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# Viking Therapeutics, Inc. (VKTX-NASDAQ)

## *VKTX: No Cardio Toxicity Concerns for VK2809…*

Based on our probability adjusted DCF model that takes into account potential future revenues of VK5211, VK2809, and VK0214, VKTX is valued at $28/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 (01/23/19) | $8.11 |
| **Valuation** | **$28.00** |

## OUTLOOK

We've recently heard from investors that are concerned about the potential for cardiotoxicity for VK2809. We investigated this potential and have concluded that those concerns are unwarranted. These fears appear to be based on a misinterpretation of prior data, not unlike what was seen with the previous fears regarding the potential for liver toxicity for VK2809 (which the Phase 2 data quickly dispelled). In this report, we evaluate the data regarding the potential (or lack thereof) for VK2809-induced cardiotoxicity, an idea which we now believe is being floated as a means to detract from the stellar results seen in the Phase 2 clinical trial and the drug's potential in NASH.

## SUMMARY DATA

| | |
|---|---|
| **52-Week High** | **$19.65** |
| **52-Week Low** | **$3.88** |
| **One-Year Return (%)** | **71.82** |
| **Beta** | **3.01** |
| **Average Daily Volume (sh)** | **2,629,263** |
| **Shares Outstanding (mil)** | **71** |
| **Market Capitalization ($mil)** | **$580** |
| **Short Interest Ratio (days)** | **N/A** |
| **Institutional Ownership (%)** | **60** |
| **Insider Ownership (%)** | **4** |
| **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 2018 Estimate** | **-22.3** |
| **P/E using 2019 Estimate** | **-22.3** |

| | |
|---|---|
| **Risk Level** | **High** |
| **Type of Stock** | **Small-Value** |
| **Industry** | **Med-Biomed/Gene** |

## ZACKS ESTIMATES

**Revenue**
(In millions of $)

| | Q1 (Mar) | Q2 (Jun) | Q3 (Sep) | Q4 (Dec) | Year (Dec) |
|---|---|---|---|---|---|
| 2017 | 0 A | 0 A | 0 A | 0 A | 0 A |
| 2018 | 0 A | 0 A | 0 A | 0 E | 0 E |
| 2019 | | | | | 0 E |
| 2020 | | | | | 0 E |

**Earnings per Share**

| | Q1 (Mar) | Q2 (Jun) | Q3 (Sep) | Q4 (Dec) | Year (Dec) |
|---|---|---|---|---|---|
| 2017 | -$0.23 A | - $0.21 A | -$0.22 A | -$0.14 A | -$0.79 A |
| 2018 | -$0.08 A | -$0.13 A | -$0.11 A | -$0.09 E | -$0.40 E |
| 2019 | | | | | -$0.34 E |
| 2020 | | | | | -$0.35 E |

## WHAT'S NEW

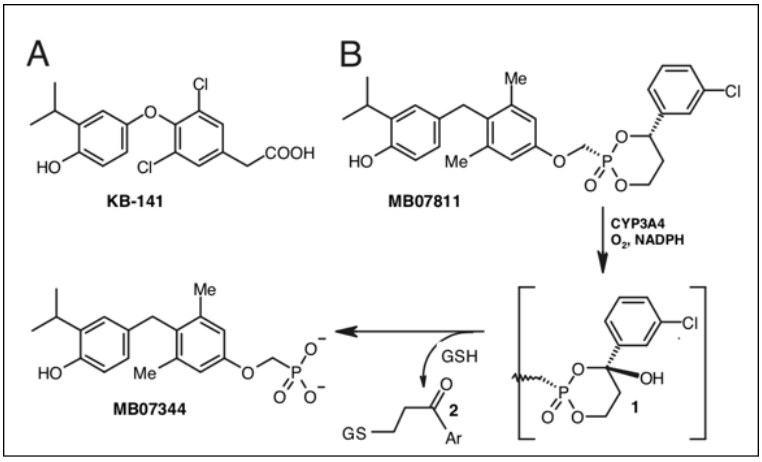
**Business Update**

*No Cardiovascular Toxicity Concerns for VK2809*

Following release of the Phase 2 data for VK2809 in patients with hypercholesterolemia and nonalcoholic fatty liver disease (NAFLD), which we believe show the potential for VK2809 to be a best-in-class treatment in NAFLD and nonalcoholic steatohepatitis (NASH), we began to hear from investors who were questioning the potential for VK2809-induced cardiotoxicity. While we are unsure of where these concerns originated from, we analyzed the available data and have concluded that any concerns surrounding cardiotoxicity from VK2809 are unwarranted. For interested investors, we provide a link to what we believe are the most pertinent manuscripts (Erion *et al*., 2007; Fujitaki *et al*., 2008).

Before getting into the data it is necessary to give a brief overview of VK2809 and its activation. VK2809 is an agonist of the thyroid hormone receptor beta (TRβ). There are two major isoforms of the thyroid hormone receptor (TR), TRα and TRβ, which have markedly different expression patterns. TRα expression is highest in the heart and brain while TRβ expression is highest in the liver (Bookout *et al*., 2006). Multiple studies have shown a clear role for TRα in regulating heart rate (Wikström *et al*., 1998) and TRβ regulating serum cholesterol levels (Weiss *et al*., 2002; O'Shea *et al*., 2002). Thus, compounds that can selectively alter TRβ function with little to no effect on TRα could have a beneficial effect on lipid metabolism while avoiding potentially deleterious cardiac side effects.

As shown in the following figure, VK2809 (referred to in the figure as MB07811) is a prodrug that is converted to its active form by the enzyme cytochrome P450 3A4 (CYP3A4). CYP3A4 is expressed predominantly in the liver, but also in the small intestine.




Source: Erion *et al*., 2007

Upon oral administration, VK2809 exhibits significant first-pass hepatic extraction (~55%) and is not subject to enterohepatic recirculation. What this means is that a significant portion of the drug is quickly metabolized in the liver to the active form and is unable to reach systemic circulation. The highly negatively charged phosphonic acid group on activated VK2809 (MB07344) essentially 'traps' the molecule inside the cell (highly charged compounds typically cannot passively diffuse across the cellular membrane) and also results in a decreased affinity for cellular transporters that transport carboxylates, again decreasing the chance for the active compound to reach the circulation in appreciable amounts.

We believe that detractors of VK2809 have taken the available pharmacokinetic data and misinterpreted it in an effort to exaggerate the potential for VK2809 to both get into the heart and to activate TRα, which in theory could lead to unwanted side effects. The following table shows the level of radioactive VK2809 found in various tissues following an oral dose in rats. We believe it is important to highlight a couple of things from the table: 1) the prodrug was radiolabeled, thus it is not possible to differentiate whether the radioactive compound measured is the prodrug or the activated compound; 2) there is a large amount of drug in the stomach, small intestine, and large intestine,

which is not surprising given the drug was orally administered; 3) the amount of drug found in the heart (0.36 ± 0.36 nmol/g) is very small and close to the limit of quantitation; and 4) after 24 hours there is essentially no drug remaining in the heart and the highest concentration of drug is seen in the liver. The takeaways from this table are that it is conceivable that some activated VK2809 is entering the circulation (due to its high concentration in the intestine and the presence of intestinal CYP3A4), however it is clearly not being concentrated in the heart and any VK2809 that in fact penetrates cardiac tissue is not accumulating.

We find it most probable that any VK2809 found in the heart is the prodrug (as it can more easily passively diffuse across the cell membrane), and not the activated form. If this is the case, it is not a concern as there is no expression of CYP3A4 in cardiac tissue to convert the prodrug to its active form. In addition, there is no meaningful expression of TRβ in the heart and the prodrug itself has very low affinity for TRα (prodrug VK2809 $K_i$ = 12.5 ± 0.6 μM; activated VK2809 TRβ $K_i$ = 2.17 ± 0.41 nM). In other words, the radioactivity observed in heart is most likely due to the presence of the prodrug, which doesn't bind to TR. While our comments are speculative, we believe they're more consistent with the chemical and biological characteristics of VK2809 than the alternative proposal, which is based on the idea that activated VK2809 is present (it's far less likely to penetrate cell membranes compared with the prodrug) and that it's accumulating (it isn't).

| Tissue | 3 Hour (nmol/g) | 24 Hour (nmol/g) |
|---|---|---|
| stomach | 76.96 ± 48.75 | 0.22 ± 0.09 |
| stom. C&W | 24.52 ± 10.88 | 0.06 ± 0.02 |
| lymph(m) | 16.61 ± 17.57 | 0.05 ± 0.01 |
| small int. | 28.56 ± 22.73 | 0.40 ± 0.22 |
| sma. C&W | 39.69 ± 30.16 | 0.20 ± 0.03 |
| large int. | 1.23 ± 1.12 | 0.64 ± 0.21 |
| larg. C&W | 4.68 ± 2.85 | 2.50 ± 0.89 |
| liver | 6.67 ± 3.54 | 3.67 ± 0.64 |
| adrenal | 0.77 ± 0.46 | 0.09 ± 0.02 |
| kidneys | 0.48 ± 0.37 | 0.14 ± 0.01 |
| thymus | 0.09 ± 0.05 | 0.01 ± 0.00 |
| heart | 0.36 ± 0.36 | 0.05 ± 0.01 |

*Adapted from*: Erion *et al.*, 2007

This data is further supported by radiolabel data from experiments in monkeys. Just as in rats, following oral administration the majority of VK2809 is found in liver and any drug found in cardiac tissue is no longer present after 12 hours. This again shows that there is no cardiac accumulation of VK2809.

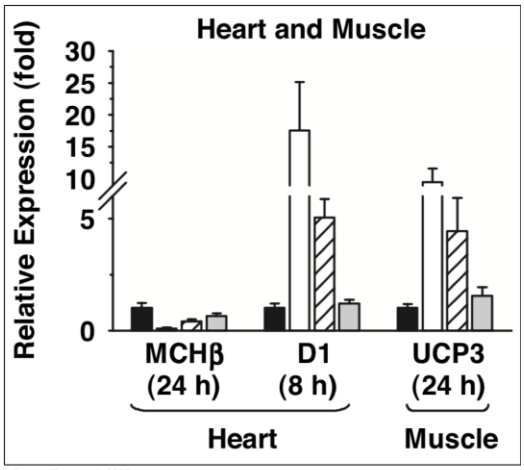
Concentrations of radiolabel in specific tissues and fluids after oral administration of 5 mg/kg [$^{14}$C]MB07811 to male cynomolgus monkeys. Other than blood and plasma, tissues were collected from monkeys 1001, 1002, and 1003 at 4, 12, and 24 h postdose, respectively.

| Tissue | Time Point | Monkey 1 (4 h) | Monkey 2 (12 h) | Monkey 3 (24 h) |
|---|---|---|---|---|
| | *h* | | | |
| Blood | 4 | 360 | 272 | 425 |
| Blood | 12 | N.S. | 92 | 136 |
| Blood | 24 | N.S. | N.S. | 67 |
| Plasma | 4 | 640 | 479 | 773 |
| Plasma | 12 | N.S. | 165 | 241 |
| Plasma | 24 | N.S. | N.S. | 120 |
| Adipose | 4, 12, 24 | 136 | 68 | 31 |
| Adrenal glands | 4, 12, 24 | 248 | 90 | 75 |
| Adipose | 4, 12, 24 | 117 | 77 | 34 |
| Heart | 4, 12, 24 | 205 | <1 | <1 |
| Kidneys | 4, 12, 24 | 1707 | 304 | 198 |
| Liver | 4, 12, 24 | 4594 | 2768 | 2095 |
| Pituitary | 4, 12, 24 | 202 | 55 | 27 |
| Skeletal muscle (pectoral) | 4, 12, 24 | 80 | <1 | <1 |
| Skeletal muscle (thigh) | 4, 12, 24 | 83 | <1 | <1 |
| Spleen | 4, 12, 24 | 156 | <1 | <1 |
| Thymus | 4, 12, 24 | N.C. | 258 | 129 |

N.C., not calculated; N.S., no sample.

*Source*: Fujitaki *et al.*, 2008

Additional data supporting the cardiac safety of VK2809 comes from gene expression data showing that in contrast to $T_3$ (white bars) or KB-141 (non-liver targeted TR agonist, striped bars), VK2809 (gray bars) did not cause an increase in expression of genes known to be sensitive to TR activation. In fact, cardiac gene expression changes due to VK2809 are similar to vehicle (black bars). Thus, even if VK2809 was getting into cardiac tissue it does not appear to have any effect on TR activation.



Source: Erion et al., 2007

Lastly, no TRα specific changes or cardiac toxicity have been seen to date in animals or humans. In rats, dosing with VK2809 caused no significant change in heart rate, body weight, left ventricular pressure, or heart weight. Perhaps most importantly, in the Phase 2 study of VK2809 there was no sign of a change in body weight, heart rate, or blood pressure, which would be indicative of potential TRα activation. Thus, we don't find any basis to support the argument that VK2809 has the potential to cause cardiotoxicity.

**Conclusion**

We are glad to have been able to take a close look at the available data regarding VK2809 cardiac safety and are confident in our conclusion that, based on the available data, there is no safety concern. We are unsure of the origin of this argument against VK2809 (although we heard it from multiple sources), but it is not supported by the published data that are often cited as evidence. We surmise that it is an attempt to detract from the very encouraging data for the drug showing a robust ability to decrease liver fat in patients with NAFLD, which we believe is an excellent read-through for a NASH population. We believe VK2809 could become a best-in-class drug, and we are looking forward to additional updates from Viking regarding the Phase 2b clinical trial in NASH patients, which we continue to anticipate initiating in the second half of 2019. In addition to that update, we anticipate the company presenting additional data from the 5 mg dosing cohort from the Phase 2 trial of VK2809 at EASL 2019 in April. Our thesis for Viking remains unchanged, and it continues to be one of our top picks among small-cap biotech stocks. Our valuation remains at $28.

## PROJECTED FINANCIALS

**Viking Therapeutics, Inc.**
Income Statement

| Viking Therapeutics, Inc. | 2017 A | Q1 A | Q2 A | Q3 A | Q4 E | 2018 E | 2019 E | 2020 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 | $13.7 | $3.0 | $5.2 | $5.7 | $4.5 | $18.5 | $18.0 | $20.0 |
| General & Administrative | $5.3 | $1.8 | $1.7 | $1.7 | $1.6 | $6.8 | $6.5 | $7.0 |
| Other Expenses | $0 | $0 | $0 | $0 | $0 | $0 | $0 | $0 |
| Operating Income | ($19.1) | ($4.8) | ($6.93) | ($7.4) | ($6.1) | ($25.2) | ($24.5) | ($27.0) |
| *Operating Margin* | - | - | - | - | - | - | - | - |
| Non-Operating Expenses (Net) | ($1.5) | $1.3 | $0.3 | $0.8 | ($0.2) | $2.1 | ($1.0) | ($1.0) |
| Pre-Tax Income | ($20.6) | ($3.6) | ($6.7) | ($6.6) | ($6.3) | ($23.1) | ($25.5) | ($28.0) |
| Income Taxes Paid | $0 | $0 | $0 | $0 | $0 | $0 | $0 | $0 |
| *Tax Rate* | *0%* | *0%* | *0%* | *0%* | *0%* | *0%* | *0%* | *0%* |
| **Net Income** | **($20.6)** | **($3.6)** | **($6.7)** | **($6.6)** | **($6.3)** | **($23.1)** | **($25.5)** | **($28.0)** |
| *Net Margin* | - | - | - | - | - | - | - | - |
| **Reported EPS** | **($0.79)** | **($0.08)** | **($0.13)** | **($0.11)** | **($0.09)** | **($0.40)** | **($0.34)** | **($0.35)** |
| *YOY Growth* | - | - | - | - | - | - | - | - |
| Basic Shares Outstanding | 25.978 | 44.649 | 52.767 | 61.232 | 71.400 | 57.512 | 75.000 | 80.000 |

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

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