Zacks Small-Cap Research

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Viking Therapeutics, Inc. (VKTX-NASDAQ)

VKTX: Additional Data Show Promise of VK2809 in Lipid Disorders…

Based on our probability adjusted DCF model that takes into account potential future revenues of VK5211, VK2809, and VK0214, VKTX is valued at $8/share. This model is highly dependent upon continued clinical success of those compounds and will be adjusted accordingly based upon future clinical results.

Current Price (11/15/16)	$1.16
Valuation	**$8.00**

OUTLOOK

On November 15, 2016 Viking Therapeutics, Inc. announced new clinical data from a previously completed Phase 1b trial of VK2809 in hypercholesterolemia. The results showed a rapid and significant reduction in levels of atherogenic proteins that are important markers of cardiovascular risk. These results build on the impressive reductions in LDL-cholesterol and triglycerides that were presented earlier this year and increase our confidence in the promise of VK2809's differentiated profile, and potentially de-risk the next expected efficacy readout. The company's ongoing Phase 2 trial of VK2809 in hypercholesterolemia and fatty liver disease is expected to be completed in 2Q17.

Viking also reported 3Q16 results on November 10. The company ended the quarter with $14.6 million in cash and cash equivalents, and entered an agreement during the quarter that could provide up to an additional $12.5 million in cash.

SUMMARY DATA

52-Week High	**$5.00**
52-Week Low	**$0.95**
One-Year Return (%)	**-76.80**
Beta	**0.56**
Average Daily Volume (sh)	**165,647**
Shares Outstanding (mil)	**20**
Market Capitalization ($mil)	**$23**
Short Interest Ratio (days)	**N/A**
Institutional Ownership (%)	**4**
Insider Ownership (%)	**20**
Annual Cash Dividend	**$0.00**
Dividend Yield (%)	**0.00**
5-Yr. Historical Growth Rates	
Sales (%)	**N/A**
Earnings Per Share (%)	**N/A**
Dividend (%)	**N/A**
P/E using TTM EPS	**N/A**
P/E using 2016 Estimate	**-0.9**
P/E using 2017 Estimate	**-1.1**

Risk Level	**High,**
Type of Stock	**Small-Blend**
Industry	**Med-Biomed/Gene**

ZACKS ESTIMATES

Revenue

(In millions of $)

	Q1 (Mar)	Q2 (Jun)	Q3 (Sep)	Q4 (Dec)	Year (Dec)
2015	0 A	0 A	0 A	0 A	0 A
2016	0 A	0 A	0 A	0 E	0 E
2017					0 E
2018					0 E

Earnings per Share

(EPS is operating earnings before non-recurring items)

	Q1 (Mar)	Q2 (Jun)	Q3 (Sep)	Q4 (Dec)	Year (Dec)
2015	-$1.40 A	- $1.07 A	-$0.53 A	-$0.57 A	-$3.68 A
2016	-$0.40 A	-$0.22 A	-$0.20 A	-$0.24 E	-$0.97 E
2017					-$0.89 E
2018					-$0.93 E

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WHAT'S NEW

Business Update

Viking Therapeutics, Inc. (VKTX) is a biopharmaceutical company developing treatments for metabolic and endocrine disorders. The company's lead compounds include: 1) VK5211, which is being developed for acute rehabilitation following non-elective hip fracture surgery; 2) VK2809, which is being developed for the treatment of hypercholesterolemia and fatty liver disease; and 3) VK0214, which is being developed for the treatment of X-linked adrenoleukodystrophy (X-ALD).

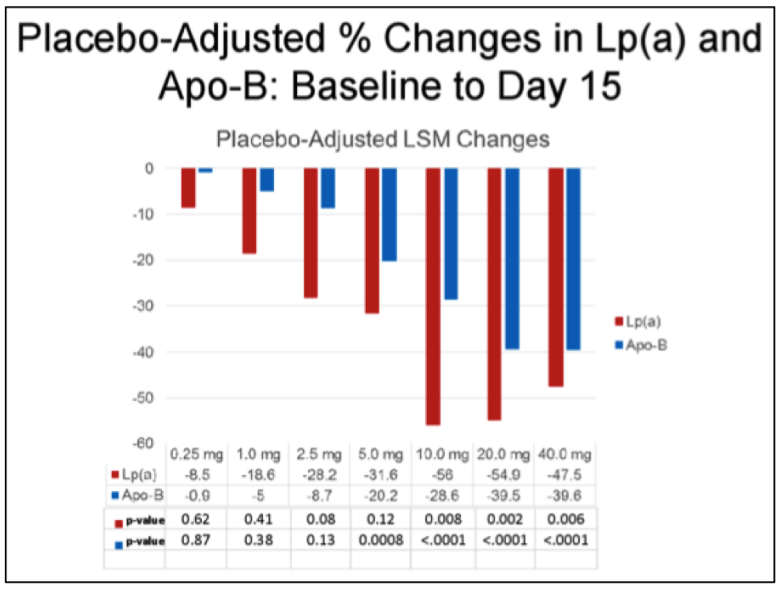
On November 10, 2016, Viking announced financial results for the third quarter of 2016 and provided a business update. The company reported a net loss of $3.8 million, or $0.20 per share, and exited the quarter with $14.6 million in cash and cash equivalents. During the third quarter, Viking entered into a $12.5 million common stock purchase agreement with Aspire Capital, providing additional access to capital on attractive terms

Viking has opened all planned US sites for the Phase 2 clinical study of VK5211 in hip fracture, and is on track to open 15 centers in Europe. For VK2809, more than half of the planned 30 study sites have been opened in the U.S. Each of those trials is expected to report topline data in the second quarter of 2017.

New Data Presented for VK2809

On Nov. 15, 2016 Viking announced additional data from its previously completed Phase 1b clinical trial of VK2809 in subjects with mild hypercholesterolemia. The data are from an analysis of the study participants' atherogenic protein levels, which demonstrated statistically significant reductions in lipoprotein(a) [Lp(a)] and apolipoprotein B-100 (apo B). The new results were highlighted in a poster presentation on November 14, 2016 at the American Heart Association (AHA) Scientific Sessions 2016 in New Orleans, LA.

As a reminder, data describing the effects on LDL-cholesterol and triglycerides were presented at the ACC conference in April (summarized below). The study was a randomized, double-blind, placebo-controlled Phase 1b trial designed to evaluate the safety, tolerability and pharmacokinetics of a range of VK2809 doses in 56 subjects with elevated serum cholesterol (n = 6 per drug-treated cohort). Following 14 days of VK2809 treatment, subjects experienced statistically significant placebo-adjusted, least square mean reductions in both Lp(a) and apo B across a range of doses. The following chart shows reductions in apo B ranged from 20.2% at 5 mg (P = 0.0008) to 39.6% at 40 mg (P < 0.0001) while reductions in Lp(a) ranged from 31.6% at 5 mg (P = 0.12) to 54.9% at 20 mg (P = 0.002). Comparable results were obtained with or without least-square mean adjustments, which account for covariates in patient characteristics.



	0.25 mg	1.0 mg	2.5 mg	5.0 mg	10.0 mg	20.0 mg	40.0 mg
Lp(a)	-8.5	-18.6	-28.2	-31.6	-56	-54.9	-47.5
Apo-B	-0.9	-5	-8.7	-20.2	-28.6	-39.5	-39.6
p-value	0.62	0.41	0.08	0.12	0.008	0.002	0.006
p-value	0.87	0.38	0.13	0.0008	<.0001	<.0001	<.0001

Source: Schoenfeld *et al*., 2016

The therapeutic importance of reducing atherogenic proteins has recently been highlighted in scientific literature. A report published in the *Mayo Clinic Proceedings* stated that Lp(a) was an independent, causal, risk factor for atherosclerosis, and that epidemiologic data show a continuous association between Lp(a) and cardiovascular disease (CVD) that is multiplied when both LDL-C and Lp(a) are elevated (Jacobson, 2013). Similarly, determination of apo B levels has been characterized as superior to any other cholesterol index to identify increased risk of CVD and assess the efficacy of lipid-lowering treatment (Barter *et al*., 2006). Thus, robust reductions of both Lp(a) and apo B, as demonstrated by VK2809 in this study, may add to the benefits of LDL-lowering therapy by further improving a patient's cardiovascular risk profile.

Cholesterol Lowering Properties of VK2809

On April 8, 2016, Viking presented positive Phase 1b clinical data for VK2809 in patients with hypercholesterolemia in a poster session at the 65th Annual Scientific Session and Expo of the American College of Cardiology. VK2809 was shown to be safe and well tolerated in doses ranging from 0.25 mg to 40 mg per day. No serious adverse events were reported and the frequency of adverse events in VK2809-treated subjects was similar to placebo-treated subjects. There were also no differences in heart rate, heart rhythm, or blood pressure between VK2809 and placebo-treated patients.

Results from the trial showed that treatment with VK2809 resulted in statistically significant placebo-adjusted reductions in low-density lipoprotein (LDL) cholesterol of 15.2% at the 5 mg dose to 41.2% at the 20 mg dose (P<0.05 for both doses). In addition, decreases in triglycerides of as much as 78.6% were seen at the 40 mg dose. The following table summarizes the placebo-adjusted percentage decreases in LDL cholesterol, triglycerides, total cholesterol, high-density lipoprotein (HDL) cholesterol, and non-HDL cholesterol.

Dose	Placebo-Adjusted LSM (SE)				
	LDL-C	Triglyceride	TC	HDL-C	Non-HDL-C
0.25 mg (N=6)	10.5 (6.6)	-9.6 (17.5)	4.0 (4.9)	2.3 (7.0)	6.0 (7.1)
1 mg (N=6)	-9.2 (6.6)	9.7 (17.1)	-2.4 (5.0)	-2.2 (6.9)	-2.6 (7.1)
2.5 mg (N=6)	-0.7 (6.6)	-36.6 (17.3)	-1.1 (4.9)	11.2 (6.9)	-4.5 (7.0)
5 mg (N=6)	-15.2 (6.6)	-34.8 (17.4)	-15.7 (4.9)	-4.1 (6.9)	-19.9 (7.0)
10 mg (N=5)	-27.1 (7.0)	-61.0 (18.5)	-24.2 (5.4)	-3.0 (7.5)	-32.0 (7.5)
20 mg (N=6)	-41.2 (6.6)	-62.1 (17.2)	-29.8 (5.0)	16.5 (6.9)	-44.2 (7.1)
40 mg (N=5)	-36.6 (7.0)	-78.6 (18.7)	-32.1 (5.3)	-2.7 (7.3)	-42.2 (7.5)

Source: Lian *et al*., 2016

The table below gives a comparison of the Phase 1b efficacy results of VK2809 and data from existing hyperlipidemia treatments.

Drug	Class	LDL-C[1]	Triglycerides	Lp(a)	Apo-B	Safety, Tolerability, Other
VK2809, 10 mg	**TR-β**	**-27**	**-61**	**-55**	**-29**	**Expect safe, additive efficacy with statins; efficacy in statin-intolerant**
Niacin, 1500 mg	Nicotinic acid	-13	-25	-17	-13	Flushing, increased plasma glucose, liver toxicity, skeletal muscle toxicity
Ezetimibe, 10 mg	Cholesterol absorption inhibitor	-19	-8	-10	-14	Modest efficacy
Colesevelam, 3.8 g	Bile acid sequestrant	-15	+5	-	-12	Modest efficacy, gastrointestinal tolerability
Atorvastatin 20 mg	Statin	-47	-36	-	-38	Skeletal muscle toxicity/pain, potential elevation in diabetes risk
Fenofibrate, 145 mg	Fibrate	-18	-36	-	-28	Gallstones, risk of kidney dysfunction, skeletal muscle toxicity

Source: Viking Therapeutics, Inc.

VK2809 Phase 2 Clinical Trial

The preceding data is the basis for Viking's Phase 2 clinical trial to evaluate VK2809 as a treatment for both hypercholesterolemia and fatty liver disease (NCT02927184). The company is enrolling patients with elevated cholesterol, fatty liver disease, and at least three risk factors for metabolic syndrome, which is considered a major driver for the onset of nonalcoholic steatohepatitis (NASH). The primary endpoint will assess changes in LDL following 12 weeks of treatment, with exploratory endpoints evaluating changes in liver fat content, inflammatory markers, and histological changes. Upon conclusion, the company hopes to be in a position to move forward in either hypercholesterolemia or NASH. Thus, it could be viewed as a two-in-one study – confirmatory on LDL and exploratory for fatty liver disease. We anticipate topline results in the second quarter of 2017.

VK5211

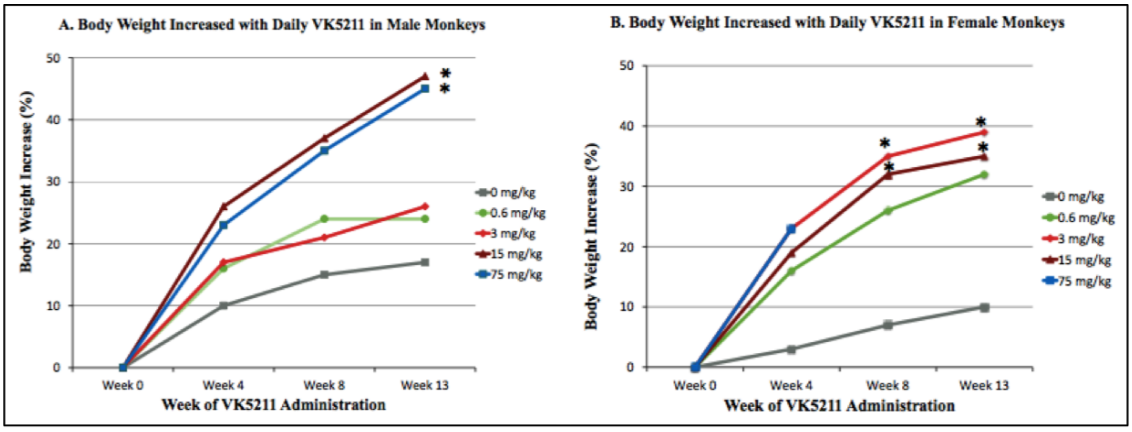
Viking's most advanced drug candidate is VK5211, a third-generation non-steroidal Selective Androgen Receptor Modulator (SARM) that is being developed for maintenance or improvement of lean body mass (LBM), bone mineral density (BMD), and function in patients recovering from non-elective hip fracture surgery. Hip fracture is associated with a number of morbidities, the majority of which are the result of deleterious changes in body composition following the injury. In the first year after a hip fracture, fat mass increases by up to 7% (Karlsson *et al*., 1996) while lean mass decreases by up to 11% (Fox *et al*., 2000). This is in comparison to healthy older females who lose approximately 1% of lean mass per year and gain approximately 1.7% in fat mass (Karlsson *et al*., 2000).

SARMs are a group of compounds designed to act as androgen receptor (AR) agonists in muscle and bone while being partial agonists in other areas of the body (e.g., prostate). The most prominent androgen, testosterone, stimulates the growth of muscle and bone (anabolic effects) as well as the prostate and sebaceous glands (androgenic effects), and is considered a non-tissue-selective androgen.

While androgens inhibit fat accumulation and increase skeletal muscle growth, two properties that make them ideal therapeutic candidates for restoring or preserving body composition following hip fracture, the use of testosterone therapy has a number of side effects including prostate growth (Meikle *et al*., 1997) and polycythemia (Snyder *et al*., 2000) in men and acne, alopecia, and hirsutism in women (Phillips *et al*., 1997) that precludes its use in a large number of patients. Thus, what would be most beneficial would be a product that provided the anabolic effects of androgen therapy with limited androgenic effects.

VK5211 was originally developed by Ligand Pharmaceuticals, Inc. (LGND) and was previously tested in preclinical models and early stage clinical trials. Two Phase 1 clinical trials showed the drug to be safe and well-tolerated at all doses following daily oral administration for up to 21 days. The drug selectively activates AR in muscle and bone, stimulating muscle and bone growth, while avoiding undesirable side effects, such as unwanted hair growth, acne, or prostate growth.

To examine the safety and physiological changes that occur after 13 weeks of VK5211 dosing, cynomolgus monkeys were orally administered VK5211 once daily at 0, 0.6, 3, 15, or 75 mg/kg. The following figure shows a significant increase in body weight in both male and female monkeys during the 13 weeks of dosing. The fact that the results were seen in females is important, as the majority of hip fractures occur in females.



Source: Bleavins *et al*., 2015

VK5211 Phase 2 Clinical Trial

In November 2015, Viking initiated a Phase 2 study in patients ≥ 65 years of age who have suffered a hip fracture within the past three to seven weeks. This is a multicenter, randomized, parallel group, double blind, placebo controlled trial, where patients will be administered placebo or 0.5 mg, 1.0 mg, or 2.0 mg of VK5211 once-daily for 12 weeks (NCT02578095). A total of 120 patients are expected to enroll in the trial evenly split between the four treatment groups. The primary outcome of the trial is the change in LBM after 12 weeks of treatment. Secondary and exploratory endpoints include assessments of functional performance, quality-of-life, and activities of daily living. In the third quarter update, Viking noted that all clinical sites in the U.S. are opened while 30 of the planned 33 international sites have been opened, including 15 European sites. We anticipate topline results will be reported in the second quarter of 2017.

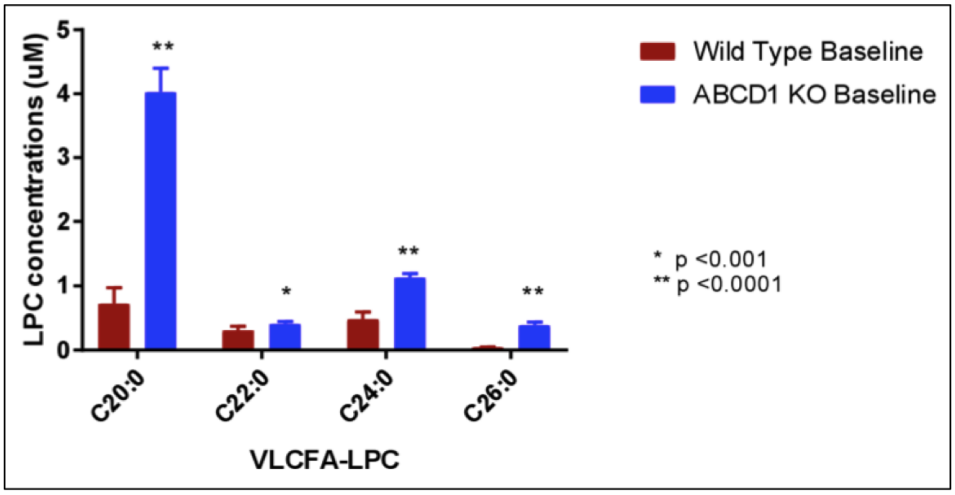
VK0214

VK0214 is a thyrod receptor beta (TRβ) agonist being developed for the treatment of X-linked adrenoleukodystrophy (X-ALD), an orphan neurodegenerative disease that affects approximately 8,000 individuals in the U.S. and 12,000 in Europe. In contrast to VK2809, which is activated by the liver specific cytochrome P450 isoenzyme CYP3A4, VK0214 is a TRβ agonist that is activated by carboxyesterases that are ubiquitously expressed in the body. The drug also has a different pharmacokinetic and pharmacodynamic profile, thus potentially making the drug more suitable for a disease such as X-ALD, which is more diffuse than hypercholesterolemia or fatty liver disease.

On September 26, 2016, Viking presented positive proof-of-concept preclinical data showing that VK0214 is active in a mouse model of X-ALD. For a review of the scientific rationale for using VK0214 in X-ALD, please see our previous report.

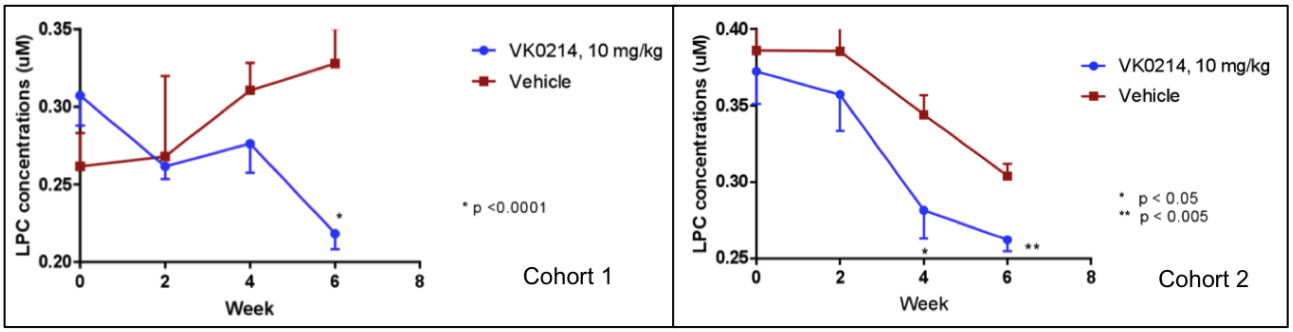
VK0214 Preclinical Data

To study the potential use of VK0214 in X-ALD, Viking tested VK0214 in the ABCD1 knockout (KO) mouse model, and while this model does not recapitulate the inflammation seen with severe forms of X-ALD, these mice do develop a phenotype similar to adrenomyeloneuropathy **(**AMN) with advanced age along with an accumulation of certain very long chain fatty acids (VLCFAs) in tissues and plasma (Lu *et al*., 1997). The following graph shows an accumulation of different VLCFAs in the blood of ABCD1 KO mice used in this experiment compared to wild-type controls. VLCFAs are denoted as “CX:Y” with “X” corresponding to the number of carbons in the fatty acid chain and “Y” corresponding to the number of double bonds in the chain.



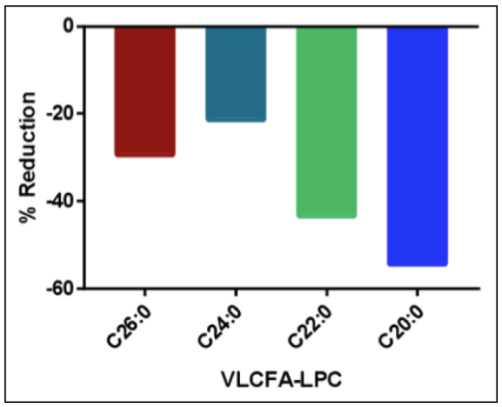
Source: Masamune *et al*., 2016

A pilot experiment was performed in 16 ABCD1 KO mice randomized 3:1 to receive VK0214 or placebo once daily for six weeks. Mice receiving VK0214 demonstrated rapid reductions in the mean level of C26:0, while control animals saw no reduction. After six weeks of treatment, mice receiving VK0214 had a 40% reduction in whole blood C26:0 levels relative to controls (P<0.0001). A second cohort of 20 mice randomized 1:1 to receive VK0214 or placebo for six weeks again showed a statistically significant decrease in C26:0 levels in mice treated with VK0214 (P<0.005). In the second cohort of mice, those receiving placebo also showed a reduction in C26:0 levels, which may have been due to the lipid-based nature of the vehicle, similar to results seen with Lorenzo’s Oil.



Source: Masamune *et al*., 2016

Plasma taken from Cohort 2 mice was analyzed for the presence of different VLCFAs at two, four, and six weeks. The results show mean reductions of approximately 20-60% in the levels of C26:0, C24:0, C22:0, and C20:0 in mice treated with VK0214 compared to placebo at week six, as shown in the following figure. The importance of reducing shorter chain VLCFAs is unclear, however it may suggest a potential reduction in available substrates for elongation to the more problematic C24:0 and C26:0 VLCFAs, leading to even greater long term reductions in these longer chain VLCFAs.



Source: Masamune *et al*., 2016

Overall, we believe the positive preclinical data reported here provide strong support for the development of VK0214 for X-ALD. The data presented by Viking are in alignment with the hypothesis that induction of *ABCD2* expression leads to increased metabolism and clearance of VLCFAs and treatment with VK0214 could represent a novel treatment options for X-ALD patients. The company is continuing work on characterizing the long-term impact of VK0214 treatment on VLCFA accumulation in tissues. We anticipate IND-enabling studies to continue and for the company to file an IND and initiate a clinical trial with VK0214 in the second half of 2017.

Financial Update

On November 10, 2016, Viking announced financial results for the third quarter of 2016. The company reported a net loss of $3.8 million, or $0.20 per share, which was comprised of $2.1 million in R&D expenses and $1.2 million in G&A expenses. The company exited the second quarter with approximately $14.6 million in cash and cash equivalents. In August 2016, Viking entered into a $12.5 million common stock purchase agreement with Aspire Capital Fund, LLC. Under the terms of the agreement, Aspire made an initial $500,000 purchase of Viking common stock and has committed to purchase up to an additional $12 million in shares of common stock over the next 30 months, with the timing and amount of any sale of shares of common stock to Aspire Capital controlled by Viking. In addition, in June 2016 Viking entered into a distribution agreement with Maxim, whereby Viking can sell up to approximately $5 million in common stock at prevailing market prices. Taken together, we believe Viking has sufficient access to capital for at least the next twelve months, which is importantly after the expected data readouts for the ongoing Phase 2 clinical trial.

As of Oct. 31, 2016, Viking had approximately 20.2 million shares of common stock outstanding, with Ligand Pharmaceuticals owning approximately 36% of the total number of outstanding shares. When factoring in the outstanding shares of restricted stock, stock options, and warrants the company has a fully diluted share count of approximately 33.8 million shares.

Valuation and Conclusion

For valuation purposes, we have constructed a probability adjusted discounted cash flow model that takes into account potential revenues from VK5211 in hip fracture, VK2809 in NASH, and VK0214 in ALD.

We model for peak revenues for VK5211 of approximately $600 million in the U.S. and $1 billion in the E.U. based on compelling preclinical and early clinical data. Viking is expected to announce data from the ongoing Phase 2 study of VK5211 for the treatment of hip fracture in the second quarter of 2017.

We estimate potential peak worldwide revenues for VK2809 of $2.5 billion based upon the low level of adverse events seen with the drug in early clinical testing along with data that is just as good if not better than current treatment options for dyslipidemia. We anticipate topline results from the recently initiated Phase 2 study of VK2809 in hypercholesterolemia and fatty liver disease in the second quarter of 2017.

For ALD, since it is an orphan indication we believe the drug would likely command premium pricing, thus we model for a yearly cost of $150,000 in the U.S. and $120,000 in Europe, leading to estimated peak worldwide sales of approximately $450 million.

Based on these numbers, and using an 18% discount rate, we arrive at a valuation of $8/share. We continue to believe that Viking's shares are significantly undervalued. This is particularly evident on a comparative basis with Madrigal Pharmaceuticals, Inc. (MDGL), which is developing a Phase 2 ready TRβ agonist for the treatment of fatty liver disease. We believe Viking's TRβ agonists have potentially superior efficacy and fail to see why Viking is trading at approximately 1/8th the valuation of Madrigal. Small-cap biotech investors should take a serious look at Viking ahead of data readouts in the second quarter of 2017.

PROJECTED FINANCIALS

Viking Therapeutics, Inc.
Income Statement

Viking Therapeutics, Inc.	2015 A	Q1 A	Q2 A	Q3 A	Q4 E	2016 E	2017 E	2018 E
VK5211 (Hip Fracture)	$0	$0	$0	$0	$0	$0	$0	$0
YOY Growth	-	-	-	-	-	-	-	-
VK2809 (Hypercholesterolemia)	$0	$0	$0	$0	$0	$0	$0	$0
YOY Growth	-	-	-	-	-	-	-	-
VK2809/VK0214 (ALD)	$0	$0	$0	$0	$0	$0	$0	$0
YOY Growth	-	-	-	-	-	-	-	-
Grants & Collaborative Revenue	$0	$0	$0	$0	$0	$0	$0	$0
YOY Growth	-	-	-	-	-	-	-	-
Total Revenues	**$0**	**$0**	**$0**	**$0**	**$0**	**$0**	**$0**	**$0**
YOY Growth	-	-	-	-	-	-	-	-
Cost of Sales	$0	$0	$0	$0	$0	$0	$0	$0
Product Gross Margin	-	-	-	-	-	-	-	-
Research & Development	$7.0	$1.9	$2.4	$2.1	$2.9	$9.3	$13.5	$16.0
General & Administrative	$5.0	$1.4	$1.2	$1.2	$1.4	$5.2	$5.8	$6.0
Other Expenses	$0	$0	$0	$0	$0	$0	$0	$0
Operating Income	($12.0)	($3.3)	($3.6)	($3.3)	($4.3)	($14.4)	($19.3)	($22.0)
Operating Margin	-	-	-	-	-	-	-	-
Non-Operating Expenses (Net)	($11.4)	($0.3)	($0.1)	($0.5)	($0.5)	($1.5)	($2.0)	($3.0)
Pre-Tax Income	($23.4)	($3.6)	($3.7)	($3.8)	($4.8)	($15.9)	($21.3)	($25.0)
Income Taxes Paid	$0	$0	$0	$0	$0	$0	$0	$0
Tax Rate	*0%*	*0%*	*0%*	*0%*	*0%*	*0%*	*0%*	*0%*
Net Income	**($23.4)**	**($3.6)**	**($3.7)**	**($3.8)**	**($4.8)**	**($15.9)**	**($21.3)**	**($25.0)**
Net Margin	-	-	-	-	-	-	-	-
Reported EPS	**($3.68)**	**($0.40)**	**($0.22)**	**($0.20)**	**($0.24)**	**($0.97)**	**($0.89)**	**($0.93)**
YOY Growth	-	-	-	-	-	-	-	-
Basic Shares Outstanding	6.356	9.016	17.105	18.992	20.000	16.278	24.000	27.000

Source: Zacks Investment Research, Inc. *David Bautz, PhD*

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HISTORICAL STOCK PRICE



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