Zacks Small-Cap Research

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Viking Therapeutics, Inc. (VKTX-NASDAQ)

VKTX: Encouraging In Vivo *Data For VK2809 From Mouse Model of GSD 1a and Gene Expression Analysis in Mouse Model of NASH…*

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 (09/12/17)	$1.16
Valuation	**$7.00**

OUTLOOK

On September 7, 2017, Viking Therapeutics, Inc. (VKTX) announced the presentation of data from a proof of concept *in vivo* study of VK2809 in a mouse model of glycogen storage disease 1a (GSD 1a). Treatment with VK2809 for only 4 days resulted in a 69.0% reduction in mean total liver triglycerides and a 35.7% mean reduction in liver weight. The company is planning to file an IND before the end of the year such that a proof of concept study in patients with GDS 1a can be initiated.

On September 11, 2017, Viking announced positive results from a gene expression analysis conducted as part of an *in vivo* study of VK2809 in a mouse model of non-alcoholic steatohepatitis (NASH). The company is currently testing VK2809 in a Phase 2 clinical trial of patients with hypercholesterolemia and fatty liver disease, with results expected in the first half of 2018.

SUMMARY DATA

52-Week High	**$1.69**
52-Week Low	**$0.93**
One-Year Return (%)	**-11.45**
Beta	**1.07**
Average Daily Volume (sh)	**170,837**
Shares Outstanding (mil)	**28**
Market Capitalization ($mil)	**$32**
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 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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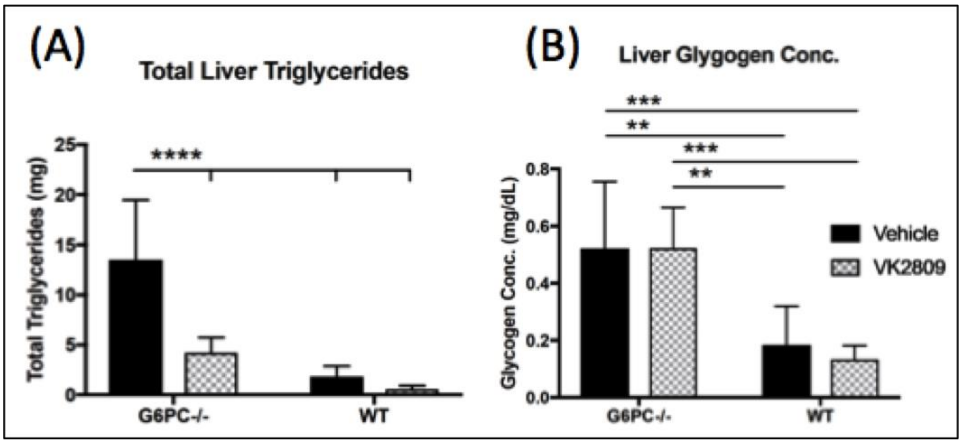
WHAT'S NEW

Business Update

Encouraging Data For VK2809 in Mouse Model of GSD 1a

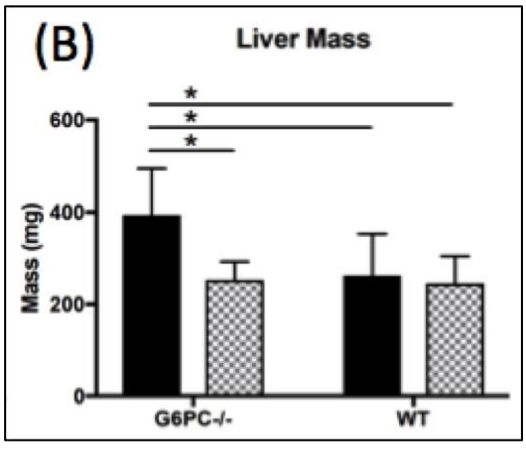
On September 7, 2017, Viking Therapeutics, Inc. (VKTX) announced encouraging results from an *in vivo* study of VK2809 in a mouse model of glycogen storage disease 1a (GSD 1a). The researchers used glucose-6-phospatase catalytic domain knockout mice (G6PC$^{-/-}$) to model the effects seen in patients who have GSD 1a, as they have a mutation that renders glucose-6-phosphatase inactive. The mice only live for approximately 10-15 days, thus the experiment was conducted on days 5-8 following birth. Since the mice are hypoglycemic, 0.1 to 0.2 mL of 10% dextrose was administered subcutaneously beginning within 3 days of birth and continuing daily throughout drug or vehicle treatment.

The results showed that after four days of treatment with VK2809 there was a statistically significant 69% decrease in mean total liver triglycerides in G6PC$^{-/-}$ mice (Panel A in the following figure; P<0.0001). This decrease brought the average liver triglycerides to within the range of the wild type control mice. However, there was no effect on liver glycogen concentration (Panel B in the following figure). The lack of an effect on liver glycogen is not of concern for treatment with VK2809, as the vast majority of the increase in liver weight due to GSD 1a is from accumulation of lipids.



Source: Koeberl *et al.*, 2017

The following figure shows that the decrease in triglycerides resulted in a statistically significant reduction in liver mass in VK2809-treated G6PC$^{-/-}$ mice (P<0.05). The average liver mass following treatment was within the range of the wild type mice.



Source: Koeberl *et al.*, 2017

Even with a very short treatment period, VK2809 was able to alleviate liver abnormalities in G6PC$^{-/-}$ mice, thus supporting the company's plan to move ahead with an investigational new drug (IND) application later in 2017 for a planned proof-of-concept study in GSD 1a patients.

Positive Data for VK2809 in Gene Expression Study

On September 11, 2017, Viking announced positive results from a gene expression analysis study that was conducted as part of an *in vivo* study of VK2809 in non-alcoholic steatohepatitis (NASH). The company had previously announced topline results from the study, in which treatment with VK2809 resulted in statistically significant reductions in a number of key measures related to the development and progression of NASH, including liver triglyceride content, liver cholesterol content, liver fibrosis, liver collagen content, and liver hydroxyproline content.

The new data concerns expression analysis of more than 20,000 genes and in particular key genes associated with NASH development and progression:

SREBF1 (also known as *SREBP-1*): a 43.7% decrease in animals treated with VK2809 (P<0.001). *SREBP-1* expression levels are known to be elevated in animal models of NASH (Morgan *et al*., 2008).

PPARD: a 64.2% increase in animals treated with VK2809 (P<0.01). *PPARD* encodes the peroxisome proliferator-activated receptor delta (PPARδ), a nuclear hormone receptor that controls the expression of a number of target genes, including those involved in lipid metabolism (Luquet *et al*., 2005).

FGF21: a 336.8% increase in animals treated with VK2809 (P<0.001). *FGF21* encodes the protein fibroblast growth factor 21, a member of the fibroblast growth factor (FGF) family. FGF21 stimulates glucose uptake in adipocytes in an additive fashion to insulin (Kharitonenkov *et al*., 2005).

COL1A1: a 36.3% decrease in animals treated with VK2809 (P<0.05). *COL1A1* encodes the major component of type I collagen, which is found in most connective tissues. Fibrosis from excess collagen deposition is a hallmark of NASH.

The company also disclosed improvements in additional NASH-related genes of interest, including *CYP7A1* (involved in rate limiting step of converting cholesterol to bile acid), *MAP3K5* (encodes ASK1, involved in MAP kinase pathway activation in response to oxidative stress), *KRT18* (*Krt18*$^{-/-}$ knockout mice develop liver steatosis, Betterman *et al*., 2016), *ACTA2* (encodes alpha-smooth muscle actin, involved in fibrogenesis), and *LGALS1* (encodes galectin-1, involved in fibrosis).

These changes in gene expression appear to correlate with the results presented previously, showing that alterations in gene expression can have positive effects on NASH-related processes, including reductions in liver fat and fibrosis.

Background on GSD 1a/VK2809

GSD 1a is a rare, orphan genetic disease caused by mutations (thus far, 84 have been identified) in glucose-6-phophatase, a key enzyme involved in the maintenance of glucose homeostasis. The enzyme catalyzes the hydrolysis of glucose-6-phosphate to glucose in the final step of gluconeogenesis and glycogenolysis. The inability of GSD 1a patients to maintain proper glucose levels and to develop hypoglycemia following a short fast is a hallmark of the disease (Chou *et al*., 2002). Patients accumulate excess glycogen in the liver and kidney, which results in progressive hepatomegaly and nephromegaly. Additional metabolic consequences include hypercholesterolemia, hypertriglyceridemia, hyperuricemia, and lactic acidemia. Accumulation of fats in the liver also contributes to hepatomegaly.

There is no cure for GSD 1a and dietary augmentation is the current standard of care for patients. For those younger than six months of age, nocturnal nasogastric infusion of glucose is utilized to avoid hypoglycemia during the night. For those older than six months, supplemental uncooked cornstarch is used as a slow release glucose source between meals. When followed strictly, dietary strategies typically allow for normal growth and puberty development, however dietary therapy fails to completely prevent the occurrence of hyperlipidemia, hyperuricemia, lactic acidemia, and accumulation of liver fat (Rake *et al*., 2002).

Background on VK2809

VK2809 is a novel, orally available, selective thyroid hormone receptor (TR) agonist. 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).

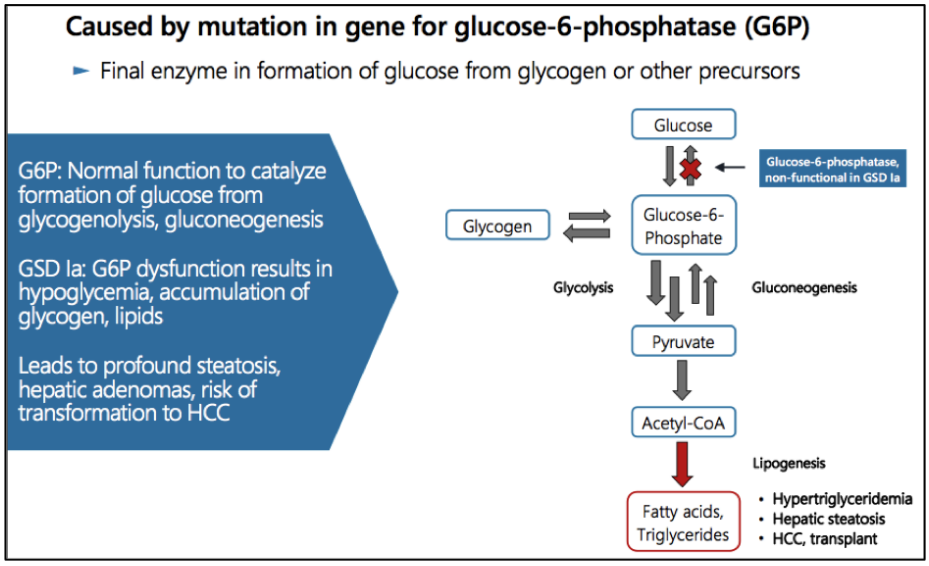
Thyroid hormones and TR agonists act through a number of mechanisms to modulate triglyceride and cholesterol metabolism, including:

- Increasing expression of LDL receptors, which leads to enhanced clearance of serum LDL (Erion *et al*., 2007)
- Decreasing apolipoprotein B (ApoB) levels, which is the major protein constituent of LDL (Ladenson *et al*., 2010)
- Impacting several aspects of reverse cholesterol transport (Pedrelli *et al*., 2010), the net effect of which is to reduce LDL
- Enhancing synthesis of apolipoprotein A-1, the predominant protein of HDL (Hargrove *et al*., 1999)
- Increasing hepatic uptake of cholesterol from HDL by increasing activity of scavenger receptor class B type-1 (Johansson *et al*., 2005)
- Increasing the activity of liver cholesterol 7α hydroxylase, which converts cholesterol into bile acids for fecal excretion (Johansson *et al*., 2005)
- Inhibiting transcription of sterol regulatory element binding protein-1 (SREBP-1), which decreases fatty acid synthesis and reduces triglycerides (Erion *et al*., 2007)

In addition to lowering plasma cholesterol levels, VK2809 was also shown to reduce hepatic steatosis in rats through increased hepatic fatty acid oxidation, with no evidence of liver fibrosis or other histological liver damage (Cable *et al*., 2009).

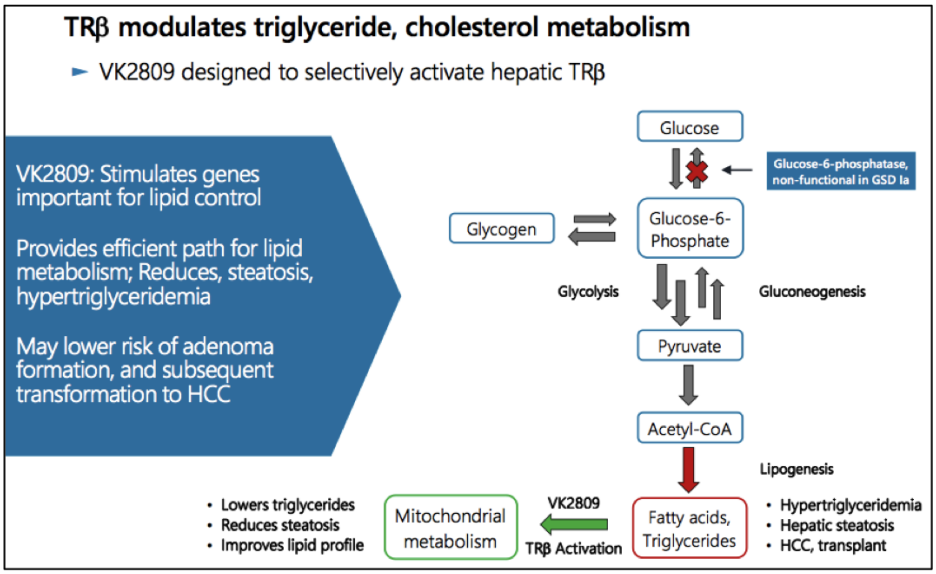
VK2809 for the Treatment of GSD 1a

The following schematic shows how impairment of glucose-6-phosphatase activity leads to accumulation of fatty acids and triglycerides in GSD 1a.



Source: Viking Therapeutics, Inc.

Based on the activity discussed above, administration of VK2809 can theoretically lead to an increase in hepatic TRβ activity, thus increasing the mitochondrial breakdown of lipids and leading to an improved metabolic profile.



Source: Viking Therapeutics, Inc.

Conclusion

The data presented above regarding VK2809 shows that Viking is in a strong position with that compound, as there are data supporting its development for either GSD 1a or NASH. It is currently unclear whether both indications would be simultaneously pursued long-term. Given the relative expenses associated with development in each indication, GSD 1a may represent an attractive option for the company to explore independent of a partner, whereas the NASH indication might be most appropriately pursued with the assistance of a larger biopharmaceutical company. It seems unlikely that a decision would be made about which indication to pursue long-term until after the Phase 2 data in fatty liver disease are known, which we anticipate in the first half of 2018.

As a reminder, Viking is expected to announce additional results from a long-term *in vivo* study of VK0214 in a model of X-linked adrenoleukodystrophy (X-ALD) in the third quarter of 2017, and results from the Phase 2 study of VK5211 in hip fracture in the fourth quarter of 2017. Viking is set up for a number of catalysts over the next six months, and with the stock trading well below our valuation of $7 per share, we believe now is the time for investors to familiarize themselves with the story ahead of those read outs.

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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