Zacks Small-Cap Research

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Viking Therapeutics, Inc. (VKTX-NASDAQ)

VKTX: Data From Phase 2 Trial of VK5211 Expected in 4Q17…

Based on our probability adjusted DCF model that takes into account potential future revenues of VK5211, VK2809, and VK0214, VKTX is valued at $7/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 (08/14/17)	$1.02
Valuation	**$7.00**

OUTLOOK

Viking Therapeutics, Inc. (VKTX) has a number of important data readouts anticipated over the next six months. The Phase 2 study of VK5211 for the treatment of hip fracture is now fully enrolled and we anticipate topline data being reported in the fourth quarter of 2017. The Phase 2 study of VK2809 in hypercholesterolemia and fatty liver disease is continuing to enroll patients and we anticipate topline data being reported in the first half of 2018. An extended preclinical study of VK0214 in a mouse model of X-linked adrenoleukodystrophy recently completed, and we anticipate results from this study being reported in the third quarter of 2017. These data represent clear inflection points for the stock, with positive results likely to cause a significant revaluation of the shares.

SUMMARY DATA

52-Week High	**$1.69**
52-Week Low	**$0.93**
One-Year Return (%)	**-22.14**
Beta	**1.09**
Average Daily Volume (sh)	**218,645**
Shares Outstanding (mil)	**28**
Market Capitalization ($mil)	**$28**
Short Interest Ratio (days)	**N/A**
Institutional Ownership (%)	**3**
Insider Ownership (%)	**8**
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)
2016	0 A	0 A	0 A	0 A	0 A
2017	0 A	0 A	0 E	0 E	0 E
2018					0 E
2019					0 E

Earnings per Share
(EPS is operating earnings before non-recurring items)

	Q1 (Mar)	Q2 (Jun)	Q3 (Sep)	Q4 (Dec)	Year (Dec)
2016	-$0.40 A	- $0.22 A	-$0.20 A	-$0.18 A	-$0.90 A
2017	-$0.23 A	-$0.21 A	-$0.19 E	-$0.19 E	-$0.81 E
2018					-$0.72 E
2019					-$0.70 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.
   - Viking anticipates data from the ongoing Phase 2 study of VK5211 in hip fracture in 4Q17.
2) **VK2809**, which is being developed for the treatment of hypercholesterolemia and fatty liver disease, with work set to begin in the second half of 2017 in glycogen storage disease type Ia (GSD Ia).
   - Viking anticipates data from the ongoing Phase 2 study of VK2809 in fatty liver disease in the first half of 2018, and to initiate a human proof-of-concept study in GSD Ia in the second half of 2017.
   - Detailed data from a study of VK2809 in an *in vivo* model of NASH will be presented at the AASLD conference in October.
3) **VK0214**, which is being developed for the treatment of X-linked adrenoleukodystrophy (X-ALD).
   - A long-term study in an *in vivo* model of X-ALD has recently finished, with results expected in the third quarter of 2017.

VK5211

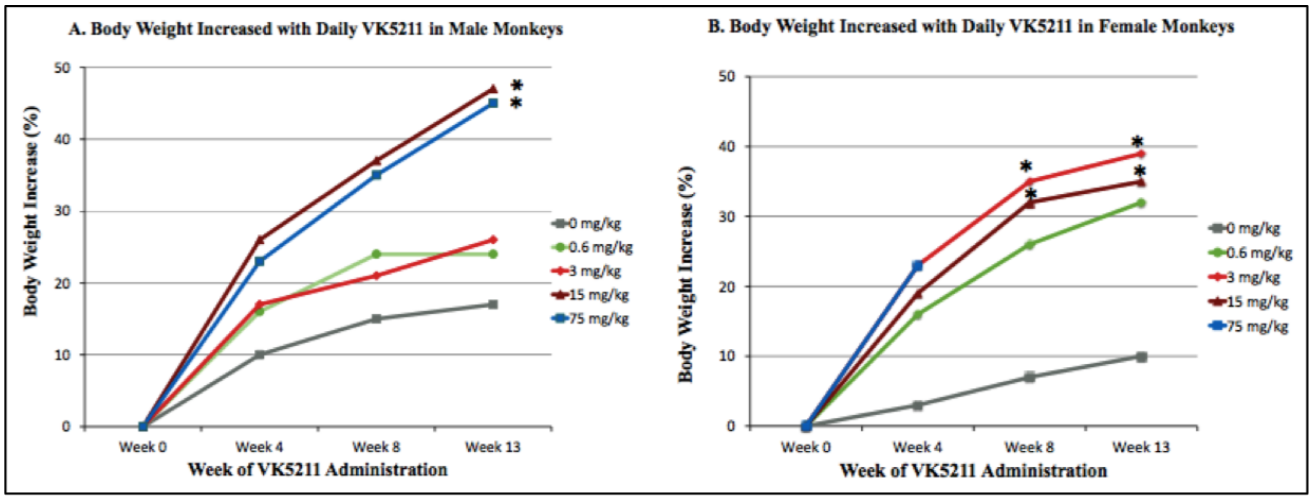
Viking's most advanced drug candidate is VK5211, a third-generation non-steroidal Selective Androgen Receptor Modulator (SARM) that is being developed for maintenance or improvement of lean body mass (LBM), bone mineral density (BMD), and function in patients recovering from non-elective hip fracture 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. Three 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 are being administered placebo or 0.5 mg, 1.0 mg, or 2.0 mg of VK5211 once-daily for 12 weeks (NCT02578095). 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.

On July 12, 2017, Viking announced enrollment has been completed with 108 total subjects and we expect topline results to be announced in the fourth quarter of 2017. We anticipate the company meeting with the FDA following release of the data to determine the next steps, including the design of future studies. At this point it is difficult to estimate the size and costs of these future studies and we'll await further guidance from the company once they've had a chance to meet with the FDA.

VK2809

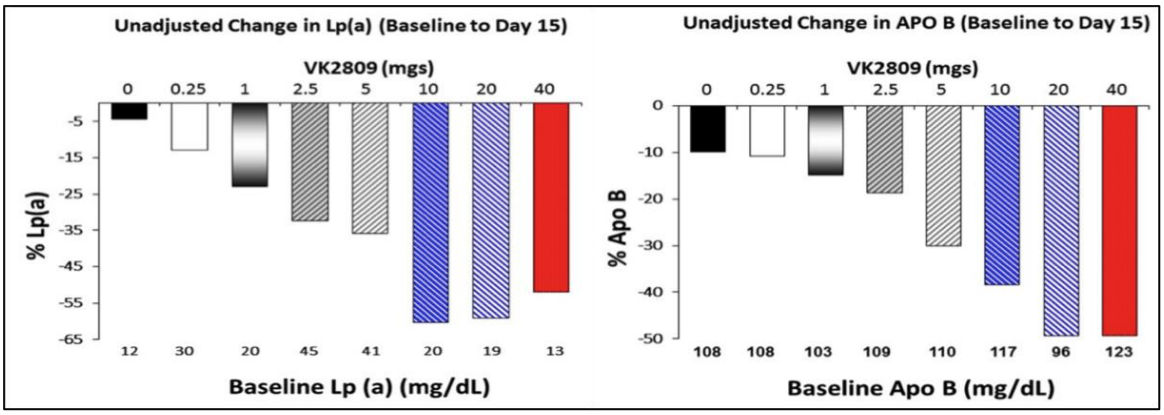
VK2809 (formerly known as MB07811) is a novel, orally available, selective thyroid hormone receptor (TR) agonist that is in development for lipid disorders such as hypercholesterolemia and fatty liver disease. It was originally developed by Metabasis Therapeutics, Inc., which was acquired by Ligand in 2009. The compound has been tested in multiple preclinical models as well as two Phase 1 clinical trials.

There are two major isoforms of TR, TRα and TRβ, which are encoded by separate genes. TRα and TRβ also have markedly different expression patterns, with TRα expression highest in the heart and brain while TRβ expression is highest in the liver (Bookout *et al*., 2006). VK2809 is a prodrug of a potent TRβ agonist that is converted to the active compound through cleavage by the liver specific cytochrome P450 isoenzyme CYP3A4 (Erion *et al*., 2007). The activated form of the drug has approximately 16-fold higher affinity for TRβ (Ki = 2.2 nM) than for TRα (Ki = 35.2 nM).

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. The Phase 1b trial was a 14-day, placebo controlled trial in patients with mild hypercholesterolemia (defined as baseline LDL cholesterol of at least 100 mg/dL). 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. Notably, this presentation received a "Best Poster" award from the American College of Cardiology.

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.

Statistically significant reductions of lipoprotein a (Lp(a)) and apolipoprotein B (Apo(B)), which are believed to be positively associated with a patient's risk of developing cardiovascular disease, were also observed in certain cohorts as shown in the following figure.



Source: Lian *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 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.

VK2809 Phase 2 Clinical Trial

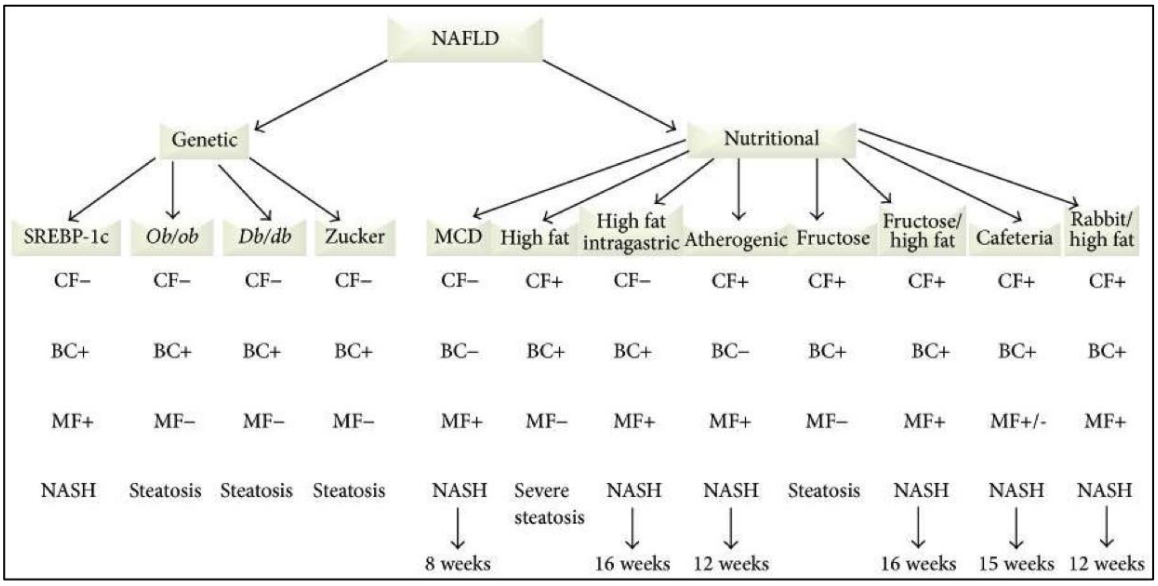
Viking is currently conducting a Phase 2 clinical trial of VK2809 as a treatment for both hypercholesterolemia and fatty liver disease. The company is enrolling patients with elevated cholesterol, fatty liver disease, and at least three risk factors for metabolic syndrome, which is considered a major 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 first half of 2018, as the company has indicated enrollment is taking a bit longer than anticipated due to increased competition for these types of patients as well as relatively stringent enrollment criteria. The company recently amended the trial protocol, after consultation with the FDA, to loosen some of the enrollment criteria that could help to speed up enrollment. For example, the trial currently requires participants to have at least 130 mg/dL LDL, thus that criterion may be lowered a bit as LDL is a secondary interest to the company.

VK2809 NASH In Vivo *Model*

Several animal models have been developed to represent the various pathophysiologic changes, morphologic changes, biochemical changes, and clinical features of NASH (Sanches *et al*., 2015). These models can be classified as nutritional (diet-induced), genetic, or a combination of nutritional and genetic. A robust model should recapitulate all of the hallmarks of the disease, including changes in metabolic profile, steatosis, inflammation, hepatocellular ballooning, fibrosis, and tumor susceptibility. The genetic models are typically best at inducing biochemical changes seen in NASH (e.g., increases in liver triglyceride and cholesterol), while the different nutritional models typically result in histopathological changes that are consistent with NASH, but lack the biochemical changes. The following chart shows the different animal models currently utilized to study NASH, with the fructose/high-fat diet-induced model best able to recapitulate the main clinical features (CF), biochemical changes (BC), morphological findings (MF), and the occurrence of NASH. We believe it is important for investors to understand that there are a number of different NASH models, thus while some compounds may show robust

activity in a certain NASH model, if that model does not best represent what is seen in the clinic it is likely of little use.



Source: Sanches *et al*., 2015

Viking utilized a mouse model of NASH using wild-type mice fed a diet that consisted of 40% fat (18% trans fat), 40% carbohydrate (20% fructose), and 2% cholesterol (Clapper *et al*., 2013). This diet has previously been shown to induce steatosis, steatohepatitis with fibrosis, and cirrhosis and mimics what may be seen in individuals with poor dietary habits. Following 33 weeks on the diet, a biopsy was performed to confirm the presence of disease characteristics, including fibrosis. Mice were then treated for eight weeks with VK2809 (10 mg/kg/day), vehicle control, or an undisclosed active control that is in late-stage clinical development for NASH. Results from a study using this same model to examine the effects of treatment with liraglutide, elafibranor, and obeticholic acid were recently presented (Feigh *et al*., 2017), thus we believe the active comparator may be one of those compounds.

VK2809 Shows Robust Activity in NASH Model

While we will have to wait for the full data set to be presented later this year at a scientific conference, the preliminary data that Viking disclosed is quite impressive. The following table shows the results obtained with VK2809 compared to both vehicle control and active control. For all parameters, VK2809 performed statistically significantly better than vehicle control. Compared to active control, VK2809 was statistically significantly better than for liver triglyceride and liver cholesterol content, and there were clear trends toward significance for liver collagen and hydroxyproline. A recent presentation showed that liver collagen significantly correlated with fibrosis in patients with non-alcoholic fatty liver disease (NAFLD), a precursor to NASH (Buzzetti *et al*., 2017). Fibrosis is a known risk-factor for adverse outcomes in patients with NAFLD (Angulo *et al*., 2015).

Parameter	VK2809 Compared to Vehicle Control	*P* value	VK2809 Compared to Active Control	*P* value
Liver Triglycerides	↓ 70%	<0.0001	↓ 56%	<0.0001
Liver Cholesterol	↓ 65%	<0.0001	↓ 58%	<0.0001
Liver Fibrosis	↓ 50%	0.01	↓ 21%	0.3
Liver Collagen	↓ 60%	<0.005	↓ 49%	0.07
Liver Hydroxyproline	↓ 46%	0.01	↓ 36%	0.06

Source: Viking Therapeutics, Inc. / Zacks SCR

VK2809 was also superior in a number of other outcomes. Animals treated with VK2809 experienced a 40% mean improvement in non-alcoholic fatty liver disease activity score (NAS; a composite measure of disease activity that is comprised of steatosis, ballooning, and inflammation) compared to animals treated with vehicle (*P*<0.0001). Fifty percent of VK2809-treated animals improved by at least two points in NAS, compared to no vehicle-treated animals (*P*=0.01), and no animals treated with VK2809 experienced a worsening in NAS while 60% of vehicle-treated

animals did (*P*<0.0001). Lastly, there were no unexpected or abnormal laboratory findings for VK2809-treated animals and all animals received all scheduled doses of drug.

The company recently completed an analysis of gene expression changes from this study. These types of analyses can provide important clues as to the underlying mechanisms responsible for the observed results. The results from this analysis are expected in the third quarter of 2017.

VK2809 in Glycogen Storage Disease Ia

On February 9, 2017, Viking Therapeutics, Inc. (VKTX) announced successful interim results from a proof-of-concept study of VK2809 in an *in vivo* model of glycogen storage disease 1a (GSD 1a). The study is ongoing, and full results will be presented at an upcoming scientific conference later this year, most likely the International Congress of Inborn Errors of Metabolism in September 2017.

Initial results from the study showed that treatment with VK2809 led to statistically significant reductions in key metabolic markers of GSD Ia, including a reduction in mean liver triglyceride content of more than 60%, a reduction in average liver weight of more than 30%, and a reduction in average liver weight as a percentage of total body weight by approximately 20%. The study utilized a glucose-6-phosphatase knockout mouse model that recapitulates a number of the phenotypic outcomes commonly seen in GSD 1a patients (Lei *et al*., 1996). For a full review of GSD Ia and the rationale for using VK2809 please see our previous report.

Importantly, these data provide the scientific rationale for moving into a proof-of-concept study in humans in the second half of 2017. While final details are yet to be determined, we anticipate the study will likely involve up to 30 patients dosed for four weeks with the primary outcome being the change in plasma triglycerides from the beginning to the end of the study. We would expect the company to assess liver fat as well, potentially as an exploratory endpoint.

On February 14, 2017, Viking announced an agreement with PoC Capital, LLC to fund the initial development of VK2809 in GSD 1a. PoC will be responsible for paying up to $1.8 million in expenses associated with VK2809 clinical studies that includes proof-of-concept studies in patients with GSD 1a. Viking will issue up to $1.8 million in common shares to PoC in exchange for the funding.

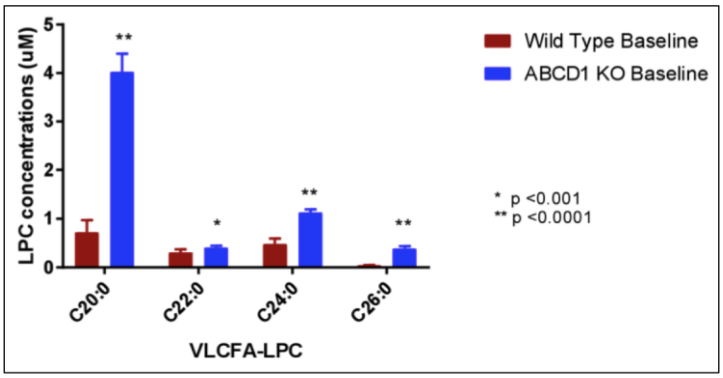
VK0214

VK0214 is a thyroid 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.

For a detailed review of the scientific rationale for using VK0214 in X-ALD, please see our previous report.

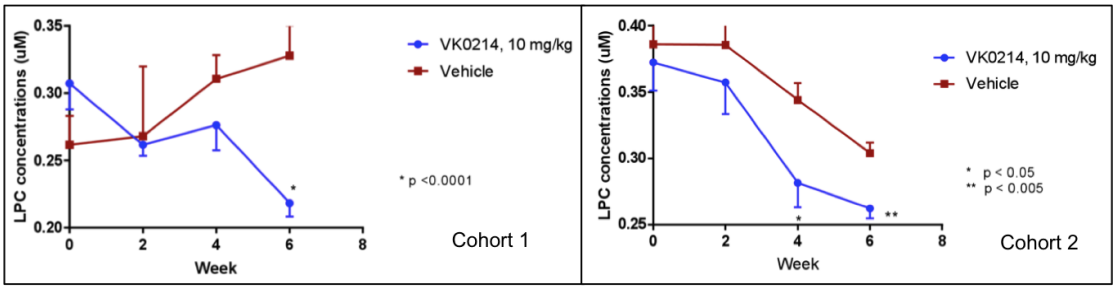
VK0214 Preclinical Data

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.



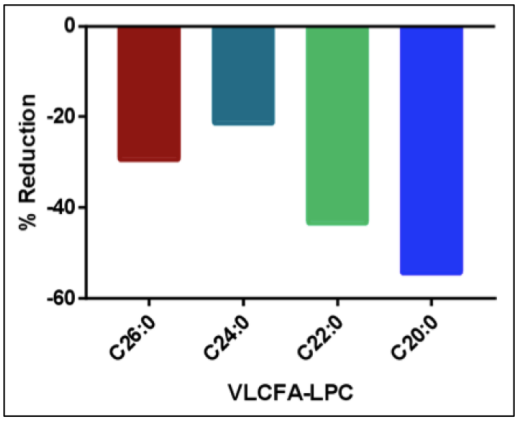
Source: Masamune *et al.*, 2016

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 in the same model, and recently announced that a 25-week study of VK0214 in the ABCD1 KO model has completed. We anticipate initial results from this long term study to be reported in the third quarter of 2017.

Financial Update

On August 9, 2017, Viking announced financial results for the second quarter of 2017. As expected, the company did not report any revenues. Net loss for the quarter was $5.2 million, or $0.21 per share. The loss included $3.7 million in R&D expenses, compared with $2.4 million in the second quarter of 2016. The increase was due to increased clinical trial activity for VK5211 and VK2809 and preclinical activity for VK0214. G&A expenses for the second quarter of 2017 totaled $1.3 million, compared to $1.2 million in the second quarter of 2016. An increase in salaries in benefits was offset by a decrease in stock compensation.

Viking exited the second quarter of 2017 with approximately $12.0 million in cash, cash equivalents, and marketable securities. During the quarter, the company completed a registered direct offering of approximately 3.7 million shares of common stock along with approximately 2.8 million warrants for net proceeds of approximately $3.9 million. We estimate the company has sufficient capital to fund operations into the second quarter of 2018.

As of July 31, 2017, Viking had approximately 27.7 million shares of common stock outstanding and when factoring in the outstanding shares of restricted stock, stock options, and warrants the company has a fully diluted share count of approximately 44.5 million.

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. We look forward to analyzing the data from the ongoing Phase 2 study of VK5211 for the treatment of hip fracture in the fourth 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 first half of 2018.

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 $7/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 TRβ agonist that is also in Phase 2 testing 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 1/7th the valuation of Madrigal. Small-cap biotech investors should take a serious look at Viking ahead of the multiple data readouts expected later in 2017.

PROJECTED FINANCIALS

Viking Therapeutics, Inc.
Income Statement

Viking Therapeutics, Inc.	2016 A	Q1 A	Q2 A	Q3 E	Q4 E	2017 E	2018 E	2019 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	-	-	-	-	-	-	-	-
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	$9.0	$3.5	$3.7	$3.7	$3.8	$14.7	$16.0	$18.0
General & Administrative	$4.8	$1.4	$1.3	$1.3	$1.3	$5.3	$5.7	$6.0
Other Expenses	$0	$0	$0	$0	$0	$0	$0	$0
Operating Income	($13.8)	($5.0)	($5.0)	($5.0)	($5.1)	($20.1)	($21.7)	($24.0)
Operating Margin	-	-	-	-	-	-	-	-
Non-Operating Expenses (Net)	($0.9)	($0.3)	($0.2)	($0.1)	($0.1)	($0.6)	($0.5)	($0.6)
Pre-Tax Income	($14.7)	($5.22)	($5.17)	($5.1)	($5.2)	($20.7)	($22.2)	($24.6)
Income Taxes Paid	($0)	$0	$0	$0	$0	$0	$0	$0
Tax Rate	*0%*	*0%*	*0%*	*0%*	*0%*	*0%*	*0%*	*0%*
Net Income	**($14.7)**	**($5.2)**	**($5.2)**	**($5.1)**	**($5.2)**	**($20.7)**	**($22.2)**	**($24.6)**
Net Margin	-	-	-	-	-	-	-	-
Reported EPS	**($0.90)**	**($0.23)**	**($0.21)**	**($0.19)**	**($0.19)**	**($0.81)**	**($0.72)**	**($0.70)**
YOY Growth	-	-	-	-	-	-	-	-
Basic Shares Outstanding	16.278	22.353	24.119	27.500	28.000	25.493	31.000	35.000

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

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HISTORICAL STOCK PRICE



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