Zacks Small-Cap Research

August 16, 2016
David Bautz, PhD
312-265-9471
dbautz@zacks.com

scr.zacks.com
10 S. Riverside Plaza, Chicago, IL 60606

Viking Therapeutics, Inc.

(VKTX-NASDAQ)

VKTX: Data From Phase 2 Studies of VK2809 for Hypercholersterolemia and VK5211 for Hip Fracture to be Reported in 2Q17…

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 (08/16/16)	$1.27
Valuation	**$8.00**

OUTLOOK

On August 10, 2016, Viking Therapeutics, Inc. announced financial results for the second quarter of 2016 and provided a business update. The company exited the second quarter with approximately $17.2 million in cash and equivalents. We believe this level will be sufficient to fund operations into the third quarter of 2017.

All clinical sites in the U.S. for the Phase 2 clinical trial of VK5211 for hip fracture are open, and the company has begun opening sites and enrolling patients in Europe. A Phase 2 clinical trial of VK2809 in patients with hypercholesterolemia and fatty liver disease will get underway this month. Topline data for both Phase 2 studies should be reported in the second quarter of 2017. With two major clinical data sets expected within 12 months, Viking represents a potentially attractive risk/reward story for investors seeking underpriced opportunities with stock-moving events on the horizon.

SUMMARY DATA

52-Week High	**$7.73**
52-Week Low	**$1.18**
One-Year Return (%)	**-81.98**
Beta	**0.46**
Average Daily Volume (sh)	**167,253**
Shares Outstanding (mil)	**19**
Market Capitalization ($mil)	**$25**
Short Interest Ratio (days)	**N/A**
Institutional Ownership (%)	**0**
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 E	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	-$0.28 E	-$0.24 E	-$1.06 E
2017					-$0.89 E
2018					-$0.93 E

© Copyright 2016, Zacks Investment Research. All Rights Reserved.

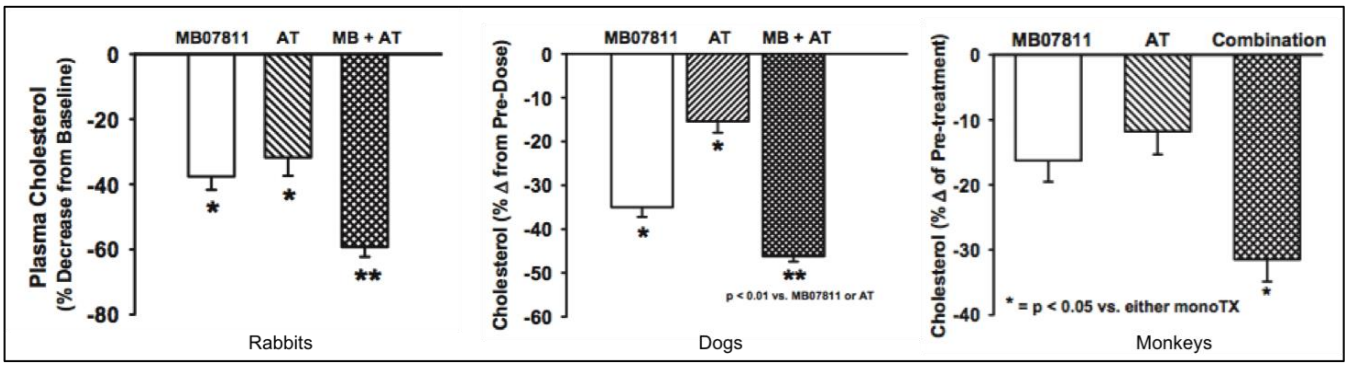
WHAT'S NEW

Business Update

VK2809 Update

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. In the second quarter update, Viking reiterated plans to initiate a Phase 2 trial of VK2809 in patients with hypercholesterolemia and fatty liver disease. We expect the study to commence this month.

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). The cholesterol lowering properties of VK2809 were exhibited in multiple studies in rabbits, dogs, and monkeys (Ito *et al*., 2009). The following figures show that VK2809 was at least as effective in lowering plasma cholesterol as atorvastatin (Lipitor®) in all three species, and an additive effect exists with the combination of VK2809 and atorvastatin.



Adapted from: Ito *et al*., 2009

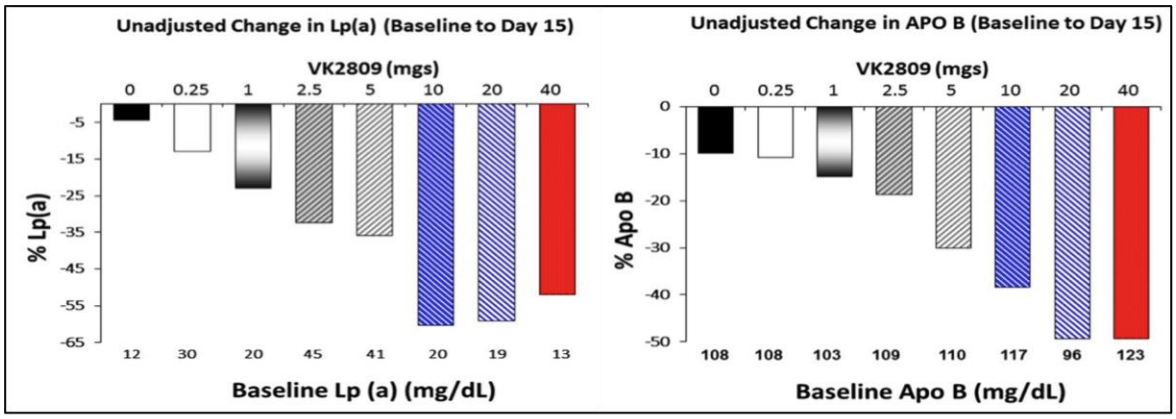
On April 8, 2016, Viking presented positive Phase 1b clinical data for VK2809 in patients with hypercholesterolemia in a poster session at the 65th Annual Scientific Session and Expo of the American College of Cardiology. The Phase 1b trial was a 14-day, placebo controlled trial in patients with mild 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.

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

In addition to the results shown above, 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 observed in certain cohorts as shown in the following figure.



Source: Lian *et al.*, 2016

Lastly, 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.

The preceding data is the basis for Viking to conduct a Phase 2 trial to evaluate VK2809 as a treatment for both hypercholesterolemia and fatty liver disease. The company is planning to target 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 the study initiating in the third quarter of 2016 with topline results reported in the second quarter of 2017.

VK0214

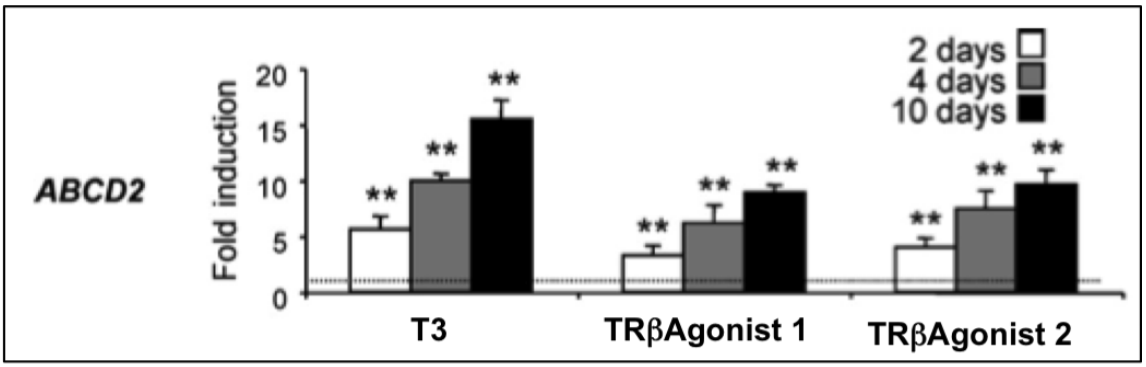
VK0214 is also a TRβ agonist, however it has a different pharmacokinetic and pharmacodynamic profile than VK2809 (shorter half-life but higher Cmax). In addition, the drug is activated by carboxyesterases, which are ubiquitously expressed in the body, thus potentially making the drug more suitable for a more diffuse disease than hypercholesterolemia and fatty liver disease. In the second quarter update, Viking highlighted promising recent data from an *in vivo* model of X-linked adrenoleukodystrophy (X-ALD) and updated plans to present these results near-term.

Viking is currently focused on the development of VK0214 for the treatment of X-ALD, which is an X-linked genetic disorder caused by a mutation in the *ABCD1* gene that encodes ALDP, a peroxisomal membrane protein (Mosser *et al*., 1993). The defect leads to an accumulation of very long chain fatty acids (VLCFA) in almost all tissues, however the adrenal glands, testes, brain, spinal cord, and peripheral nerves are most affected clinically (Moser *et al*., 2001). It is thought that the accumulation of VLCFA leads to a disruption in the cellular membrane and damage to the myelin sheath of neural cells. The damage leads to decreased motor coordination and function, visual and hearing disturbances, loss of cognitive function, and even death. In the U.S., there are approximately 8,000 patients suffering from ALD while in the E.U. there are approximately 12,000 patients.

Allogenic hematopoietic stem cell transplantation (HSCT) is the only currently available effective treatment option for ALD, but is only effective when done early in the course of the disease. Oral administration of Lorenzo's oil (LO; a 4:1 mixture of glyceryl trioleate and glyceryl trierucate) causes a rapid decrease in circulating levels of VLCFA in the bloodstream (Rizzo *et al*., 1989). This rapid decrease in circulating VLCFA levels led to the hope that LO could alter the clinical course of the disease. Unfortunately, multiple clinical trials did not show any effect of LO supplementation in patients who were already symptomatic when treatment was initiated, particularly those with CALD (van Geel *et al*., 1999; Aubourg *et al*., 1993). While a few treatment options do exist for X-ALD patients, there is still a pressing need for a more robust therapy that can offer treatment to a larger segment of the X-ALD population without unnecessary risks to the patient.

ABCD1 is one of three genes encoding peroxisomal ABC-transporters, which includes *ABCD2* and *ABCD3*. ALDR is the protein encoded by *ABCD2*, and it shares 66% homology with ALDP, the protein encoded by ABCD1 (Holzinger *et al*., 1997). When ALDR was overexpressed in cultured fibroblasts from ALD patients, impaired peroxisomal beta-oxidation was restored (Netik *et al*., 1999), suggesting that ALDR activity could compensate for the loss of ALDP activity. In addition, when murine ALDR was ubiquitously overexpressed from a transgene in *Abcd1*-deficient mice, it normalized VLCFA levels in target tissues and rescued late-onset motor coordination defects (Pujol *et al*., 2004). The reason that ALDR does not compensate for loss of ALDP function in X-ALD patients is because of complimentary expression of *ABCD1* and *ABCD2* (i.e., in tissues where *ABCD1* is expressed, *ABCD2* is not and vice versa) (Troffer-Charlier *et al*., 1998). Thus, while ALDR would appear to be able to replace the function of ALDP, it would be necessary to induce its expression in the tissues where it is not usually expressed.

Fibrates (Albet *et al*., 1997), thyroid hormones T3 and T4 (Fourcade *et al*., 2003), and 4-phenylbutyrate (Gondcaille *et al*., 2005) all stimulate the expression of *ABCD2* in the liver, thus proving that pharmacological induction of *ABCD2* expression is feasible. Furthermore, since T3 is able to induce expression of ABCD2 (through binding of TRβ), it stands to reason that a TRβ-specific agonist would also cause the same effect. In fact, TRβ-specific agonists have been shown to increase expression of *ABCD2* in a manner similar to T3 (Genin *et al*., 2009). The following figure shows the results of treating human HepG2 liver cells with either T3 or TRβ-specific agonists, with *ABCD2* expression being significantly increased compared to control treated cells (dashed line) after 2, 4, and 10 days of treatment. Thus, we believe there is ample evidence to support the development of a TRβ agonist such as VK0214 as a treatment for X-ALD.



Source: Genin *et al*., 2009

On July 26, 2016, Viking announced positive results from a study of VK0214 in an *in vivo* model of X-ALD. The model is based on the ABCD1 knockout mouse, which is intended to mirror the loss of ABCD1 transporter activity in patients suffering from X-ALD. The results of the study showed that VK0214 rapidly reduced VLCFA in plasma by more than 25% compared to placebo (P<0.01). Reductions in VLCFA were noted after only two weeks of treatment and these reductions generally increased over the course of the study. There were reductions in multiple types of VLCFAs including significantly lower levels of C26, C24, C22, and C20. The full results from the study will be presented at the upcoming 86th Annual Meeting of the American Thyroid Association, September 21-25, 2016, which we will discuss in a future report.

VK5211

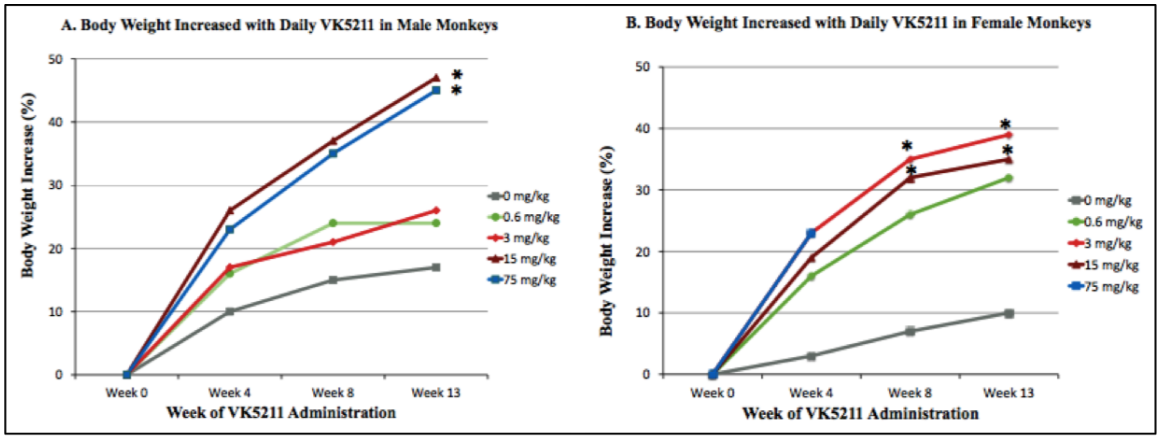
Viking's most advanced drug candidate is VK5211, a third-generation non-steroidal Selective Androgen Receptor Modulator (SARM) that is being developed for maintenance or improvement of lean body mass (LBM), bone mineral density (BMD), and function in patients recovering from non-elective hip fracture 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). In the second quarter update, Viking highlighted progress with enrollment in an ongoing Phase 2 trial of patients who are recovering from hip fracture surgery. This study is expected to be completed in the first half of 2017.

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 (then known as LGD-4033) 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.

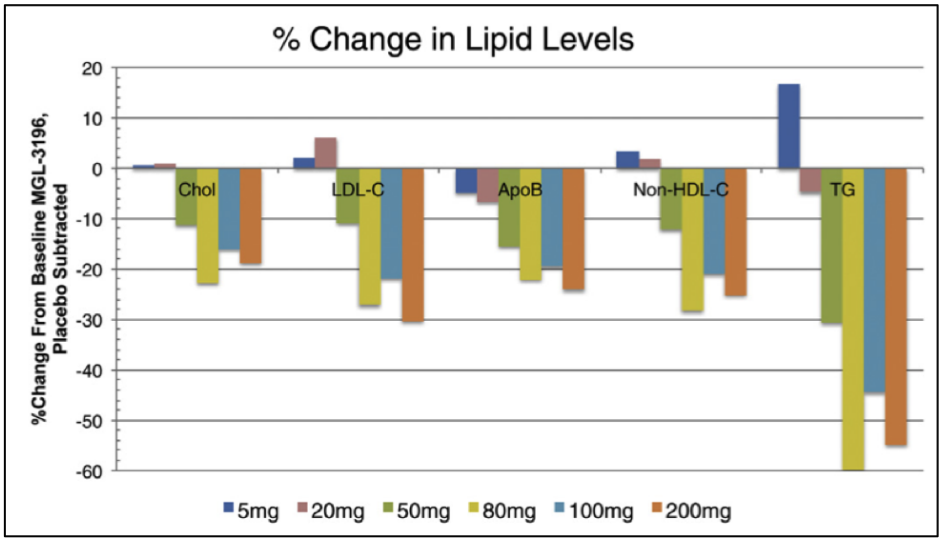


Source: Bleavins *et al*., 2015

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. All 16 clinical sites in the U.S. are now open and enrolling patients, and the company has begun opening up to 17 clinical sites in Europe. We anticipate topline results will be reported in the second quarter of 2017.

Valuation of Madrigal Pharmaceuticals Shows Viking is Significantly Undervalued

On July 22, 2016, Madrigal Pharmaceuticals (MDGL) announced the completion of the merger with Synta Pharmaceuticals. The newly combined company will be focused on the development of MGL-3196, a TRβ agonist similar to VK2809, for the treatment of NASH and hypercholesterolemia. A 2013 publication reported results from a Phase 1b study of MGL-3196 in patients with mildly elevated LDL cholesterol (>110 mg/dl) (Taub *et al*., 2013). The multi-dose study took place over two weeks and was a randomized, double blind, placebo controlled trial to examine the safety and tolerability of various doses of MGL-3196 along with the effect on lipid levels. As shown in the following figure, MGL-1396 does appear to be effective at decreasing LDL, ApoB, and triglycerides.



Source: Taub *et al*., 2013

When taking a closer look at some of the lipid values, it appears that MGL-1396 may not be as effective as VK2809. For triglycerides, of the six cohorts tested with MGL-3196, only one of them was statistically significantly different from the placebo cohort. In comparison, VK2809 was statistically significantly different from placebo in multiple cohorts for triglycerides. In addition, treatment with MGL-3196 resulted in decreases in Lp(a) of between +2.7% to -20%, compared to decreases in Lp(a) from treatment with VK2809 of -15% to -60%. Lastly, treatment with MGL-3196 resulted in decreases in ApoB of -4.9% to -24%, compared to decreases in ApoB from treatment with VK2809

of -10% to -50%. As mentioned earlier, reductions in both Lp(a) and ApoB are associated with increased cardiovascular health, thus the data so far would indicate that VK2809 is superior to MGL-1396 in this regard.

Even with a potentially superior TRβ agonist compound, the valuation for Viking in comparison to Madrigal does not reflect this fact. Following the recent public offering, Viking has approximately 19.3 million common shares outstanding. The stock has recently traded near $1.30, putting Viking's valuation at approximately $25 million. Following the merger with Synta and a 1:35 reverse split, Madrigal has approximately 11.3 million shares outstanding. Madrigal's stock recently traded at $8.00, which would give Madrigal a valuation of approximately $90 million, almost four times as much as Viking! MGL-3196 is set to enter a Phase 2 clinical trial, thus it is at the same development stage as VK2809. In addition, Viking has VK5211, which is currently in a Phase 2 clinical trial for the prevention of loss of lean body mass following hip fracture in elderly patients. Thus, based upon the valuation assigned to Madrigal we believe that Viking is significantly undervalued.

Financial Update

On August 10, 2016, Viking announced financial results for the second quarter of 2016. The company reported a net loss of $3.7 million, or $0.22 per share, which was comprised of $2.4 million in R&D expenses and $1.2 million in G&A expenses. The company exited the second quarter with approximately $17.2 million in cash and cash equivalents, compared to $14.1 million on Dec. 31, 2015. This increase in cash was the result of a public offering of 7.5 million shares of common stock and warrants at a price of $1.25 per share. The warrants have an exercise price of $1.50, are exercisable until Apr. 13, 2021, and will trade on the Nasdaq under the ticker symbol 'VKTXW'. The underwriters of the public offering fully exercised their over-allotment option and purchased 1.125 million shares for $1.24 per share, thus the aggregate gross proceeds to the company were approximately $10.8 million before deducting underwriting discounts, commissions, and other offering expenses. In addition, in June the company filed an at-the-money (ATM) sales prospectus, allowing for the sale of up to approximately $5 million in common stock at prevailing market prices. We believe this facility could provide further cash runway beyond our current expectation of the third quarter of 2017, representing an important potential means of strengthening the company's balance sheet.

As of July 31, 2016, Viking had approximately 19.3 million shares of common stock outstanding, with Ligand owning approximately 33% 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 million shares.

Valuation

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 adrenoleukodystrophy (ALD).

The pre-clinical and clinical data for VK5211 are quite encouraging, particularly the magnitude of the effect, the durability of response, and the lack of any serious adverse events. We model for peak revenues of VK5211 of approximately $600 million in the U.S. and $1 billion in the E.U.

Viking has presented compelling data for VK2809, particularly given the low level of adverse events coupled with data that rivals many of the current treatment options for dyslipidemia. We estimate potential peak worldwide revenues for VK2809 of $2.5 billion.

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, leads to a valuation of $8/share. The fact that Viking has a market cap of only $25 million is quite perplexing, particularly given the valuation of Madrigal and its TRβ agonist of approximately $90 million following the completion of its merger with Synta Pharmaceuticals. There is no discernable reason that Viking, with a TRβ agonist with potentially superior efficacy to Madrigal's, should have a market cap approximately 1/4th that of Madrigal and we believe it is only a matter of time until Viking's valuation matches the company's potential. In the meantime, we believe Viking's current valuation presents investors with a valuable opportunity to acquire shares or add to a position in a very promising small-cap biotechnology company ahead of expected data read outs in the second quarter of 2017 for both VK5211 and VK2809.

PROJECTED FINANCIALS

Viking Therapeutics, Inc.
Income Statement

Viking Therapeutics, Inc.	2015 A	Q1 A	Q2 A	Q3 E	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	$3.4	$2.9	$10.5	$13.5	$16.0
General & Administrative	$5.0	$1.4	$1.2	$1.5	$1.4	$5.5	$5.8	$6.0
Other Expenses	$0	$0	$0	$0	$0	$0	$0	$0
Operating Income	($12.0)	($3.3)	($3.6)	($4.9)	($4.3)	($16.0)	($19.3)	($22.0)
Operating Margin	-	-	-	-	-	-	-	-
Non-Operating Expenses (Net)	($11.4)	($0.3)	($0.1)	($0.5)	($0.5)	($1.4)	($2.0)	($3.0)
Pre-Tax Income	($23.4)	($3.6)	($3.7)	($5.4)	($4.8)	($17.5)	($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)**	**($5.4)**	**($4.8)**	**($17.5)**	**($21.3)**	**($25.0)**
Net Margin	-	-	-	-	-	-	-	-
Reported EPS	**($3.68)**	**($0.40)**	**($0.22)**	**($0.28)**	**($0.24)**	**($1.06)**	**($0.89)**	**($0.93)**
YOY Growth	-	-	-	-	-	-	-	-
Basic Shares Outstanding	6.356	9.016	17.105	19.500	20.000	16.405	24.000	27.000

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

© Copyright 2016, Zacks Investment Research. All Rights Reserved.

HISTORICAL STOCK PRICE



© Copyright 2016, Zacks Investment Research. All Rights Reserved.

DISCLOSURES

The following disclosures relate to relationships between Zacks Small-Cap Research ("Zacks SCR"), a division of Zacks Investment Research ("ZIR"), and the issuers covered by the Zacks SCR Analysts in the Small-Cap Universe.

ANALYST DISCLOSURES

I, David Bautz, PhD, hereby certify that the view expressed in this research report accurately reflect my personal views about the subject securities and issuers. I also certify that no part of my compensation was, is, or will be, directly or indirectly, related to the recommendations or views expressed in this research report. I believe the information used for the creation of this report has been obtained from sources I considered to be reliable, but I can neither guarantee nor represent the completeness or accuracy of the information herewith. Such information and the opinions expressed are subject to change without notice.

INVESTMENT BANKING AND FEES FOR SERVICES

Zacks SCR does not provide investment banking services nor has it received compensation for investment banking services from the issuers of the securities covered in this report or article.
Zacks SCR has received compensation from the issuer directly or from an investor relations consulting firm engaged by the issuer for providing non-investment banking services to this issuer and expects to receive additional compensation for such non-investment banking services provided to this issuer. The non-investment banking services provided to the issuer includes the preparation of this report, investor relations services, investment software, financial database analysis, organization of non-deal road shows, and attendance fees for conferences sponsored or co-sponsored by Zacks SCR. The fees for these services vary on a per-client basis and are subject to the number and types of services contracted. Fees typically range between ten thousand and fifty thousand dollars per annum. Details of fees paid by this issuer are available upon request.

POLICY DISCLOSURES

This report provides an objective valuation of the issuer today and expected valuations of the issuer at various future dates based on applying standard investment valuation methodologies to the revenue and EPS forecasts made by the SCR Analyst of the issuer's business.
SCR Analysts are restricted from holding or trading securities in the issuers that they cover. ZIR and Zacks SCR do not make a market in any security followed by SCR nor do they act as dealers in these securities. Each Zacks SCR Analyst has full discretion over the valuation of the issuer included in this report based on his or her own due diligence. SCR Analysts are paid based on the number of companies they cover. SCR Analyst compensation is not, was not, nor will be, directly or indirectly, related to the specific valuations or views expressed in any report or article.

ADDITIONAL INFORMATION

Additional information is available upon request. Zacks SCR reports and articles are based on data obtained from sources that it believes to be reliable, but are not guaranteed to be accurate nor do they purport to be complete. Because of individual financial or investment objectives and/or financial circumstances, this report or article should not be construed as advice designed to meet the particular investment needs of any investor. Investing involves risk. Any opinions expressed by Zacks SCR Analysts are subject to change without notice. Reports or articles or tweets are not to be construed as an offer or solicitation of an offer to buy or sell the securities herein mentioned.