Zacks Small-Cap Research

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Viking Therapeutics, Inc. (VKTX-NASDAQ)

VKTX: Data From Phase 2 Trial of VK5211 to be Reported Soon…

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 (11/09/17)	$2.15
Valuation	**$7.00**

OUTLOOK

Viking Therapeutics, Inc. (VKTX) is set to enter a critical period over the next six months. The company will be announcing topline results from the Phase 2 clinical trial of VK5211 in hip fracture before the end of 2017 and results from the Phase 2 clinical trial of VK2809 in the first half of 2018. Positive results from either (or both) of those trials would result in a significant revaluation of the company's share price. In addition, Viking will be filing an IND before the end of 2017 to initiate a proof-of-concept clinical trial of VK2809 in patients with glycogen storage disease 1a (GSD1a).

SUMMARY DATA

52-Week High	**$3.00**
52-Week Low	**$0.93**
One-Year Return (%)	**115.00**
Beta	**1.65**
Average Daily Volume (sh)	**993,411**
Shares Outstanding (mil)	**28**
Market Capitalization ($mil)	**$61**
Short Interest Ratio (days)	**N/A**
Institutional Ownership (%)	**6**
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)
2016	0 A	0 A	0 A	0 A	0 A
2017	0 A	0 A	0 A	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	-$0.22 A	-$0.19 E	-$0.85 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, along with the orphan disease 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.
   - Data from a study of VK2809 in an *in vivo* model of NASH was recently reported (discussed below).
3) **VK0214**, which is being developed for the treatment of X-linked adrenoleukodystrophy (X-ALD).
   - Results from a long-term study in an *in vivo* model of X-ALD were recently reported (discussed below).

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.

In October 2017, Viking hosted a key opinion leader (KOL) event that featured presentations on VK5211 as well as the hip fracture market, the lack of effective osteoporosis treatments, and regulatory outcomes for therapies targeting sarcopenia and hip fracture. An overview of the event can be found here.

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

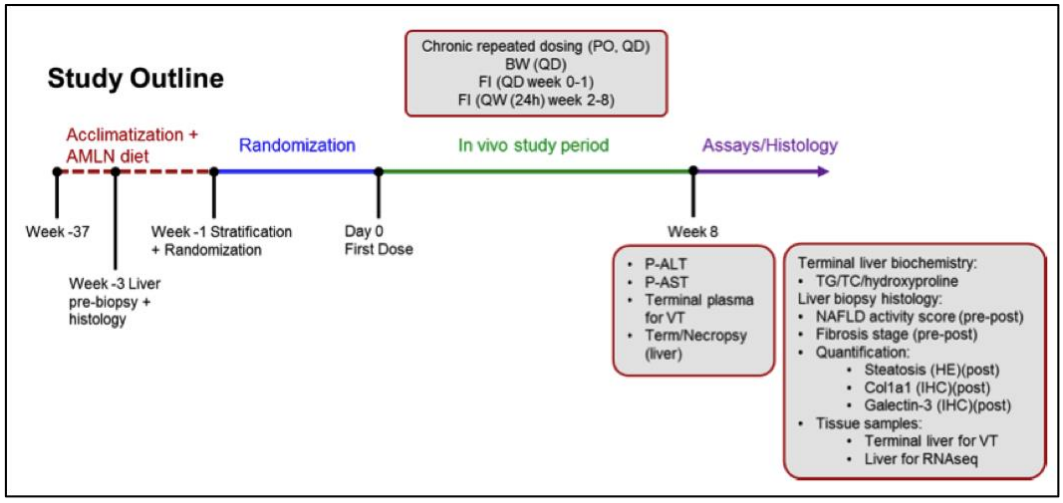
VK2809 (formerly known as MB07811) is a novel, orally available, selective thyroid hormone receptor (TR) agonist that is in development for lipid disorders such as hypercholesterolemia and fatty liver disease. It was originally developed by Metabasis Therapeutics, Inc., which was acquired by Ligand in 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).

Full Data Set for VK2809 in Diet-Induced NASH Model Presented at AASLD

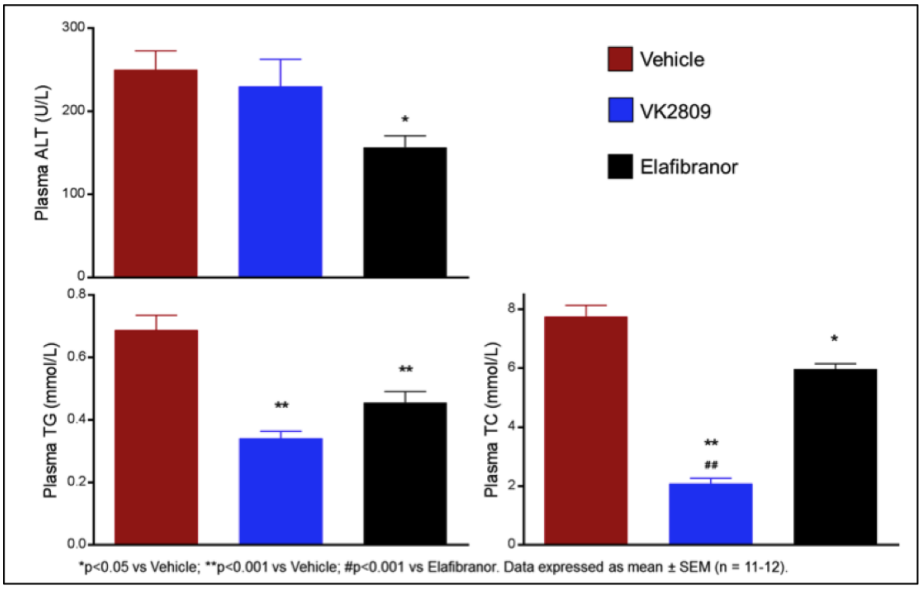
On October 24, 2017, Viking announced the presentation of results from a study of VK2809 in an *in vivo* model of non-alcoholic steatohepatitis (NASH). Statistically significant improvements in a number of key measures were seen during the study and gene expression analysis showed statistically significant changes in expression for genes associated with the development and progression of NASH.

Male wild-type C57BL/6J mice were maintained on a high fat diet for 37 weeks prior to initiation of the study. Liver biopsies were performed and the mice were stratified according to steatosis score and fibrosis stage. Treatment with once daily vehicle control, 30 mg/kg elafibranor (active control), or 10 mg/kg VK2809 continued for eight weeks while continuing the high fat diet. At the end of the study, plasma and liver lipids, liver steatosis, collagen, fibrosis, and hydroxyproline content as well as NAFLD activity score (NAS) were all evaluated. RNAseq analysis was performed on liver samples. An outline of the study is provided below.



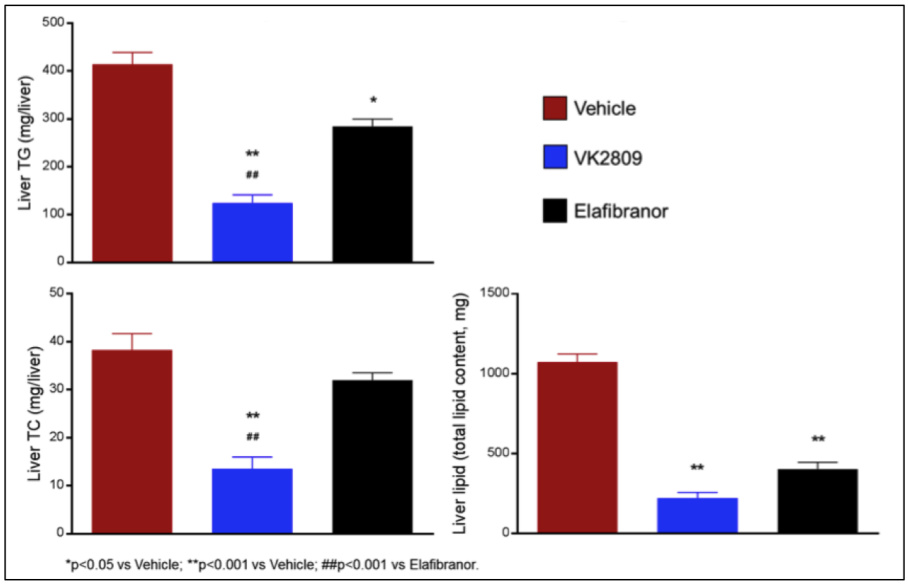
Source: Lian *et al*., 2017

Similar to what was seen in previous studies of VK2809, there was a significant decrease in plasma triglycerides (-50%) and plasma cholesterol (-73%) for mice treated with VK2809 compared to vehicle control. There was also a statistically significant decrease in plasma cholesterol for mice treated with VK2809 compared to elafibranor, which is currently in Phase III development for NASH. There was no significant difference in plasma ALT levels for VK2809-treated mice.



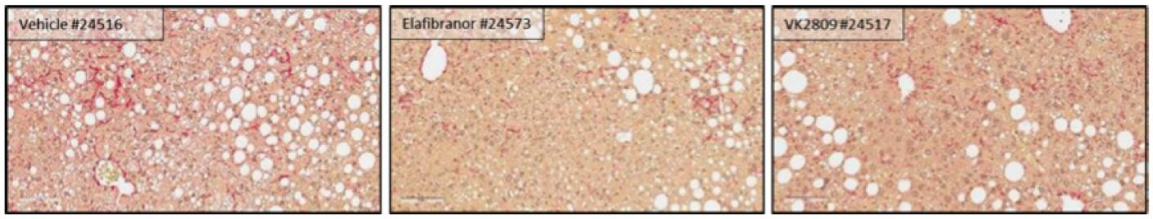
Source: Lian *et al.*, 2017

In addition, levels of liver triglycerides (-70%), liver total cholesterol (-65%) and liver total lipid content (-80%) were all significantly lower in mice treated with VK2809 compared to vehicle control mice. The difference in liver triglycerides and liver cholesterol was also significant between VK2809- and elafibranor-treated mice.



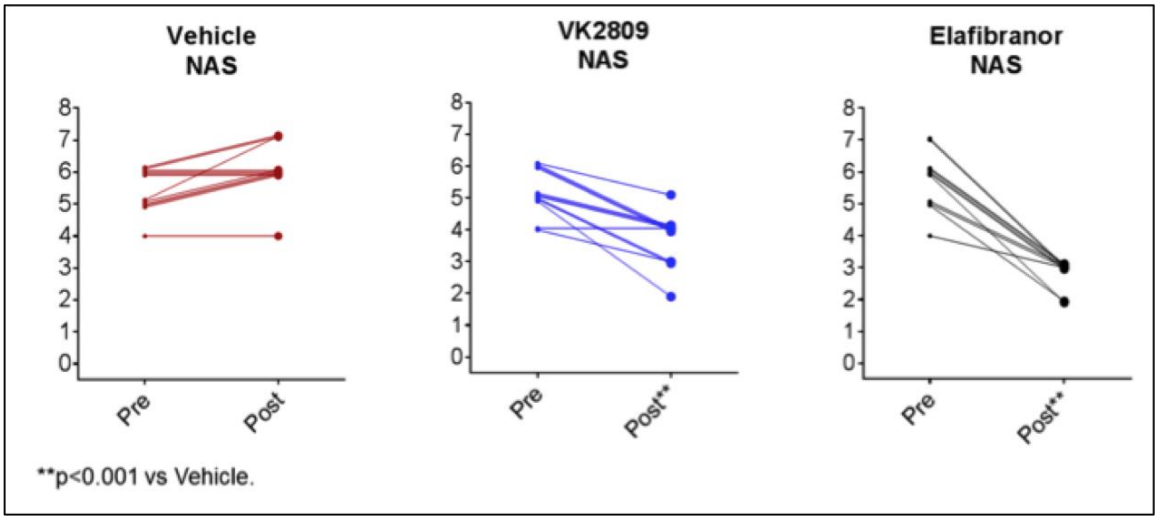
Source: Lian *et al.*, 2017

There was 50% less liver fibrosis in VK2809-treated mice compared to vehicle control-treated mice, as assessed by quantitative histological assessment of picrosirius red stain. The following figures show the level of fibrosis (red areas) in liver sections after eight weeks of treatment.



Source: Lian *et al.*, 2017

Lastly, the changes seen in lipid content and fibrosis translated to improvements in the NAFLD Activity Score (NAS) for mice treated with both VK2809 and elafibranor.



Source: Lian *et al.*, 2017

VK0214

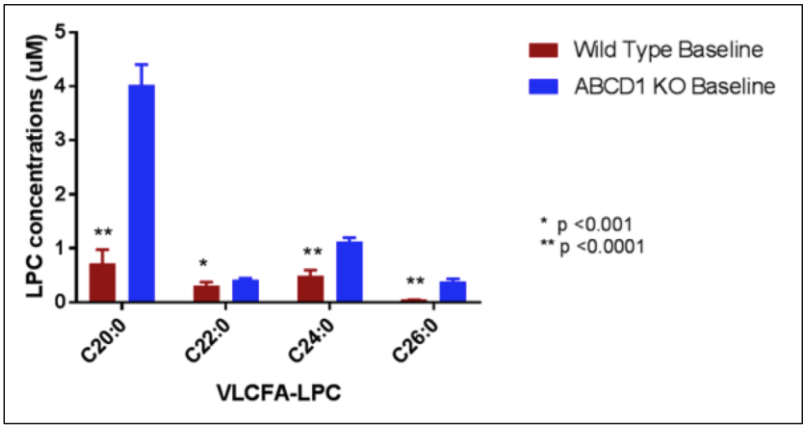
VK0214 is a thyroid receptor beta (TRβ) agonist being developed for the treatment of X-linked adrenoleukodystrophy (X-ALD), an orphan neurodegenerative disease that affects approximately 8,000 individuals in the U.S. and 12,000 in Europe. In contrast to VK2809, which is activated by the liver specific 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 Shows Promise in Treating X-ALD

On Oct. 23, 2017, Viking announced results from a 25-week proof-of-concept study of VK0214 in an *in vivo* model of X-linked adrenoleukodystrophy (X-ALD) were presented at the 87th Annual Meeting of the American Thyroid Association. Treatment with VK0214 led to statistically significant reductions in plasma levels of very long chain fatty acids (VLCFAs) compared to vehicle controls. There was also a reduction seen in brain and spinal cord VLCFAs, an exciting observation as those tissues are especially prone to degeneration in X-ALD.

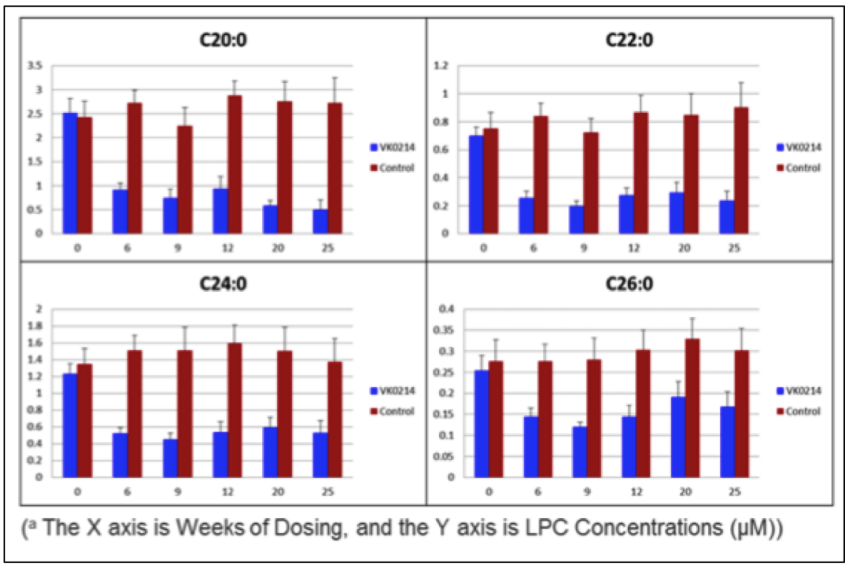
The study used the *Abcd1* knock-out (KO) mouse model, which while not displaying the inflammatory characteristics of the more severe forms of X-ALD, does recapitulate a phenotype similar to those with adrenomyeloneuropathy (AMN), the less severe form of X-ALD. As shown in the following figure, *Abcd1*$^{-/-}$ mice have elevated levels of VLCFAs compared to wildtype mice.



Source: Masamune *et al.*, 2017

Patients with X-ALD have a mutation(s) in the *ABCD1* gene that renders its product, the adrenoleukodystrophy protein (ALDP), non-functional. ALDP is responsible for transporting VLCFAs into peroxisomes for degradation, thus without proper ALDP function the VLCFAs accumulate to toxic levels. The theory behind using VK0214 to treat X-ALD is that it induces the expression of ALDR (encoded by the *ABCD2* gene), which is also a VLCFA transporter, thus compensating for the loss of ALDP.

The following charts show the level of VLCFAs over the course of the 25-week study. Treatment with VK0214 results in a dramatic decrease in VLCFA levels only six weeks after initiating treatment, and this decline is held relatively steady through the entire 25-week treatment period.



Source: Masamune *et al*., 2017

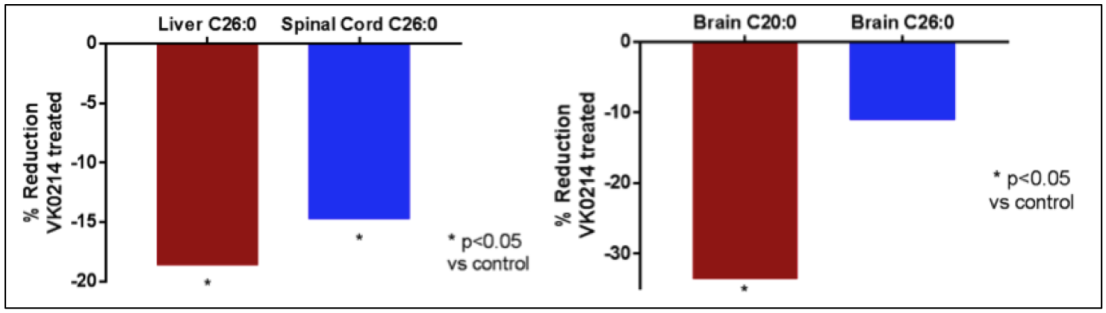
The following table shows that the greatest reductions in VLCFA levels were for shorter chain lengths, as C20:0, C22:0, and C24:0 VLCFAs showed progressive declines from week 12 to week 25, while levels of C26:0 were similar between week 12 and week 25.

VLCA-LPC Subtype	12 Weeks	25 Weeks
C26:0-LPC	-48%	-45%
C24:0-LPC	-51%	-61%
C22:0-LPC	-55%	-74%
C20:0-LPC	-57%	-82%

[a] All values statistically significant ($p<0.0001$) over control values

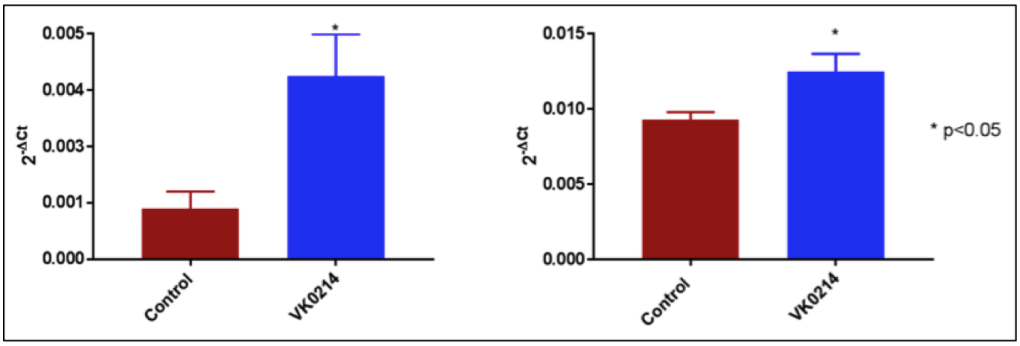
Source: Masamune *et al*., 2017

Perhaps most importantly, tissue levels of VLCFAs were shown to be lower in mice treated with VK0214 compared to mice treated with vehicle control. The following figure shows there was a statistically significant decrease in the level of C26:0 in both the liver and spinal cord. In addition, in the brain there was a statistically significant decrease in C20:0 and an 11% decrease in C26:0 that trended toward significance (P=0.07).



Source: Masamune *et al*., 2017

Lastly, as would be expected, and supporting the theory behind the use of VK0214 for treating X-ALD, there was a significant increase in the expression of the *Abcd2* gene in both the liver (+262%) and the cortex (+35%).



Source: Masamune *et al.*, 2017

Financial Update

On November 8, 2017, Viking announced financial results for the third quarter of 2017. As expected, the company did not report any revenues. Net loss for the quarter was $6.1 million, or $0.22 per share. The loss included $3.5 million in R&D expenses, compared with $2.1 million in the third 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 third quarter of 2017 totaled $1.2 million, which was in line with $1.2 million for the third quarter of 2016.

Viking exited the third quarter of 2017 with approximately $9.8 million in cash, cash equivalents, and marketable securities. During the quarter, Viking entered into stock purchase agreements for up to $16.25 million with Lincoln Park Capital, LLC, in which Viking can control the timing and amount of any future sales of common stock to Lincoln Park. We estimate the company currently has sufficient capital to fund operations into the second quarter of 2018.

As of October 31, 2017, Viking had approximately 28.5 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 44.7 million.

Conclusion

We are looking forward to the release of the topline data for the Phase 2 trial of VK5211 in hip fracture, which we expect before the end of 2017. This, along with the data from the Phase 2 trial of VK2809 in hypercholesterolemia and fatty liver disease in the first half of 2018, represent very important inflection points for the company. Positive results from these trials is sure to cause a significant revaluation of the stock price, and given the promising preclinical and Phase 1 studies for each drug we believe each trial has a good chance of succeeding.

In addition, a potentially important external catalyst for the company could be the expected 4Q17 release of Phase 2 data from Madrigal Pharmaceuticals' (MDGL) MGL-3196, which shares the same mechanism as VK2809 and is being developed for NASH. Positive results could suggest a read-through on the potential effect of VK2809 in this setting. As discussed below, MDGL shares trade at a significant premium to VKTX's.

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, however these numbers may be adjusted based on the results of the Phase 2 clinical trial.

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.

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, which is developing a TRβ agonist, MGL-3196, 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 1/8th the valuation of Madrigal. Small-cap biotech investors should take a serious look at Viking ahead of the upcoming data readouts.

PROJECTED FINANCIALS

Viking Therapeutics, Inc.
Income Statement

Viking Therapeutics, Inc.	2016 A	Q1 A	Q2 A	Q3 A	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	$3.5	$3.8	$14.5	$16.0	$18.0
General & Administrative	$4.8	$1.4	$1.3	$1.2	$1.3	$5.2	$5.7	$6.0
Other Expenses	$0	$0	$0	$0	$0	$0	$0	$0
Operating Income	($13.8)	($5.0)	($5.0)	($4.7)	($5.1)	($19.7)	($21.7)	($24.0)
Operating Margin	-	-	-	-	-	-	-	-
Non-Operating Expenses (Net)	($0.9)	($0.3)	($0.2)	($1.4)	($0.1)	($1.9)	($0.5)	($0.6)
Pre-Tax Income	($14.7)	($5.22)	($5.17)	($6.1)	($5.2)	($21.7)	($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)**	**($6.1)**	**($5.2)**	**($21.7)**	**($22.2)**	**($24.6)**
Net Margin	-	-	-	-	-	-	-	-
Reported EPS	**($0.90)**	**($0.23)**	**($0.21)**	**($0.22)**	**($0.19)**	**($0.85)**	**($0.72)**	**($0.70)**
YOY Growth	-	-	-	-	-	-	-	-
Basic Shares Outstanding	16.278	22.353	24.119	27.470	28.000	25.485	31.000	35.000

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

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