expressed on tumor cell surfaces or inactivate soluble ligands

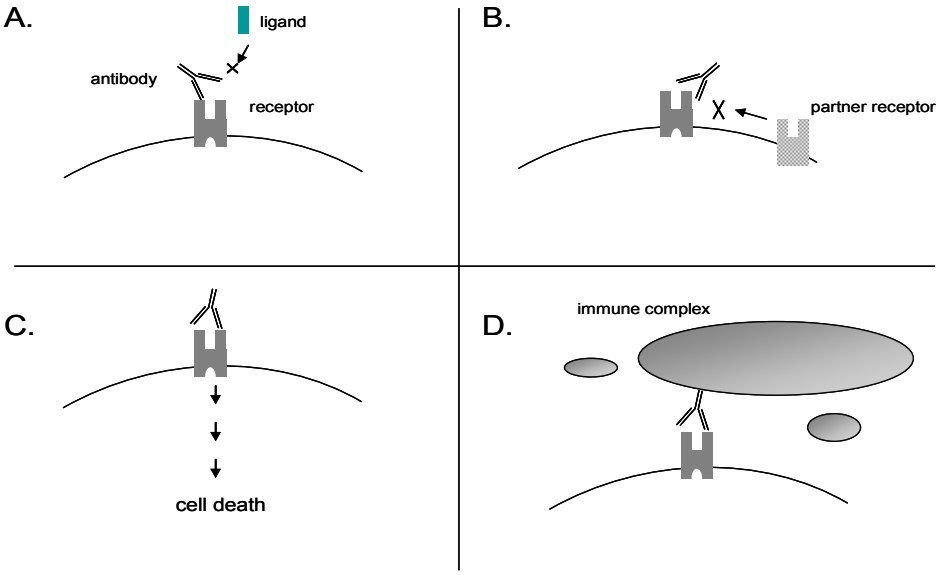
With relative ease, monoclonal antibodies can be produced to bind virtually any target with elegant specificity, whether free floating or associated with a particular pathogen or cancer cell. Clinical development of monoclonal antibodies is usually free of problems often associated with traditional small molecules, including unavoidable, undesirable, and off-target effects.

Due mostly to improvements in building more "human-like" antibodies through chimerization, humanization, and fully human antibody platforms, enormous advances have been made with therapeutic antibodies over the past decade. We believe that this trend has only just begun to emerge, with such agents already demonstrating success as treatments for cancer and autoimmune and inflammatory diseases. For example, Remicade, the largest revenue-producing therapeutic antibody, generated sales of about $6.6 billion in 2009 as a treatment for rheumatoid arthritis, Crohn's disease and other autoimmune disease indications.

By virtue of their elegant specificity, antibodies raised against specific cell surface receptors are not nearly as target promiscuous as small molecules, and in some cases may be more suitable given specific targets and tumor types being treated. While target overlap exists between the antibodies and small molecules, we believe mechanisms behind clinical effect are quite distinct. For example, receptor-targeted antibodies exert their activity through highly specific binding of cell receptors, which can have one or more downstream effects:

1. block receptor interaction with known ligands,
2. block receptor interaction with other cell surface receptors,
3. activate one or more cell death signaling pathways, and
4. attract immune effecter cell infiltration near a tumor mass.

Figure 4: Proposed mechanisms of antibody-based cancer therapies



Antibodies against cell surface receptors can elicit cell killing effects by one or more the following proposed mechanisms: A. block receptor/ligand interactions, B. block receptor interactions with other partner receptors, C. activate receptor activity leading to "signal overload" and D. recruit immune complexes to tumor sites.

Source: Canaccord Adams

Less severe adverse events typical of the older class of cytotoxic agents account for the appeal of targeted agents, the oldest examples of which include the monoclonal antibodies Herceptin and Rituxan, approved in the 1990s. More recently, small molecules designed to target kinase activities driving cancer cell growth—such as Gleevec, Tarceva, Sutent, and Nexavar—have also been met with great success, as evident from sales data shown in Figure 5.

Figure 5: Selected FDA-approved targeted therapies

Monoclonal Antibodies

Drug	Target	Indication	FDA Approval	WW Revenue 2009A ($M)
Avastin	VEGF	CRC, NSCLC, MBC, Glioblastoma	2004	$5,900
Rituxan	CD20	NHL	1997	$5,800
Herceptin	HER2	MBC	1998	$5,000
Erbitux	EGFR	CRC, H&N	2004	$683
Vectibix	EGFR	CRC	2006	$233

Small Molecule Kinase Inhibitors

Drug	Target	Indication	FDA Approval	WW Revenue 2009A ($M)
Gleevec	BCR-ABL, PDGFR, Kit	CML, GIST	2001	$3,900
Tarceva	EGFR	NSCLC, Pancreatic	2004	$1,200
Nexavar	VEGFR1,2,3, RAF	RCC, HCC	2005	$850
Sutent	VEGFR1,2,3	RCC, GIST	2006	$1,000
Sprycel	BCR-ABL, SRC, KIT, PDGFR	Gleevec-Resistant CML, ALL	2006	$421
Tykerb	EGFR/HER2	MBC	2007	$258
Tasigna	BCR-ABL, KIT, PDGFR	Gleevec-Resistant CML	2007	$212
Afinitor	mTOR	RCC	2009	$70

MBC: metastatic breast cancer, NHL: non-Hodgkin's lymphoma, CRC: colorectal cancer, NSCLC: non-small cell lung cancer, H&N: head and neck cancer, GIST: gastrointestinal stromal tumor, CML: chronic myelogenous leukemia, RCC: renal cell carcinoma, HCC: hepatocellular cancer
*Includes sales for rheumatoid arthritis as well

Source: http://www.fda.gov/Drugs, Company Reports

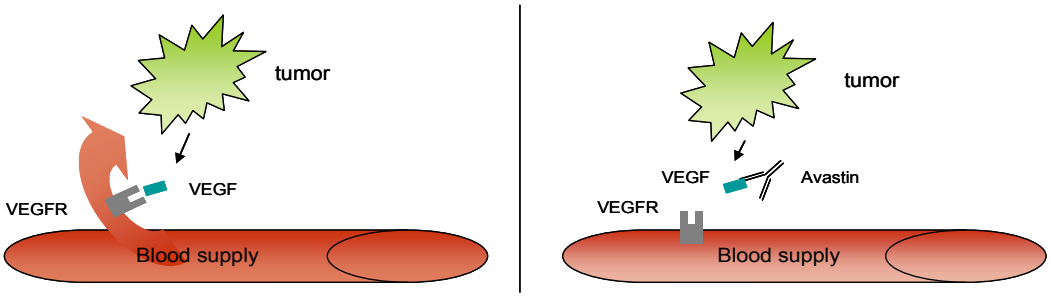
We see the landscape of new targeted agents as falling into three sub-classes:

1. anti-angiogenic agents designed to target VEGF signaling pathways,
2. EGFR kinase antagonists affecting the signaling of a family of cell surface growth factor receptors,
3. inhibitors of mTOR proteins involved in downstream cell signaling, and
4. everything else.

Inhibition of angiogenesis

First made evident by the clinical success of the antibody Avastin, inhibition of angiogenesis—the formation of new blood vessels—has proven to be an attractive pathway for both biologic anc small molecule inhibition. Designed to block the activity of vascular endothelial growth factor (VEGF), a critical mediator of blood vessel development, Avastin has been shown to extend both progression free and overall survival when included in cytotoxic chemotherapy regimens for treatment of colorectal, non-small cell lung, and breast cancer indications.

Figure 6: Theoretical mechanism of Avastin-mediated and VEGFR inhibition



Tumors and surrounding tissues secrete VEGF whose interaction with cognate receptor stimulates growth of cells destined to constitute new blood vessels. Avastin sequesters VEGF and blocks its activity theoretically leading to the ultimate starvation of the growing tumor.

Source: Canaccord Adams

Other means of blocking VEGF activity go right to the heart of the VEGF family of receptors whose inhibition by a variety of small molecules appears to provide meaningful clinical activity in a handful of treatment settings. Either way, targeting of the VEGF pathway may not be as mechanistically simple as illustrated in Figure 6. Rather, the therapeutic effects observed so far with anti-angiogenic agents when used in combination with chemotherapy (points #1-3) or as single agent (#4) may involve a combination of several proposed mechanisms:

1. "Normalization" of tumor vasculature: Many studies have confirmed vasculature in and around tumors as being disorganized, leaky, and abnormal. Inhibition of VEGF activity has been thought to "normalize" the vascular conditions around tumors effectively enhancing tumor exposure to chemotherapeutic agents and ensuring a more effective kill.

2. Prevent tumor "re-population" following cytotoxic treatment: Tumor shrinkage by chemotherapy is almost always followed by repopulation of tumor cells during the intervals where chemotherapy is not administered (e.g., carboplatin/paclitaxel and FOLFOX regimens are typically administered every three weeks in order to allow for bone marrow recovery, among other things). Due to its two- to three-week half-life, Avastin has been speculated to prevent tumor cell growth during cytotoxic treatment intervals, thereby delaying tumor progression.

3. Augmenting anti-angiogenic effects of chemotherapy: By virtue of their indiscriminate cell killing effects, chemotherapeutic agents possess anti-angiogenic properties themselves which could be augmented by simultaneous VEGF blockade.

4. VEGFR role in tumor growth: Independent of angiogenesis, tumor growth may in some instances depend on direct VEGFR signaling that may be perturbed by small molecule VEGFR antagonists. Alternatively, other kinase targets either unassociated with tumor growth or not yet identified could be targets for the small molecule VEGFR inhibitors as well.

Small molecule VEGFR inhibitors

Small molecules designed to target the various VEGF receptors (VEGFRs) rather than the circulating VEGF ligand, per se, should theoretically have the same effect as Avastin on developing tumor vasculature. With the exception of RCC treatment settings, however, no VEGFR inhibitor has had clinical success in treatment settings where Avastin is approved. For example, the VEGFR inhibitors Sutent, PTK787, and cediranib failed to demonstrate benefit in Phase III randomized colorectal cancer trials when combined with 5-FU/leucovorin regimens, while Nexavar failed to demonstrate benefit in a randomized front-line NSCLC trial. In our view, these results demonstrate that the means of VEGFR inhibition and VEGF ligand neutralization do not necessarily translate into the same end results.

Use of small molecule VEGFR inhibitors is largely confined to single-agent use for treatment of more niche oncology treatment settings, including renal cell carcinoma (RCC), hepatocellular carcinoma (HCC), and GIST where activity has been proven in randomized treatment settings. In RCC these agents may exert their activity via anti-angiogenic means, but direct anti-tumor effects by these agents cannot be ruled out.

Figure 7: Selected VEGFR inhibitors in late-stage clinical development

Drug	Company	Stage	Indication
Sutent (sunitib)	Pfizer	Marketed	RCC, GIST
Nexavar (sorafenib)	Onyx and Bayer Pharma	Marketed	RCC, HCC
Votrient (pazopanib)	GSK	Marketed	RCC
Axitinib	Pfizer	Phase III	RCC
BIBF 1120	BI	Phase III	NSCLC
Motesanib	Amgen and Takeda	Phase III	NSCLC
Tivozanib	AVEO	Phase III	RCC
Cediranib	AstraZeneca	Phase III	NSCLC, ovarian
brivanib	BMS	Phase III	HCC, CRC
Regorafenib	Bayer	Phase II	RCC, HCC, GIST
Vandetinib	AstraZeneca	Phase II	multiple

Source: Company reports and clinicaltrials.gov

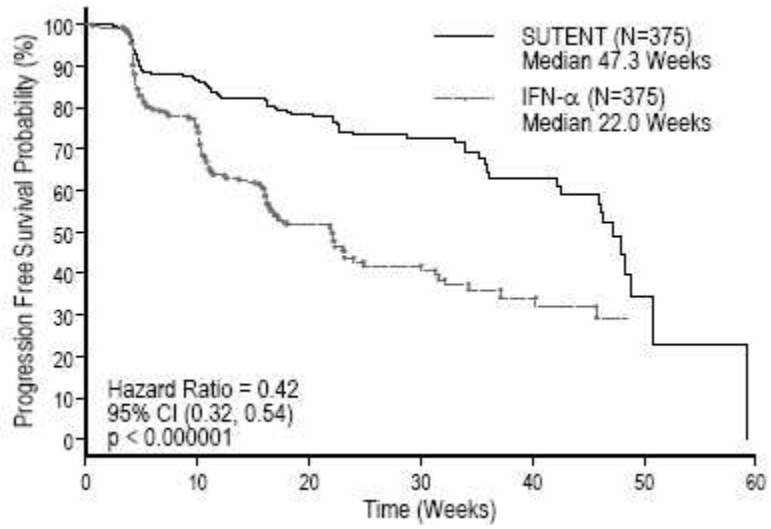
The first solid evidence that VEGFR antagonism with small molecules could play a role in the treatment of solid tumors came from results of a Phase II randomized discontinuation trial evaluating sorafenib (Nexavar) in multiple tumor types. The most responsive patients were those with RCC, whereby Nexavar demonstrated an 18-week (4x) improvement in median PFS over placebo in the randomized patient group. Not only was this benefit highly clinically meaningful, but it was also significantly greater than anything that had been achieved with cytokine therapy, the standard of care at the time.

RCC standards of care

For many years, standard of care for RCC—a tumor type that is notoriously resistant to chemotherapy—had been treated with two immunomodulating cytokines, interleukin-2 and interferon alpha. These drugs were approved for RCC treatment in the 1990s based on modest response rates and have never been shown to provide progression-free or overall survival benefits in randomized clinical trials. Furthermore, administration of both drugs is associated with debilitating side effects including fever-like symptoms, CNS disorders, and other adverse events that greatly impair quality of life.

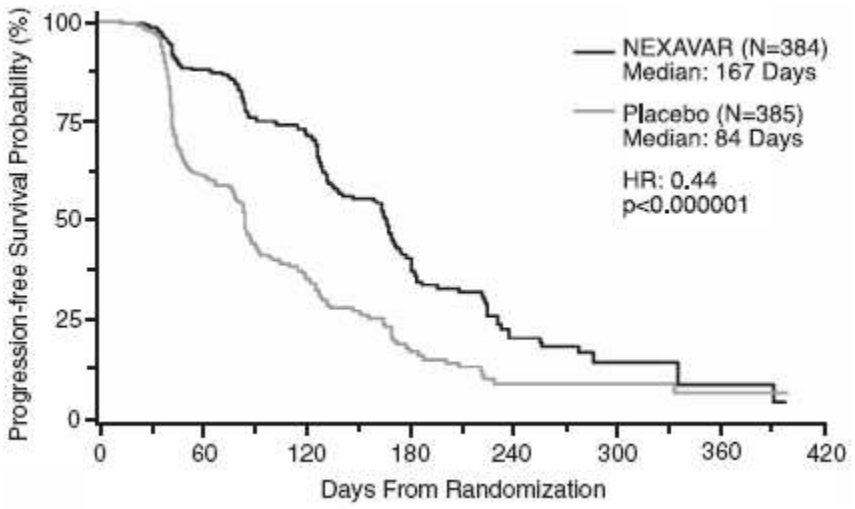
Phase III trials evaluating Nexavar and Sutent (Pfizer), another VEGFR inhibitor that had demonstrated activity in a single arm Phase II RCC trial, confirmed the validity of such an approach (Figures 8 and 9). Based on this data FDA granted marketing approval for single-agent Nexavar and Sutent as any line of treatment of Stage IV RCC. Last October FDA approved Votrient (pazopanib; GSK) with a similar label based on a Phase III study of similar design (Figure 10).

Figure 8: Results from the pivotal Phase III trial supporting Sutent approval in RCC



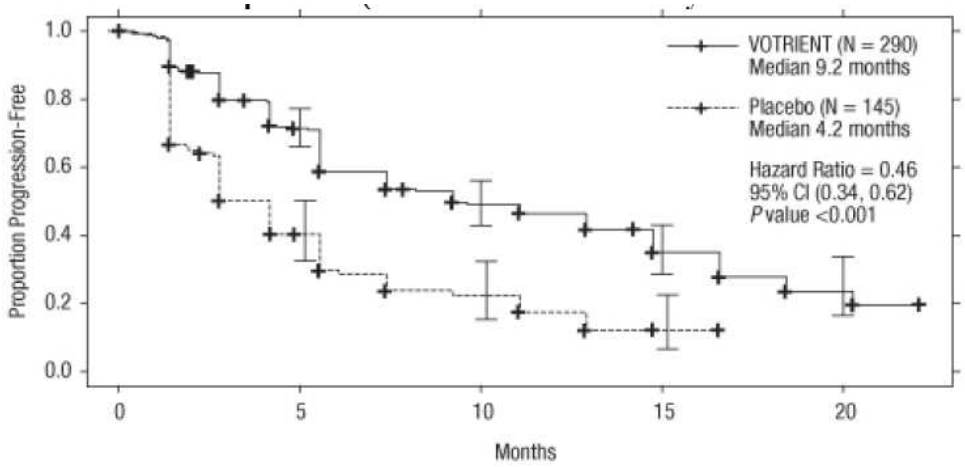
Source: Sutent package insert

Figure 9: Results from the pivotal Phase III trial supporting Nexavar approval in RCC



Source: Nexavar package insert

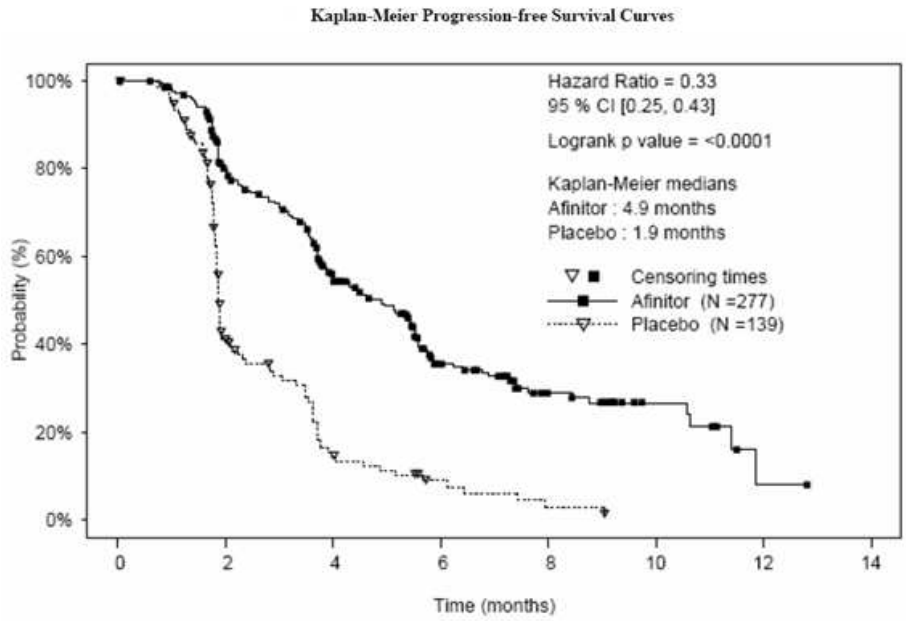
Figure 10: Results from the pivotal Phase III trial supporting Votrient approval in RCC



Source: Votrient package insert

From our conversations with treating physicians, practically all Stage IV RCC patients receive a small molecule VEGFR inhibitor (most often Sutent) as front-line treatment. If tolerability issues preclude continued administration, a second drug of this class (most often Nexavar) is tried. Oftentimes patients cycle between Sutent and Nexavar until lack of benefit is clearly established, whereupon patients are transitioned to treatment with an mTOR inhibitor. Drug of choice is most often Afinitor (everolimus) based on the extremely compelling Phase III data reported at ASCO '08 demonstrating a very meaningful 66% reduction in risk of disease progression over placebo in patients who had failed VEGFR inhibitor therapy (Figure 11).

Figure 11: Results from the pivotal Phase III trial supporting Afinitor approval in RCC



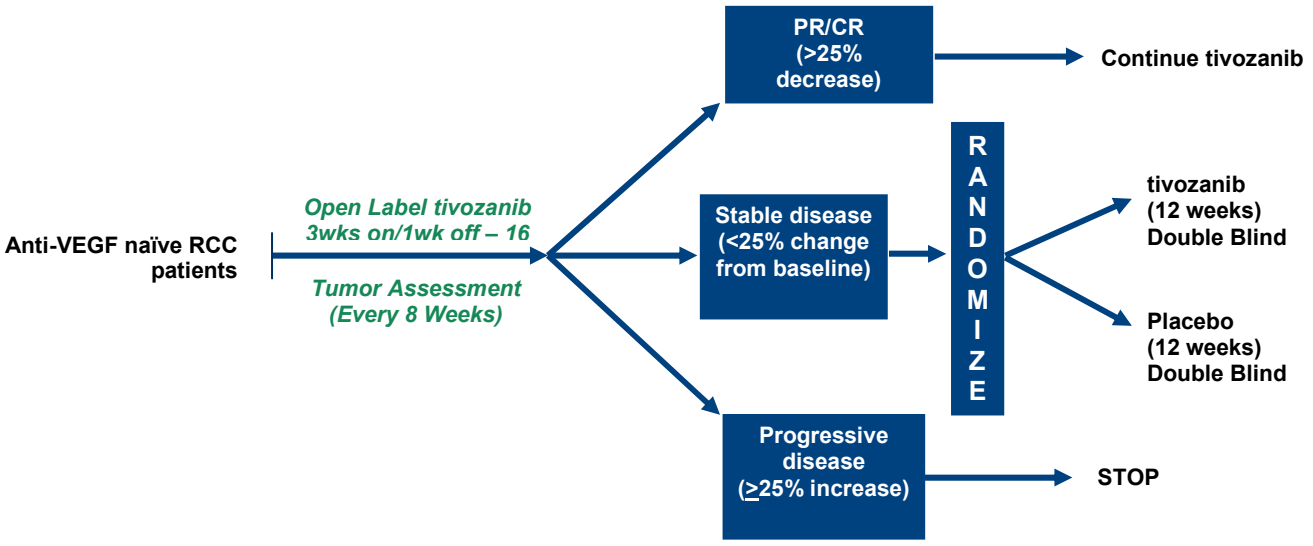
Source: Afinitor package insert

While Sutent, Nexavar, and Votrient have been shown to have high affinities for a variety of different kinase targets, their ability to inhibit the activity of VEGF receptors (VEGFRs) in particular has been attributed to their activity in RCC. Whether their action is mediated by directly targeting cancer cells or by targeting growing vasculature that feeds tumor growth remains controversial. Nevertheless, inhibition of VEGFRs appears to be a viable means of treating RCC in front-line treatment settings as evidenced by the clinical data so far.

TIVOZANIB

We believe that tivozanib could be the next newcomer to the class of VEGFR inhibitors approved for treatment of RCC. Latest results from a Phase II trial evaluating tivozanib in a randomized discontinuation design practically identical to the one providing the first evidence of Nexavar activity indicate, in our view, that tivozanib can meaningfully extend progression-free survival in patients with Stage IV RCC. This randomized discontinuation design specifically involved open-label treatment of newly diagnosed Stage IV RCC patients with tivozanib for 16 weeks (three weeks on, one week off) and tumor scans every eight weeks. Patients experiencing a >25% reduction in tumor volume remained on tivozanib therapy until disease progression. Patients experiencing a >=25% increase in tumor volume were effectively deemed treatment failures and ceased further tivozanib therapy. Patients falling in between these boundaries (effectively with stable disease at the end of 16 weeks of treatment) were randomized in a double-blinded fashion to remain on tivozanib therapy or were transitioned immediately to placebo for an additional 12 weeks (Figure 12).

Figure 12: Tivozanib Phase II randomized discontinuation trial (RDT)



Source: Company reports

Ethical considerations aside, a randomized discontinuation trial (RDT) can be an effective tool to ascertain drug candidate treatment effect since drug vs. placebo are compared in a background of patients preselected to respond to therapy. However, as in the case of AVEO's RDT trial, because most of the patients were excluded from the final analysis (either because of robust or insufficient tumor response prior to randomization), a faithful comparison between drug and placebo cannot be made. Nevertheless, notable efficacy results obtained in this study support tivozanib activity in RCC, including those listed in Figure 13.

Figure 13: Salient results from the Phase II RDT tivozanib trial in RCC

	tivozanib	placebo
Median PFS (intent-to-treat)*	11.8 months	NA
Median OS	Not reached after 500 days	175 days
Response rate (ITT)	25%	NA
SD or better (ITT)	84%	NA

* includes all patients enrolled whether randomized to tivozanib or placebo or excluded after 16 weeks of initial therapy
Source: Company reports

Drug-related adverse events were consistent with those observed in other trials evaluating small molecule VEGFR antagonists, including hypertension and fatigue (Figure 14). As discussed below, other side effects associated with competitors do not seem to be nearly as profound with tivozanib, which we believe will significantly influence physician choice with regard to best RCC treatment should tivozanib win marketing approval.

Figure 14: Grade 3/4 adverse events observed in the Phase II tivozanib RDT

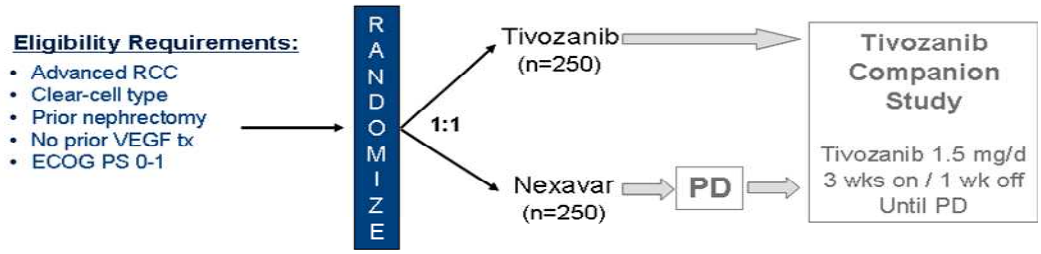
SAE	frequency
Hypertension	9%
Asthenia	2%
Diarrhea	2%
Fatigue	2%
Dyspnea	1%
Rash	1%
Cough	1%

Source: Company reports

Tivozanib registration strategy: TIVO-1

As per AVEO management, FDA will no longer accept pivotal trials for approval of new front-line RCC treatment regimens that rely on comparison to either placebo or cytokine therapy (such as those that facilitated approval of Sutent, Nexavar, and Votrient). Rather, evaluation against an active targeted therapeutic comparator is required to support an NDA that FDA will accept. To this end, AVEO has embarked on a Phase III registration trial comparing the activity of tivozanib against Nexavar, the small molecule VEGFR inhibitor with the least potency perceived of all approved molecules in this class. This Phase III trial, TIVO-1 (illustrated in Figure 15), is designed to demonstrate a three-month or greater improvement in PFS with tivozanib, which we believe should be sufficient to win FDA marketing approval.

Figure 15: Phase III TIVO-1 trial design



Source: Company reports

In our view, TIVO-1 will need to hit the following goals in order for tivozanib to ultimately achieve penetration in the RCC treatment market:

1. The trial will have to demonstrate a PFS superiority with tivozanib over Nexavar in the RCC treatment setting. We do not believe FDA will approve tivozanib if this hurdle is not met.
2. The PFS attained with tivozanib will have to be at least comparable to what was observed with Sutent (11 months) in the Phase III registration for that drug. This represents a significant portion of the tivozanib value proposition in RCC, since Sutent is the market leader in this space.
3. The serious adverse event profile needs to be consistent with what was observed in the Phase II RDT trial described above. Although tivozanib is not being compared head-to-head with Sutent, pointing to a tighter SAE profile confined to hypertension,

fatigue, and asthenia should offer a critical marketing angle when trying to win physician acceptance in the front-line RCC setting.

Giving us confidence that tivozanib can meet at least the first three of these hurdles, we put available data for tivozanib within the context of that published on other VEGFR inhibitors in development and on the market.

RCC cross-trial comparison of tivozanib efficacy results indicates that tivozanib is probably one of the most potent small molecules in late-stage development. As shown in Figure 16, comparing VEGFR affinity and PFS data from various trials evaluating VEGFR antagonists in RCC treatment settings where patients were naïve to prior TKI therapy indicates that the tivozanib efficacy benefit is at least equivalent to what has been seen with Sutent, the market leader in RCC.

Figure 16: VEGFR inhibitor activity in RCC clinical studies

	IC50 (nM)				
Compound	**R1**	**R2**	**R3**	**ORR***	**PFS**
Nexavar	N/A	90	100	2-10%	5.5 mo. (1)
Sutent	10	10	10	30-45%	10.9 mo. (1)
Votrient	10	30	47	30%	9.2 mo. (1)
Tivozanib	0.2	0.2	0.2	24%	11.8 mo. (2)
Axitinib	1.2	0.2	0.3	47%	15.7 mo. (2)
Regorafenib	16	5	46	27%	8.3 mo. (2)

* in VEGFR inhibitor naïve patients
(1) Phase 3 result
(2) Phase 2 result
Source: Rini et al., ASCO '09, Mendel et al., Clin. Cancer Res (2003), Eisen et al., ESMO '09, Nakamura et al., Cancer Res. (2006)

Despite comparable efficacy, the tolerability profile of tivozanib appears tighter than that of other members of the VEGFR inhibitor class. Besides the frequency of Grade 3/4 serious adverse events such as hypertension and fatigue that have been seen with all angiogenesis inhibitors so far, no other meaningful SAEs are apparent with tivozanib.

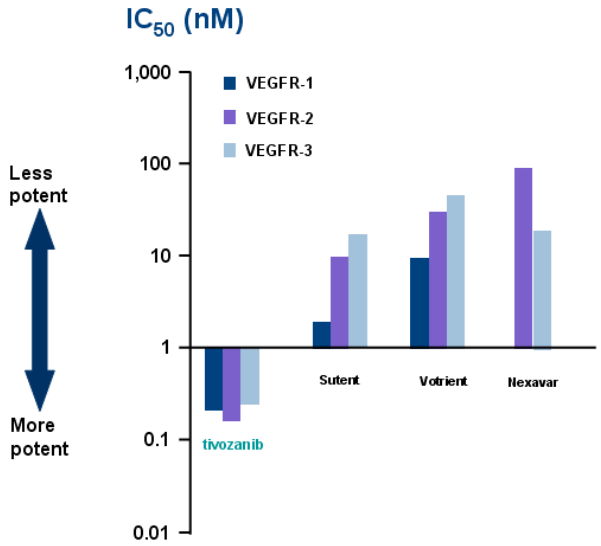
Figure 17: Selected Grade 3/4 adverse events with tivozanib vs. marketed VEGFR inhibitors

	tivozanib	Sutent	Nexavar	Votrient
Hypertension	9%	10%	3%	4%
Asthenia	2%	7%		3%
Diarrhea	2%	6%	2%	3%
Fatigue	2%	9%	5%	2%
Dyspnea	1%	4%	3%	
Rash	1%	1%		
Cough	1%	1%		
Nausea		4%		
Mucositis		3%		
Vomiting		4%		2%
Hand-foot syndrome		5%	6%	
Bleeding		3% *	2%	
lymphocytopenia				4%
Neutropenia				1%
ALT elevation				12%
AST elevation				7%
Bilirubin elevation				3%

* one episode of Grade 5 gastrointestinal hemorrhage reported
Source: SEC documents and package inserts

Affinity for the relevant targets appears most pronounced with tivozanib, which would correlate with the high potency of the drug relative to other members of the class. As shown in Figure 18, tivozanib has more or less equivalent affinity for the three relevant VEGF receptors and binds the relevant targets almost 50x tighter than Sutent, the market leader of the class.

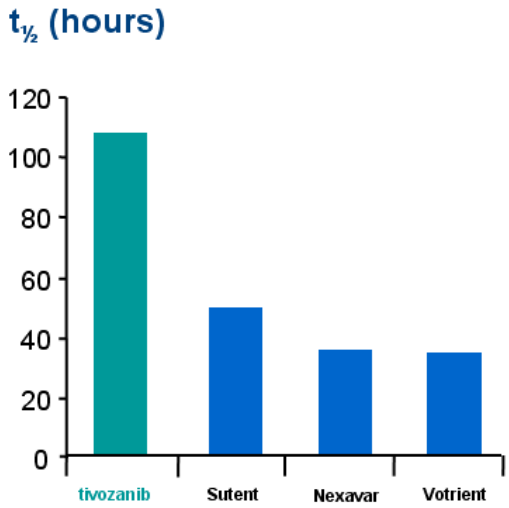
Figure 18: Relative VEGFR inhibitor affinities for relevant targets



Note: Values are slightly different from those listed in Table 16
Source: Company presentation

Complete and consistent coverage of target receptors is considered most ideal when aiming for a sustained anti-tumor effect. A longer blood stream half-life usually translates into a prolonged interaction with intended targets. The substantially longer half-life observed with tivozanib (~ 4.5 days) compared to what has been published for other compounds (Figure 19) indicates that part of the perceived superior activity of tivozanib could be due to this property. This also bodes well for tivozanib demonstrating superiority over Nexavar in TIVO-1.

Figure 19: Selected VEGFR inhibitor clinical half-lives

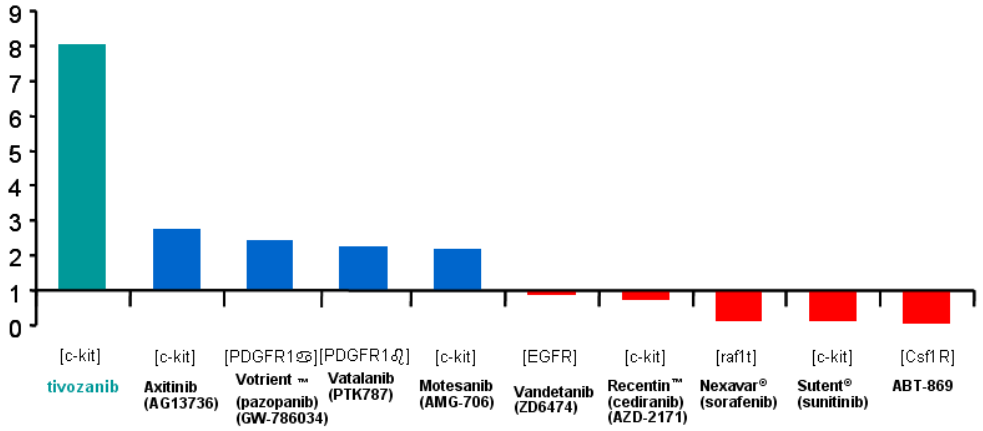


Source: Company presentation

Higher selectivity for relevant targets could also be behind the perceived superior selectivity observed with tivozanib compared to other members of the class. The promiscuous activity that appears to be consistently seen with all small molecule kinase inhibitors is thought to be behind their broad adverse event profiles. In theory, employing drugs with more selective drug-target interaction profiles should translate into lower frequencies of adverse events. Moreover, drugs with more selective profiles should theoretically have better chances of being combined with therapeutics of different classes that have distinct toxicity profiles.

As shown in Figure 20, tivozanib appears to have a much more target selective profile than its potential competitors. In our view, this selectivity is consistent with its relatively more restricted side-effect profile and the notion that higher selectivity translates into the lower frequency of adverse events. This property could be particularly relevant toward potential application in other disease indications.

Figure 20: Tivozanib relative selectivity: VEGR vs. off-target kinase activity



Calculated selectivity: mean potency of VEGFR-1, -2, -3 versus next most potent kinase [brackets]; Selectivity <1 indicates a higher potency for the off-target kinase.
Source: Company presentations

Retrospective subgroup analysis of the Phase II RDT trial evaluating tivozanib indicates that RCC patients with clear-cell histology (the most predominant class) and who had undergone prior nephrectomy had a 14.8 month median PFS, which is meaningfully superior, in our view, to the 11.8 month PFS benefit seen in the intent-to-treat. Data from the Phase III registration trial supporting Nexavar approval in RCC showed a 5.5-month median PFS in patients, all of whom had clear cell histology and 94% of whom had prior nephrectomy. The Phase III TIVO-1 trial will exclusively enroll patients with clear-cell histology and with prior nephrectomy, thus potentially optimizing the success of this trial.

In our view, the facts that 1) only 10% of patients in the Phase II RDT required tivozanib dose reduction and 2) only 4% required dose interruption speak well to the exceptional tolerability of this drug relative to the potential competition. In the Phase III RCC registration trial for Sutent, for example, 32% of patients dose reduced while 38% experienced dose interruptions.

Risks to TIVO-1 success

While we believe the prevailing evidence supports a TIVO-1 positive outcome, we call attention to several risks that could potentially complicate trial success:

1. TIVO-1 is the first randomized head-to-head Phase III trial ever to compare two different small molecule VEGFR inhibitors in the RCC treatment setting. Approvals of Sutent, Nexavar, and Votrient were all based upon the outcome of trials comparing drug to placebo or cytokine therapy. Although the prevailing evidence indicates that Nexavar is less potent than tivozanib, the former still has activity in RCC. Hence, by virtue of being compared directly to another active drug, the hurdle for tivozanib success in TIVO-1 is higher than that for the other VEGFR inhibitors approved for treatment of RCC

2. Median overall survival has yet to be reported from the tivozanib Phase II RDT, but in our view, needs to be superior to the median OS of 17.8 months reported with Nexavar in the Phase III TARGET trial in order to maintain confidence in TIVO-1. Moreover, practically all patients enrolled in TARGET had had prior cytokine therapy indicating that the 17.8 OS reported is probably not representative of the treatment-naïve patient population enrolling in TIVO-1.

   As a caveat, results from a randomized Phase II trial evaluating Nexavar vs. Nexavar/interferon in a treatment-naïve patient population showed a median PFS of 5.7 months with Nexavar. This outcome is consistent with what was observed in the ITT from the TARGET trial (5.5 months) indicating that the line between front- and second-line treatment may be blurry with respect to predicting outcomes. To our knowledge, overall survival results have not been reported from this trial.

3. While the outcome of TIVO-1 should favor tivozanib, in our view, the tolerability profile of the drug will need to remain consistent with what was reported in the Phase II RDT in order to maintain the tivozanib value proposition in RCC. Recall, the only major adverse events reported with tivozanib were hypertension, asthenia and fatigue consistent with what has been observed with other members of its class. In our view, the cleanliness of this safety profile will have to be recapitulated in TIVO-1 in order for physicians to consider making the switch from Sutent, all else being equal. Exposing a new patient pool of 250 subjects to tivozanib could reveal some unforeseen side effects.

Tivozanib potential in expanded indications

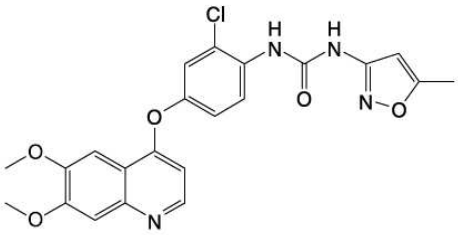
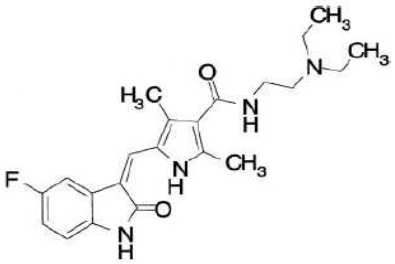
AVEO moves tivozanib through early-stage clinical trials evaluating its safety and potential efficacy when used in combination with other oncologic therapeutics, including Torisel, 5FU/oxaliplatin, and paclitaxel. If AVEO hopes to successfully partner tivozanib, demonstrating that the drug can be combined with other therapeutic agents could be critical, especially since its most likely competitor, Sutent, has failed to show applicability when used in combination with other agents after evaluation in a number of completed studies.

While the immediate value proposition for tivozanib lies in its potential to treat RCC, the longer-term value of the drug relies on its potential to treat other larger market disease indications, in our view. Other small molecule VEGFR inhibitors have had success demonstrating meaningful activity in other indications, including Nexavar in hepatocellular carcinoma (HCC) and metastatic breast cancer (MBC) as well as Sutent in

GIST. However, results from other randomized trials designed to evaluate these and other agents of the class indicate that their potential might be limited. For example, Nexavar failed to demonstrate activity in melanoma and NSCLC indications, cederanib and PTK787 failed to show benefit in a front-line colorectal cancer treatment settings, motesanib failed to show benefit in NSCLC, and Sutent failed to demonstrate benefit in three different MBC treatment settings.

Such treatment failures make it problematic in predicting the potential of tivozanib as treatment for other disease indications beyond RCC. Comparing and contrasting its efficacy and safety to Sutent may be convenient when speculating whether tivozanib will ultimately be the VEGFR inhibitor of choice for treating RCC. However, as shown in Figure 21, while the structures of tivozanib and Sutent are quite distinct, both drugs share the same high affinity targets. This indicates to us that the panorama of clinical potential may be the same for both drugs. Hence, with regards to the potential of tivozanib in MBC, which we believe could be the next major indication in which to move the drug forward based on the clinical trial success of Nexavar, the failure of Sutent in this indication creates uncertainty as to whether tivozanib will perform in this setting, in our view.

Figure 21: Tivozanib and sunitinib structure comparison

tivozanib	sunitinib (Sutent)
	

Source: Nakamura et al., Cancer Res. (2006) and Mendel et al., Clin. Cancer Res. (2003)

Figure 22: IC50s of tivozanib vs. sunitinib for on-ligand dependent phosphorylation of selected targets

	tivozanib	sunitinib
VEGFR-1 (FLT-1)	0.2	10
VEGFR-2 (KDR)	0.2	10
VEGFR-3	0.2	10
C-KIT	1.6	~ 50
FLT-3	422	250
FLT-3 (ITD)	N/A	50
PDGFR-beta	1.7	10

Source: Nakamura et al. Cancer Res (2006), Mendel et al., Clin Cancer Res. (2003), Abrams et al. Mol. Cancer Ther. (2003)

Market analysis

Given the available treatments and past sales histories for Sutent and Nexavar in particular, we estimate that the worldwide RCC market is about $1.0B. We estimate 80%, 18%, and 2% market shares for Sutent, Nexavar and Votrient (most recently approved), respectively, in 2010. We estimate this market is largely saturated and should continue growing at about 7% per year based on increased RCC incidence and price increases.

If tivozanib wins approval in both territories and AVEO's development and regulatory strategy goes as planned, we model for a slow ramp of tivozanib sales as the drug begins to take market share from Sutent. All drugs are expected to continue to be cycled in and out until bona fide VEGFR inhibitor failure is ascertained.

For now we do not consider the potential of Pfizer's axitinib as potential competition based on the little insight we have on its Phase III program. Phase II data indicates that that hypertension could be a significant tolerability issue with the drug and its half-life appears to be the shortest of the class. Comparable affinity for relevant VEGFR targets and maybe others could be behind the impressive time to progression of 15.7 months in 52 patients. Other side effects such as stomatitis and liver enzyme elevations could also be of issue with axitinib.

Figure 23: Tivozanib revenue assumptions

	2011E	2012E	2013E	2014E	2015E	2016E	2017E	2018E	2019E	2020E
# eligible Stage IV patients	12,853	12,982	13,112	13,243	13,375	13,509	13,644	13,780	13,918	14,057
% treated	80%	80%	80%	80%	80%	80%	80%	80%	80%	80%
% treated with TKI's	95%	95%	95%	95%	95%	95%	95%	95%	95%	95%
share tivozanib	0%	0%	5%	20%	45%	65%	70%	70%	70%	70%
share Nexavar	18%	18%	18%	18%	18%	18%	18%	18%	18%	18%
share Sutent	80%	80%	75%	60%	35%	15%	10%	10%	10%	10%
share Votrient	2%	2%	2%	2%	2%	2%	2%	2%	2%	2%
# patients treated with tivozanib	0	0	498	2,013	4,574	6,673	7,259	7,331	7,405	7,479
average duration of therapy (months)	11	11	11	11	11	11	11	11	11	11
patient months per year	0	0	5,481	22,142	50,317	73,407	79,845	80,643	81,450	82,264
cost per month	0	0	$5,000	$5,000	$5,250	$5,250	$5,513	$5,513	$5,788	$5,788
sales (U.S.) ($M)	**0**	**0**	**27.4**	**110.7**	**264.2**	**385.4**	**440.1**	**444.5**	**471.4**	**476.2**
ex U.S. sales as % U.S.	0%	0%	0%	10%	20%	30%	40%	50%	50%	50%
ex U.S. sales ($M)	**0**	**0**	**0**	**11.1**	**52.8**	**115.6**	**176.1**	**222.3**	**235.7**	**238.1**
TOTAL US and ex-US sales (ex-Japan) ($M)	**0.0**	**0.0**	**27.4**	**121.8**	**317.0**	**501.0**	**616.2**	**666.8**	**707.2**	**714.2**

Source: Canaccord Adams estimates

OTHER PIPELINE THERAPEUTIC CANDIDATES

AV-299

AVEO is developing AV-299, a monoclonal antibody designed to target the soluble HGF-1 growth factor. Targeting HGF-1, the ligand of the C-MET receptor, is thought to be one of many different means of perturbing a particular cell signaling pathway implicated in tumor growth. This approach is supported by compelling preclinical data involving mouse models for cancer, but remains largely theoretical with respect to application in the clinic.

Phase I clinical data has demonstrated that AV-299 is well tolerated. With clinical development partner Merck, AVEO has moved this antibody into a randomized Phase II clinical trial in combination with the EGFR inhibitor, gefitinib, for treatment of Asian patients with non-small cell lung cancer (NSCLC) in the front-line treatment setting.

Recently, Arqule announced that a randomized Phase II trial comparing erlotinib (a drug similar to gefitinib) in combination with its C-MET inhibitor hit its primary endpoint of improvement in progression-free survival. This result, in our view, gives validation toward C-MET pathway targeting approaches for treating NSCLC and suggests that AV-299 inhibition of HGF-1 may result in the same additive effect when used in combination with another EGFR inhibitor.

AVEO is responsible for all clinical development of AV-299 through Phase II at Merck's expense. We believe AVEO will receive a milestone payment upon potential Phase III commencement. AVEO is entitled to co-promote AV-299 in certain oncology indications if marketed.

Due to the relatively early stage of this program, future sales estimates for AV-299 are not reflected in our model.

AV-203

AV-203 is a monoclonal antibody designed to target the HER-3 protein involved in cell signaling. HER3 is a member of the EGFR family of receptors, which includes validated targets such as EGFR1 and HER2. Phase I results indicated that AV-203 is well-tolerated.

Biogen IDEC has option rights to develop AV-203 outside of North America after a Phase II proof-of-concept is completed.

Due to the early stage of this program, future sales estimates for AV-203 are not reflected in our model at this time.

DRUG DISCOVERY PLATFORM

AVEO owns exclusive rights to a unique mouse tumor model that enables validation of oncology drug candidates in a setting that resembles the human condition much more accurately than do other mouse tumor models. Typical mouse cancer models are severely limited since most involve exogenous implantation of cancer cell lines (often under the skin) of immunocompromised animals. Preclinical validation of compounds relying on such models often depends largely on their ability to reduce tumor volume in this highly artificial system.

AVEO's model goes beyond the limitations of these conventional preclinical mouse cancer models. Through use of conventional mouse genetic techniques, specific genes known to play important growth regulatory roles in specific organ tumor types can be selectively activated in otherwise normal mice that slowly results in the development of the desired malignancy. For example, overexpression of the HER2 gene product can be induced specifically in the mammary glands of mice, resulting in a truer recapitulation of human breast cancer than what has been typically derived before. AVEO mice develop tumors specifically within organ systems that the mice are transgenically designed to mimic.

ALLIANCES

AVEO has a number of corporate alliances that we model providing non-dilutive sources of cash going forward. Selected relationships include the following.

Merck

In addition to Merck option on AV-299 development, Merck is supporting R&D efforts in connection with the discovery and validation of cancer targets utilizing AVEO's target identification platform in mice. In connection with this aspect of the collaboration AVEO has received $15M in cash payments and $5M from an equity investment since 2003.

OSI Pharmaceuticals

In return for rights to AVEO's discovery platform for purpose of target validation and small molecule discovery, OSI has made cash payments and equity investments in AVEO. OSI also continues to provide research support to AVEO in connection with R&D efforts to which OSI has rights.

FINANCIALS

Revenues

We estimate AVEO will continue booking revenues related to its corporate collaborations for at least the next 10 years and begin modeling tivozanib sales in 2013. We estimate $27M, $122M, and $317M in WW (ex-Japan) tivozanib product sales in 2013, 2014 and 2015, respectively. Pending additional data, future tivozanib application in indications beyond RCC or potential AV-299 and AV-203 sales are not factored in our model at this time.

Op-ex

Excluding the impact of a $10M milestone payment to Kirin this year, we model for 8-10% R&D growth during 2011-2014, which could increase depending on progress with AV-299 and AV-203.

Earnings

We model for full-year profitability in 2014 and $0.75, $2.34, and $3.67 in EPS in 2014, 2015, and 2016, respectively.

Balance sheet

AVEO reported $51M in cash, cash equivalents and liquid securities as of December 2009. After raising $81M in its IPO, we estimate the company finished Q1/10 with $108M. Based on our estimated cash spend of $56M in 2010, we believe cash should be sufficient into 2012, excluding potential upfront payments from unannounced business development activities. AVEO has outstanding long-term debt of $20M, which we model will be paid down by $7.5M in 2010, $7.5M in 2011 and $4.8M in 2012.

VALUATION

Consistent with how we view most small-cap biotech companies as potential take-out candidates, we value AVEO based on a modified DCF model assuming tivozanib product launch in 2013 for treatment of RCC. Other potential indications for tivozanib, sales estimates for earlier-stage therapeutic candidates AV-299 and AV-203, and value of net operating losses carry forwards are not considered in our valuation at this time. AVEO's estimated cash balance of $108M after completion of the IPO is not considered, but the company's debt balance is. A summary of model assumptions follows:

- Tivozanib launch in 2013 for treatment of RCC;
- A potential acquirer would dissolve all company R&D efforts after integration;
- Out-year operating margins (including COGs and SG&A) of 46% in 2013 and eroding to 35% on tiered royalty payments to Kirin on tivozanib sales;
- Tax rate of 35%;
- 70% chance of TIVO-1 success; and
- A platform technology value of $100M.

Figure 23: AVEO DCF valuation

	2010E	2011E	2012E	2013E	2014E	2015E	2016E	2017E	2018E	2019E	2020E	2021E	2022E
Tivozanib U.S.	0.0	0.0	0.0	27.4	110.7	264.2	385.4	440.1	444.5	471.4	476.2	480.9	485.7
Tivozanib ex-U.S. (ex-Japan)	0.0	0.0	0.0	0.0	11.1	52.8	115.6	176.1	222.3	235.7	238.1	240.5	242.9
Total tivozanib sales	**0.0**	**0.0**	**0.0**	**27.4**	**121.8**	**317.0**	**501.0**	**616.2**	**666.8**	**707.2**	**714.2**	**721.4**	**728.6**
gross margin				80%	80%	80%	77%	77%	77%	77%	77%	77%	77%
SG&A				43%	38%	35%	33%	32%	32%	31%	31%	31%	31%
U.S. operating profits			**0.0**	**12.5**	**54.9**	**137.4**	**198.8**	**230.5**	**232.8**	**250.5**	**253.0**	**255.5**	**258.1**
operating margin				**45.6%**	**45.1%**	**43.3%**	**39.7%**	**37.4%**	**34.9%**	**35.4%**	**35.4%**	**35.4%**	**35.4%**
tax rate				35%	35%	35%	35%	35%	35%	35%	35%	35%	35%
post-tax net income				**8.1**	**35.7**	**89.3**	**129.2**	**149.8**	**151.3**	**162.8**	**164.4**	**166.1**	**167.7**
revenue weighted handicap factor				70%	70%	70%	70%	70%	70%	70%	70%	70%	70%
probability adjusted				5.7	25.0	62.5	90.5	104.9	105.9	114.0	115.1	116.3	117.4
NPV	**0.0**	**0.0**	**0.0**	**4.0**	**15.9**	**35.5**	**45.8**	**47.4**	**42.8**	**41.1**	**37.1**	**33.4**	**30.1**

discount rate	12%
sum NPVs	333.1
technology value	100.0
debt	(20.4)
shares outstanding	29.6
value per share	**$13.94**

Source: Canaccord Adams estimates

INVESTMENT RISKS

1. TIVO-1 could fail to hit its primary endpoint

2. Competitive products in development could appear similar or better than tivozanib

3. Deteriorating market conditions could complicate future financing needs

APPENDIX: SMALL MOLECULE VEGFR ANTAGONISTS

Sunitinib (Sutent) – Pfizer

Approved in 2006 for treatment of GIST and iStage IV RCC, Sutent is the best selling small molecule VEGFR inhibitor in a market where two other drugs are available and is the drug of choice for first-line treatment of RCC. With sales of $1.0B and $1.0B in 2008 and 2009 in all indications, respectively, we estimate that Sutent commands about 72% of the worldwide RCC treatment market.

While Sutent commands the lead in the RCC treatment setting, other major clinical setbacks, including Phase III failures in non-small cell lung and metastatic breast cancers, have hindered penetration of this drug into larger market indications. Most recently, Sutent was shown to have meaningful clinical activity in a neuroendocrine pancreatic cancer indication. Sutent continues through late-stage evaluation in HCC, NSCLC, and hormone refractory prostate cancer indications.

Sorafenib (Nexavar) – Onyx and Bayer Pharmaceuticals

Nexavar was the first VEGFR antagonist approved for treatment of RCC by virtue of the TARGET Phase III trial results. Nexavar has also been granted worldwide approval for treatment of HCC based on results of the SHARP and another Phase III trial which demonstrated meaningful benefits in overall survival vs. placebo.

With penetration in the RCC indication under continued pressure from Sutent and by the newly approved mTOR inhibitor, everolimus (Afinitor), for treatment of VEGFR resistant RCC, the growth potential for Nexavar lies in the HCC market where it remains the only approved treatment for advanced disease. Nexavar has also demonstrated meaningful clinical activity in metastatic breast cancer when combined with capecitabine (Xeloda) and as a single agent for treatment of medullary thyroid cancer.

Pazopanib (Votrient) – Glaxo Smith Kline

Newly approved in 2009, Votrient has yet to gain significant share of the RCC treatment segment. Adverse events reported in its Phase III registration trial, including liver enzyme elevations, myelosuppression, and perceived cardiotoxicity will likely stifle market penetration, in our view.

Axitinib – Pfizer

Perhaps a drug with equivalent potency as tivozanib, axitinib moves forward in a Phase III trial vs. placebo for treatment of RCC. Phase II data suggests that axitinib could be regarded as a more potent sunitinib, but its future is uncertain, in our view, given tolerability issues observed so far.

Vandetanib, cediranib, motesanib, vatalanib, BIBF 1120, regorafenib, ABT-869

All of these drugs have been or are undergoing Phase II and Phase III evaluation. Vandetanib, cediranib, and vatalanib, failed to demonstrate meaningful activity in the solid tumor indications where evaluated and probably will not move forward through the FDA regulatory process, in our view. Roche recently cancelled development of ABT-869 given the crowded field. Future Phase III development plans for regorafenib may include front-line colorectal cancer treatment where others have failed.

Figure 24: AVEO annual income statement ($M except EPS)

	2009A	2010E	2011E	2012E	2013E	2014E	2015E	2016E	2017E	2018E	2019E	2020E
Product revenues												
tivozanib (U.S.)	0.0	0.0	0.0	0.0	27.4	110.7	264.2	385.4	440.1	444.5	471.4	476.2
tivozanib (ex-U.S.)	0.0	0.0	0.0	0.0	0.0	11.1	52.8	115.6	176.1	222.3	235.7	238.1
other	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Total product revenues	**0.0**	**0.0**	**0.0**	**0.0**	**27.4**	**121.8**	**317.0**	**501.0**	**616.2**	**666.8**	**707.2**	**714.2**
Collaboration revenues	20.7	33.6	22.5	24.7	27.2	29.9	32.9	36.1	39.8	43.7	48.1	52.9
Total Revenues	**20.7**	**33.6**	**22.5**	**24.7**	**54.6**	**151.7**	**349.9**	**537.2**	**656.0**	**710.5**	**755.2**	**767.1**
COGs	0.0	0.0	0.0	0.0	(5.5)	(24.4)	(63.4)	(115.2)	(141.7)	(153.4)	(162.6)	(164.3)
as % product sales					20%	20%	20%	23%	23%	23%	23%	23%
Research & Development	(51.8)	(62.2)	(55.9)	(61.5)	(67.7)	(74.4)	(81.9)	(87.6)	(93.8)	(100.3)	(107.3)	(114.9)
General & Administrative	(10.1)	(11.1)	(12.2)	(13.5)	(14.8)	(16.3)	(17.9)	(19.7)	(21.7)	(23.9)	(26.2)	(28.9)
Total Operating Expenses	**(61.9)**	**(73.3)**	**(68.2)**	**(75.0)**	**(88.0)**	**(115.1)**	**(163.2)**	**(222.6)**	**(257.2)**	**(277.6)**	**(296.2)**	**(308.0)**
Operating Income	(41.2)	(39.7)	(45.7)	(50.3)	(33.4)	36.6	186.6	314.6	398.8	433.0	459.0	459.1
operating margin	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Interest income	0.1	0.3	0.3	0.1	0.0	0.0	0.0	0.4	1.2	2.2	3.4	4.6
Interest expense	(2.8)	(2.8)	(2.0)	(1.6)	(0.8)	(0.8)	(0.8)	(0.8)	(0.8)	(0.8)	(0.8)	(0.8)
Other, net	(0.3)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Pre-Tax Income	**(44.2)**	**(42.2)**	**(47.4)**	**(51.8)**	**(34.2)**	**35.8**	**185.8**	**314.2**	**399.2**	**434.4**	**461.6**	**462.9**
Taxes (benefit)	(0.1)	0.0	0.0	0.0	0.0	0.0	65.0	110.0	139.7	152.0	161.5	162.0
tax rate	0.0	0.0	0.0	0.0	0.0	0.0	0.4	0.4	0.4	0.4	0.4	0.4
Net Income	**(44.1)**	**(42.2)**	**(47.4)**	**(51.8)**	**(34.2)**	**35.8**	**120.8**	**204.2**	**259.5**	**282.4**	**300.0**	**300.9**
EPS (basic)	($27.43)	($1.44)	($1.42)	($1.39)	($0.83)	$0.79	$2.45	$3.83	$4.53	$4.61	$4.59	$4.34
EPS (diluted)	($27.43)	($1.44)	($1.42)	($1.39)	($0.83)	$0.75	$2.34	$3.67	$4.35	$4.44	$4.44	$4.20
Basic Shares (MM)	1.6	29.4	33.4	37.4	41.4	45.4	49.3	53.3	57.3	61.3	65.3	69.3
Diluted Shares (MM)	1.6	29.4	33.4	37.4	41.4	47.7	51.7	55.6	59.6	63.6	67.6	71.6

Source: Company data and Canaccord Adams estimates

Figure 25: AVEO quarterly income statement ($M except EPS)

	2009A FY2009	Mar-10 1Q10E	Jun-10 2Q10E	Sep-10 3Q10E	Dec-10 4Q10E	2010E FY2010
Collaboration revenues	20.7	5.0	18.5	5.0	5.0	33.6
Total Revenues	**20.7**	**5.0**	**18.5**	**5.0**	**5.0**	**33.6**
Research & Development	(51.8)	(14.9)	(15.2)	(15.7)	(16.3)	(62.2)
General & Administrative	(10.1)	(2.7)	(2.7)	(2.8)	(2.9)	(11.1)
Total Operating Expenses	**(61.9)**	**(17.6)**	**(18.0)**	**(18.5)**	**(19.2)**	**(73.3)**
Operating Income	(41.2)	(12.6)	0.5	(13.5)	(14.2)	(39.7)
Interest income	0.1	0.1	0.1	0.0	0.0	0.3
interest expense	(2.8)	(0.7)	(0.7)	(0.7)	(0.7)	(2.8)
Other Income, net	(0.3)	0.0	0.0	0.0	0.0	0.0
Pre-tax income	**(44.2)**	**(13.1)**	**(0.1)**	**(14.2)**	**(14.9)**	**(42.2)**
Tax benefit	0.1	0.0	0.0	0.0	0.0	0.0
Net Income	**(44.1)**	**(13.1)**	**(0.1)**	**(14.2)**	**(14.9)**	**(42.2)**
Basic shares	1.6	27.6	29.6	30.0	30.3	29.4
EPS	(27.4)	($0.47)	($0.00)	($0.47)	($0.49)	($1.44)

Source: Company data and Canaccord Adams estimates

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