LAIDLAW & COMPANY
Est. 1842
London
New York
San Francisco
Stamford
Ft. Lauderdale

EQUITY RESEARCH
Company Report

June 8, 2015

Viking Therapeutics (VKTX - $ 7.89)

Healthcare/Biotechnology

Multiple Assets in Place, Two Advancing Programs Target Large Unmet Needs With Only Modest Competition

Ticker:	**VKTX**
Rating:	**Buy**
Price Target:	**$ 20.00**

We are initiating coverage of Viking Therapeutics with a Buy rating and 12-month price target of $20. VKTX is ready to start two POC clinical studies for VK5211 and a thyroid-β agonist in 2H15 and 1Q16, respectively, as treatment in post hip fracture surgery rehabilitation and X-ALD.

- **VK5211, a first-in-class therapy in post hip fracture surgery rehabilitation with Phase II study to start in 3Q15, supported by promising Phase I results.** VK5211 is a non-steroidal selective androgen receptor modulator (SARM) that has showed encouraging clinical results in a Phase I healthy volunteers study for the elevation of lean body mass and leg press force. A Phase II POC dose optimizing trial will start in 3Q15 to evaluate VK5211 as an acute therapy in rehabilitation post hip fracture surgery with results expected possibly in 2Q16.

- **Thyroid β agonist in X-linked adrenoleukodystrophy (X-ALD) Phase I study to start 1Q16.** VKTX is scheduled to start a POC Phase I study in X-ALD (in AMN subtype) likely in 1Q16 after it has chosen an appropriate thyroid β agonist candidate (VK0214/VK2809). Top-line results are expected in 2H16. Thyroid β agonist stimulates ABCD2 production and could provide therapeutic benefit compensating the loss of ABCD1 – etiological cause of X-ALD.

- **VK2809 also has potential to treat dyslipidemias and NASH – all have large commercial potential.** VK2809 has demonstrated encouraging results in fasting LDL-C and triglyceride reductions from an earlier Phase Ib study. By reducing liver fat, it also showed potential as a NASH treatment. Although VKTX will not advance VK2809 clinically in either indication near-term without additional financial support, we believe this is a hidden gem for VKTX's value.

- **Additional assets are "free call options" at the current valuation.** Other assets not to be developed near-term include VK0612 (Phase IIb in T2D), EPOR agonist and DGAT-1 inhibitor (both preclinical).

- **Substantial upside at the current valuation.** With two differentiated assets in place for clinical development near term, we believe VKTX shares remain undervalued at current levels. Clinical data reporting over the next 4 – 5 quarters could significantly change VKTX share value, in our opinion, should the outcome be positive. Our $20 price target is based on peer comparable, probability adjusted DCF and sum-of-the-parts analyses.

Trading Data:

Last Price (06/05/2015)	$ 7.89
52–Week High (5/5/2015)	$ 10.23
52–Week Low (6/1/2015)	$ 7.51
Market Cap. (MM)	$ 76
Shares Out. (MM)	10

Earnings Estimates: (per share)

(Dec)	1Q	2Q	3Q	4Q	FY	P/E
FY-15E	0.28	-0.16	-0.26	-0.34	-0.64	N.A.
FY-14A	-0.07	NA	NA	-2.01	-5.23	N.A.
FY-13A	0.00	NA	NA	NA	-0.07	N.A.
FY-12A	NA	NA	NA	NA	-0.07	N.A.

Source: Laidlaw & Company estimates

Yale Jen, Ph.D.
Managing Director/Senior
Biotechnology Analyst
(212) 953-4978
yjen@laidlawltd.com

Investment Thesis

Our $20 price target is supported by peer comparable, sum-of-the-parts and DCF analyses.

- **We are initiating coverage of Viking Therapeutics (VKTX) with a Buy rating and a 12-month price target of $20.** Viking Therapeutics is a mid-clinical stage biotechnology company focusing on the development of therapeutics in endocrinological and orphan diseases near term. The company in-licensed majority of its clinical assets from Ligand Pharmaceuticals. Leading assets that will enter clinical developments include VK5211 in a post hip fracture surgery rehabilitation Phase II study, and a thyroid β agonist in X-linked adrenoleukodystrophy (with focus on adrenomyeloneuropathy or AMN patients) Phase I trial. VKYX's thyroid β agonist, VK2809, also has the potential as treatment in dyslipidemias and nonalcoholic steatohepatitis or NASH. Also available for future development if additional funding becomes available are clinical study ready VK0612 as a potential oral type 2 diabetes (T2D) treatment; and two pre-clinical programs, EPOR agonist and DGAT-1 inhibitor.

VK5211, a SARM, has exhibited encouraging clinical results in healthy volunteers for the elevation of lean body mass and leg press force..

A Phase II POC, dose optimizing trial that evaluates VK5211 as an acute therapy for rehabilitation after recent non-elective hip fracture surgery is to start in 3Q15, with the top-line results are expected in 2Q16.

- **Supported by promising Phase I results, VK5211, a non-steroidal selective androgen receptor modulator (SARM), is starting a post hip fracture surgery rehabilitation Phase II study in 3Q15 with top-line results expected in 2Q16.** VKTX's lead program, VK5211, is a non-steroidal selective androgen receptor modulator (SARM) that has exhibited encouraging clinical results in healthy volunteers for the elevation of lean body mass and leg press force. VK5211 exhibited high muscle specificity (> 500x selectivity) vs. testosterone of its activity. VKTX is scheduled to commence a Phase II POC, dose optimizing trial in 3Q15 to evaluate VK5211 (≤5 mg) as an acute therapy for rehabilitation in patients who have undergone recent non-elective hip fracture surgery. The primary endpoint is change in lean body mass; while secondary endpoints include changes in bone mineral density (BMD), functional status, re-hospitalization rates and quality of life (QOL). The top-line results are expected in 2Q16. VKTX could advance VK5211 development in 2H16 or 2017 pending positive data from the Phase II study. We are optimistic on the outlook of VK5211 in hip fracture rehabilitation since 1) anabolic effect exerted by testosterone has demonstrated improvements post hip fracture surgery; and 2) VK5211 could stimulate the growth of both muscle and bone, while other drugs in development mainly stimulate muscle growth. Together, we view VK5211 is the company's more advanced and most risk mitigated clinical program in active development. We estimate annual peak sale potential for VK5211 could exceed $1 billion.

- **By increasing ABCD2 expression, thyroid β agonist prospects (VK0214/VK2809) could potentially become a disease-modifying treatment for X-linked adrenoleukodystrophy (V-ALD), such as adrenomyeloneuropathy (AMN).** VKTX is developing thyroid β

Yale Jen, Ph.D.
yjen@laidlawltd.com

agonist, either VK0214 or VK2809, as a potential treatment for X-linked adrenoleukodystrophy (X-ALD) with adrenomyeloneuropathy (AMN) subtype as the first indication. By augmenting ABCD2 expression in order to compensate the diminished ABCD1 expression caused by genetic mutations, thyroid β agonist could potentially become a disease-modifying treatment for X-ALD. VKTX is scheduled to start a Phase I study likely in 1Q16 to evaluate one of the two thyroid β agonists as a potential treatment in X-ALD patients (mainly the AMN type). The company currently is evaluating VK0214 and VK2809 to determine which drug candidate will advance into clinical study. The Phase I study is an open label multicenter, dose-finding trial with changes in VLCFA as the primary endpoint, while other measures of the study include safety and tolerability, functional status, and quality of life. We estimate the study to be completed possibly in mid-2016 with top-line results shortly thereafter. In addition, management believes that thyroid-β agonists could also afford therapeutic benefits to cerebral adrenoleukodystrophy (CALD) patients, and expects development could start in the future after advancements in AMN are already in place. We estimate annual peak sale potential for thyroid β agonist in X-ALD could exceed $750MM.

The company currently is evaluating VK0214 and VK2809 to determine which drug candidate will advance into clinical study. A Phase I study is likely to start in 1Q16 and complete possibly in mid-2016 with top-line results shortly thereafter. Changes in VLCFA are primary endpoint.

- **Thyroid β agonist (VK2809) also has potential in treating indications, such as dyslipidemias and NASH – all represent larger commercial opportunities.** In addition to treating X-ALD, thyroid β agonist (VK2809) also has demonstrated dose-related reductions in fasting LDL-C (ranging from 15% – 41% with statistical significance) and fasting triglyceride levels at day 14 from a Phase Ib study. The study also showed statistically significant reductions of lipoprotein a (Lp(a)) and apolipoprotein (Apo(B)) in certain cohorts. The drug is safe and well-tolerated and without serious adverse events identified. In addition, VK2809 has potential as a treatment for NASH since pre-clinical studies exhibited potential benefits in reducing in fat content in the liver. VK2809 treatment also did not show any impact on transaminase activities. VKTX will not actively advance VK2809 into clinical studies in either indication near-term unless additional financial support becomes available. Given the potential VK2809 has demonstrated in the two indications and the larger commercial opportunity, we believe VK2809 in these two indications could be a hidden gem for VKTX's value.

VK2809 has demonstrated dose-related reductions in fasting LDL-C and fasting triglyceride levels at day 14 from a Phase Ib study. VK2809 has potential as a treatment for NASH as pre-clinical studies exhibited potential beneficial in reducing in fat content in liver.

- **Additional assets are "free call options" at the current valuation.** VKTX also has several clinical assets that have larger market potential. VK0612 is a Phase IIb-ready drug candidate for type 2 diabetes. Another is a pre-clinical EPO receptor (EPOR) agonist program as a potential treatment of anemia. A third is a diacylglycerol acyltransferase-1 (DGAT-1) inhibitor program (pre-clinical as a potential treatment of lipid disorders such as obesity and dyslipidemia). Although VKTX also will not advance these assets clinically near term unless additional financial supports become available, we believe they are also hidden gem, similar to that of VK2809 in dyslipidemias and NASH, for VKTX's shareholders.

VKTX has several clinical assets that have larger market potential: VK0612 (Phase IIb-ready drug candidate in T2D), EPOR agonist and DGAT-1 inhibitor.

- **Valuation is favorable.** We believe VKTX shares are undervalued, based on diversified multiple shots on goal coupled with a positive

Yale Jen, Ph.D.
yjen@laidlawltd.com

outlook for the two assets to be advanced clinically near term. It is noted that major catalysts of reporting clinical top-line results of the two lead products could be available within the next four to five quarters. We believe VKTX shares could materially appreciate should the outcome of these events be positive. Accordingly, our $20 price target is supported by probability-adjusted sum-of-the-parts, peer comparable and probability-adjusted DCF analyses. We are recommending VKTX shares to long-term oriented investors with high risk tolerance.

Yale Jen, Ph.D.
yjen@laidlawltd.com

Company Description

Viking Therapeutics is a mid-clinical stage biotech company focused on development of therapeutics for endocrinological and orphan diseases near term. The company currently has two lead products in development: 1) **VK5211**, a non-steroidal selective androgen receptor modulator (SARM) that VKTX is starting post hip fracture surgery rehabilitation Phase II study in 3Q15 with top-line results expected in 2Q16; and 2) a **thyroid β agonist**, either VK0214 or VK2809, to be used as a potential treatment for X-linked adrenoleukodystrophy (X-ALD) with adrenomyeloneuropathy (AMN) subtype. A Phase I study is scheduled to start in 1Q16 with top-line results expected in mid- to 2H16. In addition, VKTX also has multiple clinical assets to be further developed once the company obtains more financial support. These assets include: 1) VK2809 in dyslipidemias, for which it has already exhibited positive results from a Phase Ib trial, and NASH; 2) VK0612, a Phase IIb-ready drug candidate for type 2 diabetes) 3) an EPO receptor (EPOR) agonist program, pre-clinical as a potential treatment of anemia; and 4) a diacylglycerol acyltransferase-1 (DGAT-1) inhibitor program, which is pre-clinical as a potential treatment of lipid disorders such as obesity and dyslipidemia.

Yale Jen, Ph.D.
yjen@laidlawltd.com

Anticipated milestones in 2015 and beyond and pipeline

Product	Indication	Event	Timing	Importance
VK5211	Hip fracture	Initiate Phase IIa study	**3Q15**	***
		Report pre-clinical primate data	**2H15**	***
		Report Phase IIa study results	**2Q16**	****
VK0214/VK2809	X-Linked Adrenoleukodystrophy (X-ALD)	Complete pre-clinical POC studies	**Mid-15**	***
		Initiate Phase I POC study	**1Q16**	***
		Potentially report Phase I study top-line results	**Mid-16**	****
VK2809	Cholesterolemia	Potentially start Phase II study with additional financial supports	**4Q15**	***
	NASH	Potentially start Phase II study with additional financial supports	**4Q15**	***

***** / ***** Major catalyst event that could impact share price very significantly while *** event is more informative*

Source: Laidlaw & Company and company presentation.

Yale Jen, Ph.D.
yjen@laidlawltd.com

Viking Therapeutics Pipeline

Development Pipeline						
Product	**Indication**	**Preclin**	**I**	**II**	**III**	**Comments**
VK5211	Post hip fracture surgery rehabilitation					Phase II study to start in 2H15 with results available in 2016
VK2809/VK0214	X-ALD					Phase I study to start in 1Q16 with results available in 2016
VK2809	Dyslipidemia					Commence Phase IIa trial if resources available
	Nonalcoholic steatohepatitis (NASH)					Commence Phase IIa trial if resources available
VK0612	Type 2 diabetes (T2D)					Commence Phase IIb or drug-drug interaction studies if resources available
EPOR	Anemia					Conduct further preclinical studies and file an IND in 2016
DGAT-1	Obesity, dyslipidemia					Conduct further preclinical studies and file an IND in 2016

Source: Laidlaw & Company and company presentation

Yale Jen, Ph.D.
yjen@laidlawltd.com

Supported by Encouraging Phase I Study Results, VK5211 Exhibits Promise in Rehabilitating Hip Fracture Post Surgery

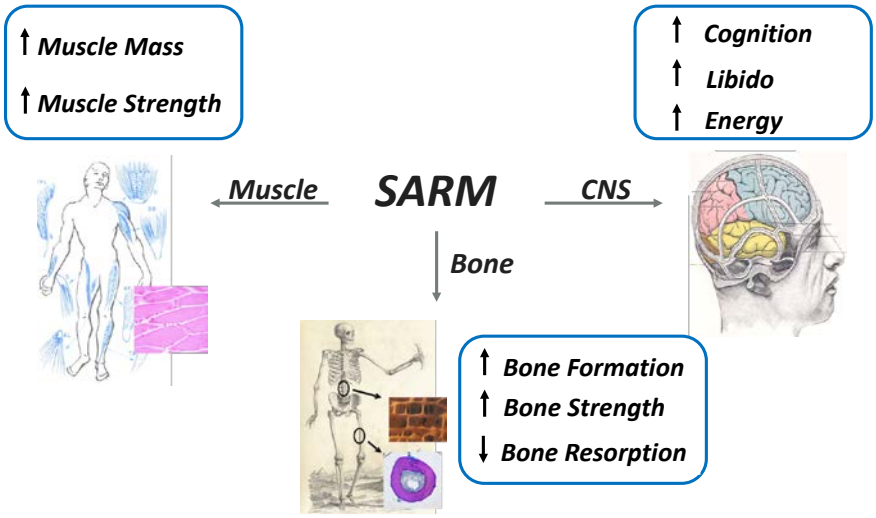
VK5211 is a non-steroidal selective androgen receptor modulator (SARM)

VKTX's lead clinical asset is VK5211, a non-steroidal selective androgen receptor modulator (SARM) that has exhibited promising clinical results in healthy volunteers. We view VK5211 is the company's most risk mitigated clinical program in active development.

SARM is a class of hormones that bind to cellular androgen receptors and exhibit a highly tissue specific activation or inhibition. The major impetus for SARM developments are for the potential anabolic effects on skeletal muscle and bone.

What is selective androgen receptor modulator (SARM)? SARM is a class of hormones that bind to cellular androgen receptors and exhibit a highly tissue specific activation or inhibition. SARM could be steroidal or non-steroidal. The developments were pioneered by Ligand Pharmaceuticals and the University of Tennessee. Tissue and functional specificity is the major distinction between SARM and a traditional androgen receptor ligand, such as testosterone. The androgen receptor is involved in a complex signal transduction pathway that ultimately results in greater expression of specific genes. The major impetus for SARM developments are for the potential anabolic effects on skeletal muscle and bone.

Figure 1:SARMs exhibit therapeutic impact in multiple systems



Source: Company presentation

SARM could provide clinical impacts on multiple fronts. SARM could generate varying activating or inhibitory activities in different tissues. In men, SARMs may be able to: 1) stimulate testosterone's action in bone, muscle and

Yale Jen, Ph.D.
yjen@laidlawltd.com

brain; 2) block testosterone's action in the prostate and skin; and 3) either cross or not cross into the central nervous system to affect libido (Figure 1).

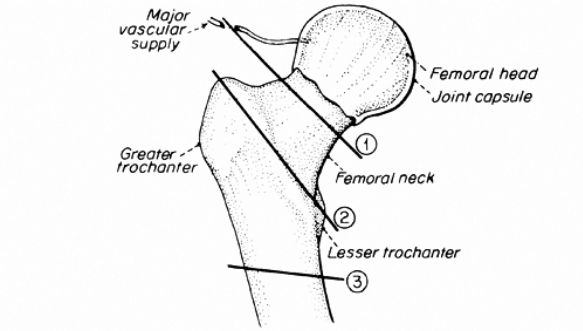
Even after surgical repair, patients suffering from prior hip fracture frequently are still associated with profound and lasting repercussions, mainly in loss of independence and with high risk of mortality

What happens after hip fracture? Hip fracture is a frequent injury among older adults. Even after surgical repair, patients suffering from prior hip fracture frequently are still associated with profound and lasting repercussions, mainly in loss of independence and with high risk of mortality (overall 25% within one year). The increased mortality risks are proportional to patient's age. For example, more than 1/3 of hip fracture patients over the age of 80 and only 6% of those older than 90 are alive and walking one year after injury. The U.S. statistics indicate that approximately 70% of those who survive the acute post-operative stage are discharged to a nursing home; while only 25% of hip fracture victims ever regain their former level of independence. It is estimated that ~90% of hip fractures are due to a fall. Four fall-related factors could help to predict the propensity of hip fracture for an individual: 1) slow gait; 2) difficulty in doing a heel-to-toe walk; 3) small calf circumference; and 4) poor vision and poor depth perception. It is estimated that approximately 80% of hip fractures occur in women and their vulnerability were mainly due to post-menopause bone density declines (~30% between the ages of 50 and 80). In men, age-related changes in the basic metabolic activity in bone contribute to bone loss. There are three types of most common hip fractures (Figure 2):

There are three types of most common hip fractures: Intracapsular, intertrochanteric and subtrochanteric

- Intracapsular fractures – break occurs below the ball or in the neck of the femur
- Intertrochanteric fractures – break occurs between the greater trochanter and lesser trochanter
- Subtrochanteric fractures – break occurs below the lesser trochanter or further down the femur

Figure 2: Types of hip fractures



TYPE OF FRACTURE	ANATOMY	IMPLICATIONS
① Subcapital (Intra-capsular)	Disrupts blood supply to femoral head	Higher incidence of non-union and necrosis of femoral head
② Intertrochanteric ③ Subtrochanteric	Blood supply to femoral head intact	Lower incidence of non-union and necrosis of femoral head

Source: Kane, Robert L.; Ouslander, Joseph G.; Abrass, Itamar B. (2004) Essentials of Clinical Geriatrics, 5th Edition

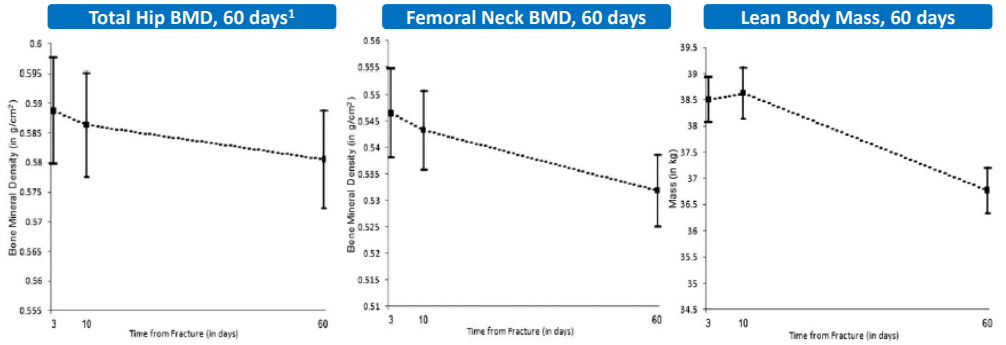
Yale Jen, Ph.D.
yjen@laidlawltd.com

Negative changes in body composition that occur during the year after hip fractures are associated with increased disability, recurrent fracture, and mortality.

From a pathophysiological prospective, negative changes in body composition that occur during the year after hip fracture are associated with increased disability, recurrent fracture, and mortality. After hip fracture surgery; many patients have experienced elevated rates of metabolic breakdown of muscle tissue (typically the type 2 muscle fibers) and loss of bone mineral density (BMD).

A more recent study has placed the adverse body composition changes much closer to and within 10 days to 2 months post-fracture.[1] In this study, researchers demonstrated that during this period, significant decreases were noted in the total body mass (−1.95 kg, P < 0.001), lean mass (−1.73 kg, P < 0.001), total hip BMD (−0.00812 g/cm2, P = 0.04) and femoral neck BMD (−0.015 g/cm2, P = 0.03) (Figure 3).

Figure 3: Deleterious body composition changes occur during first 60 days after hip fracture



Source: Company presentation

BMD loss after hip surgery could exceed 12 times the background rate for patients with osteoporosis. All participants in this study have received corrective hip fracture surgery. In summary, a catabolic state develops after operation and malnutrition, which is either present or develops after surgery, contributes to a poor outcome.

Given the more rapid onset of deleterious body composition changes occurred shortly post hip fracture, we believe there is a significant unmet need in developing therapeutics to slow or even reverse the body composition changes to improved rehabilitation outcomes after hip fracture surgery.

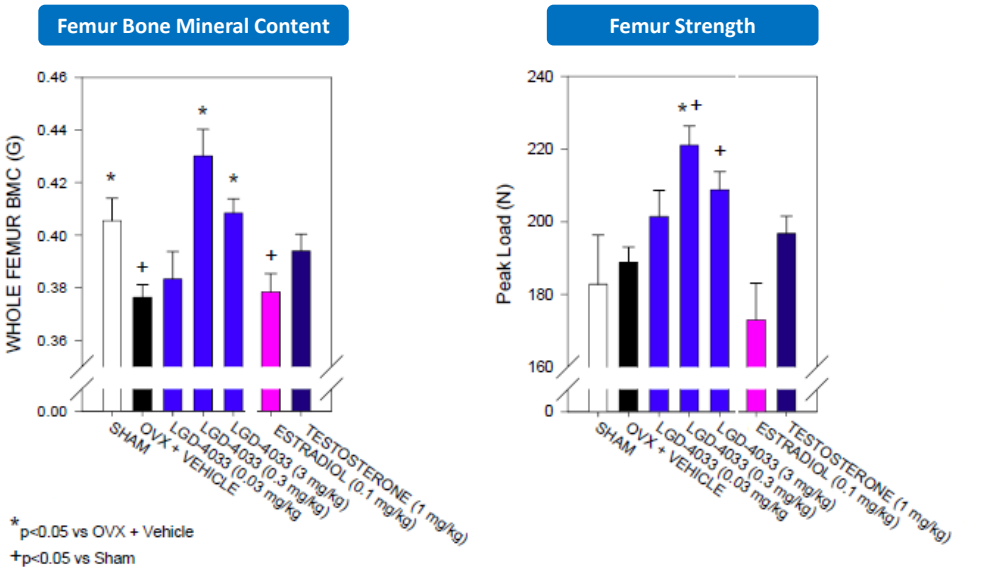
VK5211 exhibited positive effects in rodent bone models Licensed from Ligand Pharmaceuticals, VKTX's lead product, VK5211 (LGD-4033, a selective androgen receptor modulator or SARM) has demonstrated encouraging muscle and bone buildup in pre-clinical and clinical studies.

From a pre-clinical study in an ovariectomized (ovx) rat system that models skeletal responses, such as high bone turnover, and subsequent bone loss similar to that in a post-menopausal condition, LGD-4033 demonstrated anabolic

[1] *D'Adamo, C., et. al., (2014) Age and Ageing 43: 275–280*

effects by increasing the bone formation rate on femur (Figure 4, left), and restoring the strength of femur bone (Figure 4, right). In addition, the same study also demonstrated that LGD-4033 in an intact female rat study could also increase skeletal muscle mass at multiple sites, such as plantaris, gastrocnemius and biceps brachii. Further, the increased muscle mass is correlated with the increased muscle fiber cross sectional diameter.

Figure 4: Preclinical study demonstrated VK5211 could increase bone formation rate and strength of femur bone



Source: Company presentation

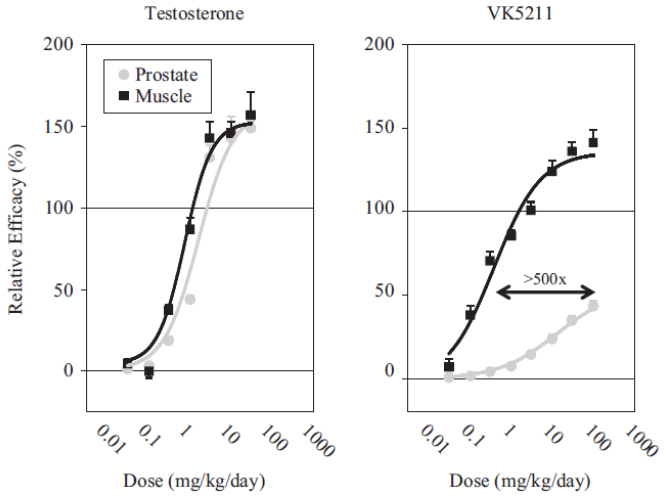
Although it is well known that testosterone could also have a beneficial impact on muscle and bone increases, this medication is not widely used due to potential safety concerns, such as prostate growth in men, and hair growth and masculinization in women due to the drug's lack of selectivity. By examining the tissue selectivity of androgen in a castrated rat model, a system that exhibits the rapid muscle atrophy and effect of growth upon androgen-based treatment, VK5211 demonstrated > 500x selectivity for maintaining muscle weight.

Testosterone, on the other hand, exhibited similar effects non-discriminately on both muscle and prostate tissue (Figure 5). As such, VK5211 could potentially provide a better therapeutic profile relative to testosterone due to its high tissue-selectivity for muscle[2]. Further, LGD-4033 binds to androgen receptor with high affinity (Ki of ~1nM). As such, VK5211 could be more potent than testosterone.

[2] *Vajda, E.G., et. al., Gerontological Society of America 62nd Annual Scientific Meeting, Nov. 18-22, 2009*

Yale Jen, Ph.D.
yjen@laidlawltd.com

Figure 5: VK5211 activiies exhibit high tissue specificity



Source: Company presentation

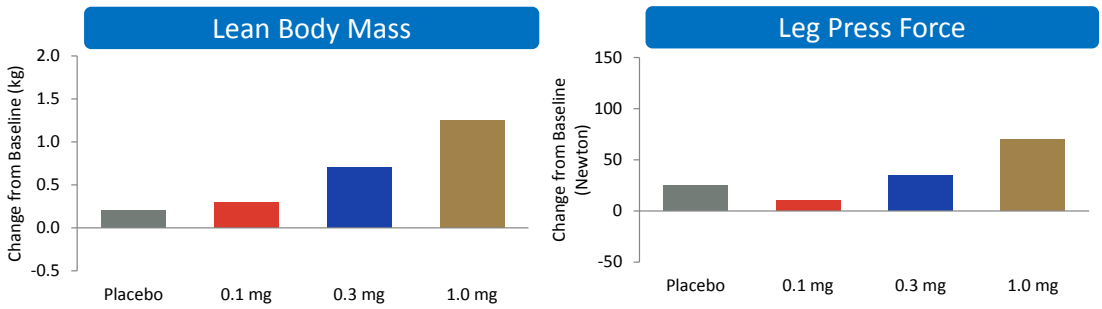
Phase I healthy volunteer results support VK5211's tissue specific lean body mass increase activities. Ligand Pharmaceuticals conducted a Phase I study evaluating LGD-4033 in healthy volunteers and demonstrated the drug's effect in increased lean body mass even during a short study period, and without change in prostate-specific antigen. The drug also exhibited as safe and had a favorable PK profile[3].

The Phase I study illustrated a dose dependent lean body mass but not fat mass. The 1.0mg LGD-4033 (VK5211) group demonstrated a statistically significant mean change vs. placebo (p=0.047).

The study illustrated a dose dependent lean body mass or LBM (Figure 6, left) increase but not in fat mass. Further, the 1.0mg LGD-4033 (VK5211) group demonstrated a statistically significant mean change vs. placebo (p=0.047). The authors stated it was "remarkable" as the significant dose-dependent gains in LBM given the short study duration were not designed to demonstrate maximal effects.

In addition, LGD-4033 (VK5211) also demonstrated change (mean value) in leg press strength (Newton) from baseline (Figure 6, right).

Figure 6: Phase I study demonstrated that VK5211 could enhance lean body mass and leg press force



Source: Company presentation

[3] *Basaria, S., et. al., J Gerontol A Biol Sci Med Sci. 2013;68:87-95*

Yale Jen, Ph.D.
yjen@laidlawltd.com

On the safety side, LGD-4033 was safe and well tolerated at all doses. LGD-4033 also displayed a prolonged elimination half-life (24–36 hours) and linear PK.

The Phase I trial was a placebo-controlled, double-blind, randomized 76-healthy men study. Subjects were randomized to receive either placebo or LGD-4033 (0.1, 0.3, or 1.0 mg) daily for 21 days. The measures include blood counts, chemistries, lipids, prostate-specific antigen, electrocardiogram, hormones, lean mass and fat mass. Muscle strength was measured during and for 5 weeks after intervention.

VK5211 has demonstrated several features: 1) potential to improve lean body mass; 2) potential improvements in bone growth and density; 3) satisfactory safety profile; 4) unique mechanism of action; and 5) potential to be dosed once-daily orally.

Together, VK5211 has demonstrated several features: 1) potential to improve lean body mass – a major contributor to morbidity, disability, re-fracture risk; 2) potential improvements in bone growth and density -- an important factor for increased mortality and re-fracture risk; 3) satisfactory safety profile; 4) unique mechanism of action; and 5) potential to be dosed once-daily orally due to its long plasma half-life.

Next step. VKTX is scheduled to commence a Phase II proof-of-concept (POC) trial in 2H15 to evaluate VK5211 as an acute therapy in rehabilitation for patients who have undergone recent non-elective hip fracture surgery. The study will be a randomized, double-blind, placebo-controlled Phase II trial that intends to enroll 90 – 120 patients, possibly in 10 – 12 clinical sites in the U.S. It plans to evaluate up to three doses of VK5211 for 12 weeks of therapy. VKTX expects the VK5211 doses to be tested at or below 5 mg. The primary endpoint is change in lean body mass; while secondary endpoints include changes in BMD, functional status, re-hospitalization rates and quality of life (QOL). The top-line results are expected in 1H16. VKTX could advance VK5211 development in 2H16 or 2017 pending positive data from the Phase II study.

VKTX is scheduled to commence a Phase II POC trial in 2H15 to evaluate VK5211 as an acute therapy in rehabilitation for patients who have undergone recent non-elective hip fracture surgery.

In addition to post hip surgery rehabilitation improvement, VK5211 could potentially have utility in other indications, such as cancer cachexia. One future indication contemplated by VKTX is cachexia of non-small cell lung cancer (NSCLC) patients with annual incidence of 95,200 in the U.S.

Competitive landscape. We believe the competition in therapeutics for post hip surgery rehabilitation is relatively modest with Bimagrumab from Novartis / Morphosys AG is the most visible competitor. A more tangentially related development is an anti-myostatin antibody, LY2495655, developed by Eli Lilly and this drug currently is in a Phase II study for older fallers with low muscle strength.

We believe the competition in therapeutics for post hip surgery rehabilitation is relatively modest with Bimagrumab from Novartis / Morphosys is the most visible competitor.

Bimagrumab is fully human HuCAL antibody against activin type IIB receptor (ActRIIB) initially developed by Morphosys and partnered with Novartis. Bimagrumab is undergoing clinical studies in several indications, which include sporadic inclusion body myositis (sIBM in musculoskeletal - Phase III), cachexia (both cancer- and COPD-related), hip fracture surgery and sarcopenia (all are in Phase II). Bimagrumab is an antagonist against the ActRIIB receptor

Yale Jen, Ph.D.
yjen@laidlawltd.com

and its mechanism of action is to prevent the binding of natural negative muscle growth regulation ligand, such as myostatin and activin. Bimagrumab has received a breakthrough therapy designation for sIBM. NVS announced recently that it expects to complete the sIBM trial in 4Q15 and file for approval in 2016. The bimagrumab in post hip fracture surgery Phase II study started in 2Q14. It is a randomized, multicenter, double blind, placebo-controlled, and 210-patient Phase II study. Primary endpoints are total lean body mass change at week 24 from baseline measured by DXA; and change from baseline to week 24 in total lean body mass. Some of the secondary endpoints include 1) change from baseline in gait speed at week 24 and week 52; 2) change from baseline to week 24 and from baseline to week 52 in gait speed (meters/sec); and 3) change from baseline in short physical performance battery at week 24 and week 52. Estimated study completion date on clinicaltrial.gov is July 2017.

The mechanism of action of **LY2495655** is by suppressing the activities of myostatin. The drug could down-regulate the negative muscle protein synthetic AKT/mTOR pathway. As such, it could stimulate protein synthesis in muscle fibers, which results in muscle hypertrophy. A recent presentation at the 2015 World Congress on Osteoporosis, Osteoarthritis and Musculoskeletal Diseases (WCO) indicated that LY2495655 significantly improved efficacy over placebo for older fallers, and without safety issues. The results indicated that appendicular lean body mass was significantly increased with LY2495655 vs. placebo by 0.43 kg (2.5%; P <0.001) at week 24. Further, although the placebo and LY2495655 treatment groups showed similar chair rise (16.4 vs 16.8 s, respectively) and hand grip (19.9 vs 20.0 kg) results at baseline, the active treatment group showed greater comorbidities (8.9% vs 11.5%; P=0.011) and trends to more previous fractures (32.7% vs 40.2%; P=0.060), osteoporosis (33.3% vs 47.1%; P=0.061), and vitamin D deficiency (17.2% vs 28.4%; P=0.066).

It was a randomized, double-blind, placebo-controlled and 201-patient Phase II trial. Eligible patients were elderly ($\geq$75 years old) fallers ($\geq$1 fall in prior year) with low muscle strength in standing up (chair rise 5 times without arms, $\geq$12 seconds) and hand grip (women $\leq$21; men $\leq$37 kg). The primary endpoint is the differences in change of appendicular lean body mass at week 24 from baseline for LY2495655 vs. placebo.

Our take on VK5211 Comparing other competing developments and the potential that the anabolic effect could improve the post hip fracture surgery rehabilitation demonstrated by testosterone; we believe these combined factors could bode well for the potential success of VK5211 in improving hip fracture rehabilitation.

- Given testosterone treatment has already exhibited improvements after hip fracture; we believe this fact could mitigate certain clinical risks as to whether VK5211 might provide therapeutic benefits under a similar setting. Further, VK5211 has a better safety profile than testosterone.

Yale Jen, Ph.D.
yjen@laidlawltd.com

One report that examined the use of testosterone +vitamin D3+calcium (anabolic group) vs. calcium only (control group) in elderly women after hip fracture operation indicated anabolic group did not lose muscle volume during the first 12 months whereas the control group did ($p<0.01$)[4]. Further, there was less bone loss in the proximal tibia in the anabolic group than in the control group. The speed of gait and the Harris hip score were significantly better in the anabolic group after six and 12 months.

- The prior Phase I study also demonstrated that VK5211 could stimulate the growth of both muscle and bone, while other in development drugs mainly stimulate muscle growth.

VK5211 has 10 issued and 10 pending patents with earlier expirations between 2025 and 2028. For other SARM products, there are 31 issued and 10 pending patents with earlier expirations between 2017 and 2026.

Solid intellectual property support for VK5211 and other SARM products. VKTX has established a broad intellectual property protection for its VK5211 and other SARM products. For VK5211, there are 10 issued and 10 pending patents with earlier expirations between 2025 and 2028. For other SARM products, there are 31 issued and 10 pending patents with earlier expirations between 2017 and 2026. The geographic coverages of patents are for the U.S. and internationally.

VK5211 in post hip fracture surgery rehabilitation market model assumptions. We assume VK5211 in post hip fracture surgery rehabilitation treatment could receive approval in 2019 after a positive Phase III pivotal study. We model $13k annual treatment costs per patients in the U.S., which is in-line with potential benefits for avoiding costs associated with some of post hip fracture surgery deterioration, including the high propensity of death. We assume VK5211 as an acute therapy and one treatment course encompasses 90 days treatment starting shortly after hip fracture surgery since recent data suggested that muscle loss and BMD deterioration could start within a few weeks post-surgery. We assume product launch in U.S. could start in 2019 and together, we project the annual peak sales could exceed $1 billion (Figure 7a and b).

Figure 7a: VK5211 in post hip fracture surgery revenue model

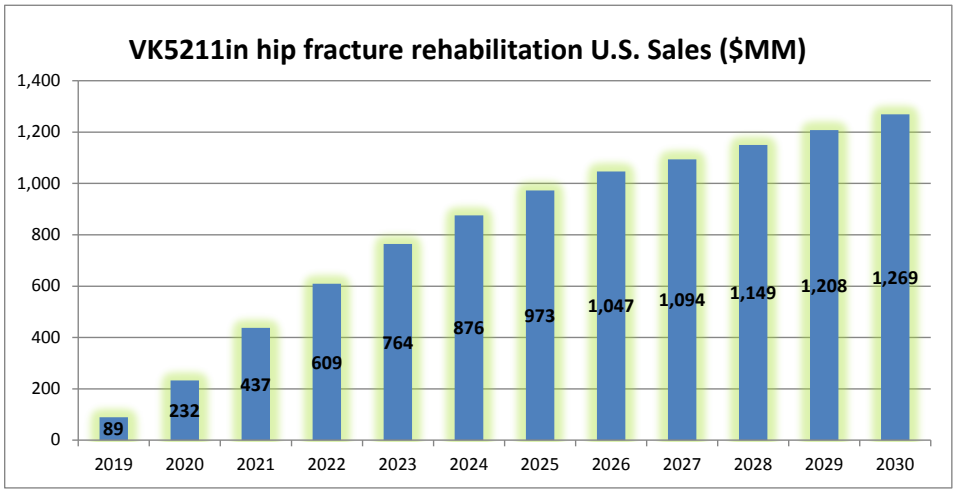
VK5211 in hip fracture revenue model	2019	2020	2021	2022	2023	2024	2025	2026	2027	2028	2029	2030
Total U.S. hip fracture incidences	342,265	349,110	356,093	363,214	370,479	377,888	385,446	393,155	401,018	409,038	417,219	425,564
% of penetration by VK5211	2%	5%	9%	12%	14%	16%	17%	17%	17%	17%	18%	18%
VK5211 treated patients	6,845	17,456	32,048	43,586	53,349	59,706	64,755	68,016	69,376	71,173	73,013	74,899
VK5211 annual treatment costs ($)	13,000	13,317	13,642	13,975	14,316	14,665	15,023	15,390	15,765	16,150	16,544	16,948
U.S. VK5211 in hip fracture sales ($MM)	**89**	**232**	**437**	**609**	**764**	**876**	**973**	**1,047**	**1,094**	**1,149**	**1,208**	**1,269**

Source: Laidlaw & Company estimates

[4] *Hedstrom, M., et. al., (2002) J Bone Joint Surg ;84-B:497-503*

Yale Jen, Ph.D.
yjen@laidlawltd.com

Figure 7b: VK5211 in post hip fracture surgery revenue model



Source: Laidlaw & Company estimates

Yale Jen, Ph.D.
yjen@laidlawltd.com

Thyroid-β Agonists for X-Linked Adrenoleukodystrophy (X-ALD)

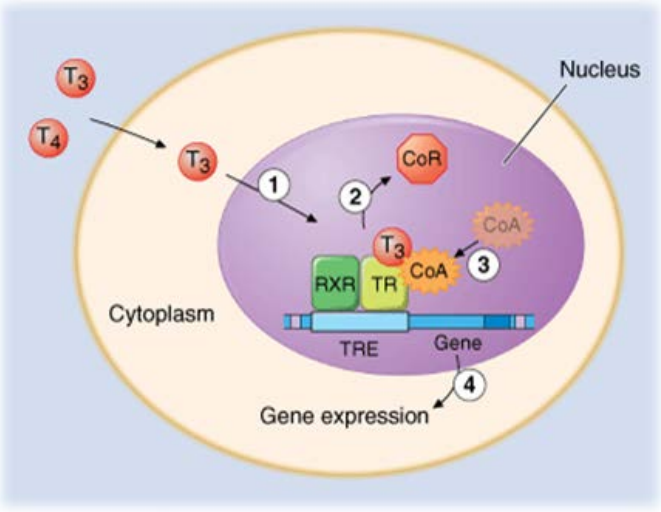
VK2809 and VK0214 are two leading Thyroid-β Agonists

In addition to VK5211, the second lead assets that VKTX will develop near term are thyroid-β agonists (VK2809 and VK0214) as a potential treatment for X-link adrenoleukodystrophy (X-ALD) and possibly several lipid disorders, such as dyslipidemias and nonalcoholic steatohepatitis (NASH).

The biological effects of thyroid hormones are mediated via the activation of thyroid hormone receptors, which in turn modulate the expression of numerous target genes found in various tissues throughout the body

Thyroid receptor overview. Secreted from thyroid gland, thyroid hormones [namely the thyroxine (T4) and triiodothyronine (T3)] are released into the blood stream and transported throughout the body to control metabolism, growth and the physiological function of nearly all organs. The biological effects of thyroid hormones are mediated via the activation of thyroid hormone receptors, which in turn modulate the expression of numerous target genes found in various tissues throughout the body (Figure 8). Several of the recognized effects of thyroid hormones include reduction of lipids and atherogenic lipoproteins associated with cardiovascular disease, induction of weight loss through increased energy expenditure, and induction of ABCD2 expression via induced peroxisomal biogenesis and β-oxidation. To capitalize on these effects, VKTX is developing several thyroid-β agonists that potentially could treat X-ALD, dyslipidemias and NASH.

Figure 8: Molecular pathway of how thyroid agonist affects gene expression



Source: Fauci AS, Kasper DL, Braunwald E, Hauser SL, Longo DL, Jameson JL, Loscalzo J: *Harrison's Principles of Internal Medicine*, 17th Edition: http://www.accessmedicine.com

Source: Company presentation

Yale Jen, Ph.D.
yjen@laidlawltd.com

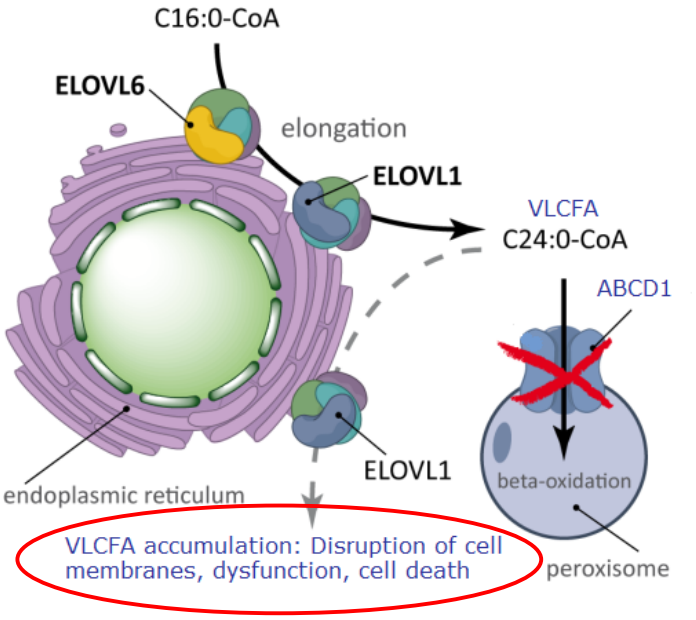
To be successful an effective pharmaceutical agent targeting thyroid hormone receptor would require high target specificity to reduce deleterious effects.

There are four different isoforms of thyroid hormone receptor (TRα1, TRα2, TRβ1 and TRβ2) and they are expressed differentially across different tissues. Different thyroid hormone receptor isoforms' diversified expression patterns resulting in varying impact on different tissues. For example, TRβ are expressed mainly in liver and brain; and they modulate lipid and triglyceride levels. TRα are expressed mainly in cardiac tissues; and they modulate heart rate and contraction. To be successful any effective pharmaceutical agent would require high target specificity to reduce deleterious effects. For instance, a less specific TRß agonist that interacts with both TRα and TRß receptors could activate extra-hepatic thyroid receptors expressed in the heart, resulting in deleterious effects of activation increased respiration and cardiac tissue hypertrophy.

The function of ABCD1 is to transport CoA-activated very long-chain fatty acids (VLCFA) from the cytosol into the peroxisome for degradation. The accumulation VLCFA could lead to the disruption of cell membrane.

X-linked adrenoleukodystrophy summary. Initially called "Addison–Schilder disease", X-linked adrenoleukodystrophy (X-ALD) is a rare and often fatal condition caused by mutations (a total of 634 have been identified) in the gene encoding for the ATP binding cassette transporter D1 (ABCD1). The function of ABCD1 is to transport CoA-activated very long-chain fatty acids (VLCFA) from the cytosol into the peroxisome for degradation. The accumulation VLCFA could lead to the disruption of cell membrane (Figure 9). When this occurs in neural cells, it can cause damage to the myelin sheath, resulting in decreased motor coordination and function, visual and hearing disturbances, loss of cognitive function, dementia, seizures, adrenal dysfunction and possibly death.

Figure 9: Molecular mechanism of X-ALD pathogenesis (ABCD1 blockage, VLCFA accumulation)



Source: Company presentation

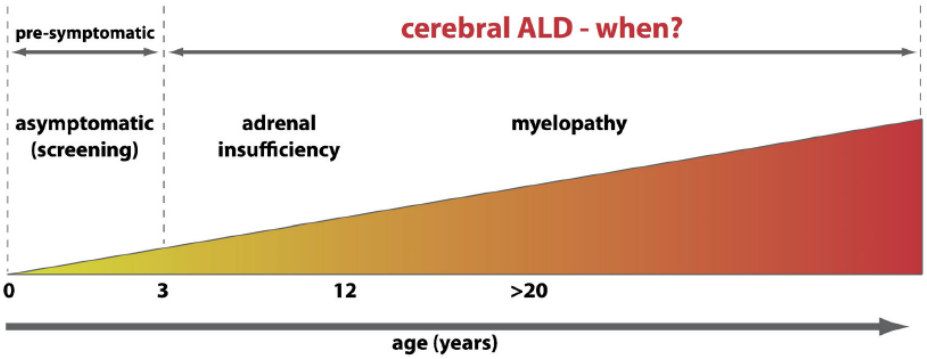
Since the ABCD1 gene is located on the X chromosome, the manifestation of X-ALD symptoms are more likely to be reflected in males relative to females since the latter has two copies of X chromosome.

Yale Jen, Ph.D.
yjen@laidlawltd.com

X-ALD can be further categorized into two different subtypes, cerebral adrenoleukodystrophy and adrenomyeloneuropathy, mainly determined by the presence or absence of brain inflammation.

X-ALD can be further categorized into two different subtypes, cerebral adrenoleukodystrophy and adrenomyeloneuropathy, mainly determined by the presence or absence of brain inflammation. Earlier symptoms in male X-ALD patients are usually adrenocortical insufficiency; with onset often occurring between ages six and 12 years. Chronic myelopathy develops in adults most often starting in the third decade of life (Figure 10)[5]. More than 70% of all male X-ALD patients suffer from adrenocortical insufficiency, which usually is the initial presentation prior to the onset of neurological symptoms.

Figure 10: The clinical spectrum of X-linked adrenoleukodystrophy (X-ALD)



Source: Engelen, M., et. al. Curr. Neurol Neurosci Rep. (2014) 14: 486-493

Cerebral adrenoleukodystrophy (CALD): It is the most severe form of ALD and it accounts for ~35% of the total X-ALD cases based on a report presented at Orphanet[6]. Due to the progressive inflammatory destruction of myelin, patients suffer severe loss of neurological function and this eventually could lead to death. It is estimated that in male ALD patients, ~35% to 40% present cerebral involvement at a younger age (5 to 12 years of age); while up to 20% develop cerebral involvement later in life (20 to 35 years of age). The most frequent first clinical manifestations of disease in male CALD children are learning and behavioral problems. Without intervention, CALD patients typically experience rapid degeneration into a vegetative state within three to five years, often resulting in death within 10 years of diagnosis.

Adrenomyeloneuropathy (AMN) is the more common and is considered as the default form of ALD in patients surviving beyond childhood. AMN accounts for approximately half of all patients diagnosed with ALD.

Adrenomyeloneuropathy (AMN): It is the more common and is considered as the default form of ALD in patients surviving beyond childhood. Symptoms of AMN patients generally are progress more slowly, resulting from non-inflammatory disruption of the axons in the spinal cord. AMN accounts for approximately half of all patients diagnosed with ALD. Symptoms of AMN patients include weakness in the legs, impaired vibration sense, incontinence and impotence. In some AMN patients who progress into more severe motor disability, such as lower limb paralysis, within a 3 to 15 year period, the use of a wheelchair or cane is needed. Disease progression of many AMN sufferers could worsen over time as 35% of them experience a rapid progression of myelopathy; while ~40% could progress into CALD, with varying degrees of

[5] *Engelen, M., et. al. Curr. Neurol Neurosci Rep. (2014) 14: 486-493*
[6] *http://www.orpha.net/consor/cgi-bin/OC_Exp.php?Lng=GB&Expert=43.0*

associated inflammations. Most AMN patients also suffer with adrenocortical insufficiency. All adult males with ABCD1 mutations and ~65% female are categorized as AMN. The age AMN is diagnosed in males is usually made between the ages of 20 and 50 and in females after the age of 65.

We conservatively estimate the prevalence of X-ALD in the U.S. is of 7,708. X-ALD is the most common hereditary peroxisomal disorder, and there are no approved or disease-modifying drug treatments. .

A much milder form of X-ALD is called Addison's disease and patients might only experience symptom of adrenocortical insufficiency. The symptoms could begin anytime between childhood and adulthood. Many patients could also develop other types of symptoms by the time they reach middle age.

According to the estimate of prevalence of 1 in 21,000 males[7], we conservatively estimate the prevalence of X-ALD in the U.S. is of 7,708. It is also estimated that 65% of carrier females develop the disease by 60 years old. X-ALD is the most common hereditary peroxisomal disorder. There are no approved or disease-modifying drug treatments for X-ALD. For early stage CALD patients, allogenic hematopoietic cell transplantation (HCT) could help to halt the progression of the disease mainly by re-acquiring a properly functioning copy of the ABCD1 gene contributed by a donor. However, recent data suggest AMN can develop later in life even among successfully transplanted patients. Treatments for X-ALD symptoms, such as the progression of chronic myelopathy, are mainly symptom alleviation and supportive care by rehabilitation physicians.

For early stage CALD patients, allogenic hematopoietic cell transplantation (HCT) could help to halt the progression of the disease.

For AMN patients, treatments are often limited to symptom alleviation, such as against spasticity, sphincter dysfunctions, impotence and neuropathic pain, and active physical rehabilitation. Dietary therapy, such as "Lorenzo's oil" (a 4:1 mixture of glyceryl trioleate and glyceryl trierucate), sometimes used in combined with a diet has been shown to reduce VLCFA in plasma. However, no evidence suggested this treatment is able to improve, halt or even slow down the progression of the disease.

Potential mechanism of action of thyroid-β agonists treating X-ALD. Although the cause of X-ALD is mutations of the ABCD1 gene, resulting in impairment of transporting CoA-activated VLCFA from the cytosol into the peroxisome for degradation; there are two other ABC transporters. These are ABCD2 and ABCD3, and are localized in the peroxisomal membrane. With substantially less effectiveness (↓45x) in mediating direct or indirect transport of C26:0-CoA, across the peroxisomal membrane, endogenous ABCD3 is unable to rescue the metabolic defect in X-ALD patients. ABCD2 is also not expressed at relevant amounts under normal circumstances and therefore, did not compensate for the loss of ABCD1 in X-ALD. One approach is stimulating ABCD2 production both hepatic and non-hepatic (such as glial cells) via thyroid-β agonists (an established regulatory relationship) possibly in an appropriate manner. With ABCD2 as the closest homolog of ABCD1, it is possible that such an approach could compensate for the loss of ABCD1 and reverse the accumulation VLCFA in X-ALD patients. Prior pre-clinical studies

By stimulating ABCD2 both hepatic and non-hepatic (such as glial cells) production via thyroid-β agonist possibly in an appropriate manner, and the fact that ABCD2 is the closest homolog of ABCD1, it is possible that such approach could compensate the loss of ABCD1 and reverse the accumulation VLCFA in X-ALD patients.

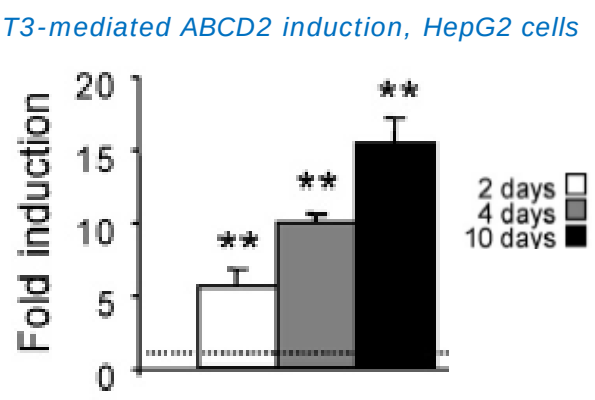
[7] *Matern, D., Presentation at the 2014 SACHDNC Meeting.*

Yale Jen, Ph.D.
yjen@laidlawltd.com

of both *in vitro* analyses and transgenic knockout mouse models have demonstrated ABCD2 overexpression prevents VLCFA accumulation and restores β-oxidation. Further, the highly selective TRβ agonists could have the advantage of being devoid of side effects, such as the cardiotoxicity associated to TRα activation.

Pre-clinical study of thyroid-β agonist demonstrated potential treatment potential in X-ALD. One example[8] of an *in vivo* study that demonstrates thyroid-β agonist has stimulated ABCD2 production in tissue culture (HepG2 cells) is illustrated in Figure 11. In this study, by applying a thyroid-β agonist (3,3',5-triiodo-l-thyronine sodium salt) in human HepG2 cells, ABCD2 gene expression was demonstrated in a continued increase manner from day 2 to day 10.

Figure 11: Thyroid-β agonist stimulate ABCD2 gene expression in HepG2 cell line



Source: Company presentation

VKTX is developing two thyroid-β agonists (VK2809 and VK0214) and will determine which one to move forward into Phase I study in an X-ALD Phase I study in 1Q16. The company is also in collaboration with researchers at University of Amsterdam (Drs. Stephan Kemp and Ronald Wanders), a major X-ALD research center, to conduct more in-depth preclinical analyses with results expected possibly in mid-2015. In addition, the company is also evaluating two different thyroid-β agonists (VK2809 and VK0214) as a more proper drug candidate for treating X-ALD and we also expect a decision could be made in mid-2015.

Our understanding is that both VK2809 and VK0214 are active while the former is more liver targeted while the latter has more systemic effect. One of the decisions is to determine which one could potentially provide greater therapeutic impact on X-ALD patients.

Next step. Although details are limited, we believe the upcoming Phase I study will be an open label multicenter, dose-finding trial that evaluates VK2809 or VK0214 in 6 – 12 X-ALD patients (mainly AMN type) in up to three doses to

[8] *Genin, E. C., et. al., (2009) J Steroid Biochemistry & Molecular Biology 116 37–43*

Phase I study will be an open label multicenter, dose-finding trial that evaluates VK2809 or VK0214 in 6 – 12 X-ALD patients (mainly AMN type) in up to three doses to assess safety and tolerability, changes in VLCFA, functional status and quality of life through 12 weeks of therapy in the U.S.

assess safety and tolerability, changes in VLCFA, functional status and quality of life through 12 weeks of therapy in the U.S. VKTX expects to start the study in early 2016 and possibly complete in mid-2016 with top-line results shortly thereafter. If the outcome is positive, VKTX plans to start a discussion with the FDA, possibly in 2H16 / 2017 to identify the clinical path going forward. In addition, management believes that thyroid-β agonists could also afford therapeutic benefits to cerebral adrenoleukodystrophy (CALD) patients as well and expects development could start after advancements in AMN are already in place.

The most advanced competitor is the gene therapy Lenti-D, currently is in Phase II/III clinical trial in children CALD (n=15) developed by Bluebird Bio

Competition: The competition for therapeutic developments in X-ALD is also relatively modest. The most advanced one is the gene therapy **Lenti-D**, currently in a Phase II/III (Starbeam) clinical trial in children CALD (n=15) developed by Bluebird Bio. The Lenti-D approach utilizes an *ex vivo* insertion of a functional copy of the ABCD1 gene driven by an Lentivirus vector into a patient's own $CD34^+$ hematopoietic stem cells (HSCs) to restore portion of the ABCD1 functions. An earlier French Phase I/II study (TG04.06.01 with n=4), that evaluated an approach similar to that of Lenti-D, demonstrated prolonged disease stabilization, significant reduction in neuro-inflammation and substantially improved mortality (vs. ~20% mortality rate in the same two-year window post-allogeneic HSCT).

We view the clinical results of the gene therapy as promising and Lenti-D therapy potentially could be approved, since allogeneic HSCT is effective. Lenti-D therapy might be more eligible for pediatric CALD patients when a matching hematopoietic stem cell donor cannot be found. We believe Lenti-D's treatment coverage could be more limited due to its likely very high costs and more limited size of the eligible patient cohort (children CALD with n~ 200) addressed by the therapy.

Another development in X-ALD treatment is **MD1003** by France-based privately owned MedDay Pharmaceuticals. MD1003 is a highly concentrated formulation of biotin (≥300 mg /day), which could activate enzymes involved in cellular energy production and myelin synthesis. The company is conducting a MD1003 in adult X-ALD (in AMN type) Phase IIb/III clinical study in Europe. The trial is funded by the European Leukodystrophy Association (ELA) and involves four reference centers in France, Germany and Spain. MD1003 is also in a Phase IIb/III clinical study in primary and secondary progressive multiple sclerosis (MS). Although promising, we believe the probability of success for MD1003 could be modest since the drug essentially is a high dose vitamin. In addition, the therapeutic impact of MD1003 could only be symptom mitigation, not disease modification as it does not tackle the cause of the disorder.

VK0214 has is one issued U.S. patent with earlier expiration in 2024. For other TRß agonists, there are one issued and two pending patents with earlier expiration in 2026.

Solid intellectual property support for VK0214 and other TRß agonists. VKTX has established a broad intellectual property protection for its VK0214 and other TRß agonists. For VK0214, there is one issued U.S. patent with earlier expiration in 2024. For other TRß agonists, there are one issued and two pending patents with earlier expiration in 2026.

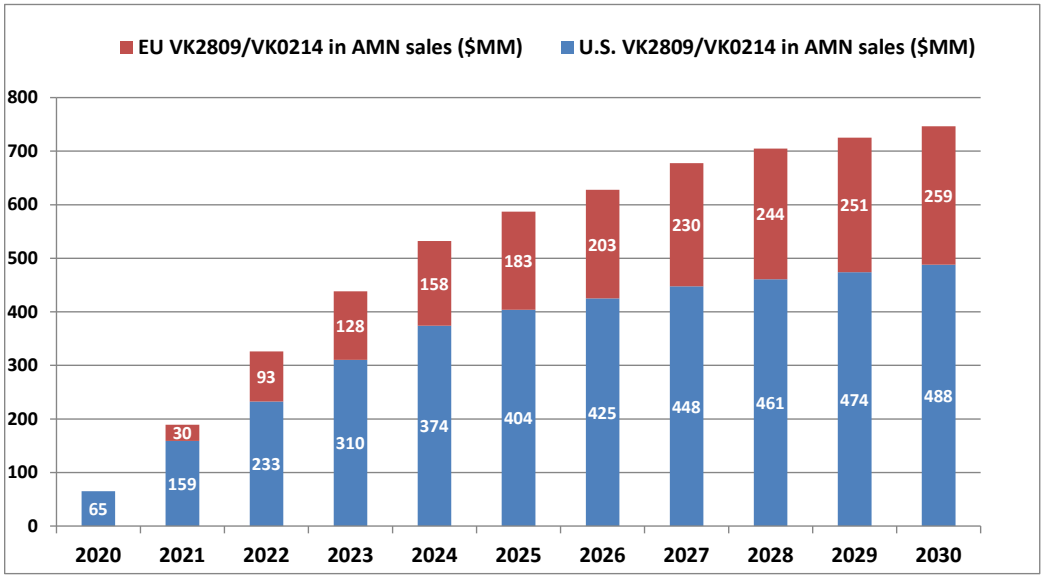
Thyroid-β agonist in X-linked adrenoleukodystrophy revenue model assumptions. We assume VK2809/VK0214 in X-ALD (more specifically AMN) treatment could potentially receive approval in 2020 after a positive pivotal (Phase II/III) study. We model a $200k annual treatment costs per patients in the U.S., which is in-line with the mid-end of ultra-orphan disease therapy costs. We assume product launch in ex-U.S. could start in 2021 and together, we project the annual global peak sales could reach $750MM (figure 12a and b).

Figure 12a: Thyroid-β agonists in X-linked adrenoleukodystrophy revenue model

Thyroid-β agonists in X-linked adrenoleukodystrophy Revenue Model	2014	2020	2021	2022	2023	2024	2025	2026	2027	2028	2029	2030
Total U.S.X-linked adrenoleukodystrophy prevalence	7,708	8,134	8,207	8,281	8,355	8,431	8,506	8,583	8,660	8,738	8,817	8,896
% of adrenomyeloneuropathy (AMN) patients	50%	50%	50%	50%	50%	50%	50%	50%	50%	50%	50%	50%
Adrenomyeloneuropathy patients	3,854	4,067	4,103	4,140	4,178	4,215	4,253	4,291	4,330	4,369	4,408	4,448
% of penetration by VK2809/VK0214		8%	19%	27%	35%	41%	43%	44%	45%	45%	45%	45%
VK2809/VK0214 treated patients		325	780	1,118	1,462	1,728	1,829	1,888	1,949	1,966	1,984	2,002
VK2809/VK0214 annual treatment costs ($)		200,000	204,000	208,080	212,242	216,486	220,816	225,232	229,737	234,332	239,019	243,799
U.S. VK2809/VK0214 in AMN sales ($MM)		**65**	**159**	**233**	**310**	**374**	**404**	**425**	**448**	**461**	**474**	**488**
Total EU adrenomyeloneuropathy (AMN) patients	3,276	3,457	3,488	3,519	3,551	3,583	3,615	3,648	3,681	3,714	3,747	3,781
% treated by AFM11VK2809/VK0214			5%	15%	20%	24%	27%	29%	32%	33%	33%	33%
AMN patients treated with VK2809/VK0214			174	528	710	860	976	1,058	1,178	1,226	1,237	1,248
VK2809/VK0214 annual treatment costs ($)			173,400	176,868	180,405	184,013	187,694	191,448	195,277	199,182	203,166	207,229
EU VK2809/VK0214 in AMN sales ($MM)			**30**	**93**	**128**	**158**	**183**	**203**	**230**	**244**	**251**	**259**
Global VK2809/VK0214 in AMN sales ($MM)		**65**	**189**	**326**	**438**	**532**	**587**	**628**	**678**	**705**	**725**	**747**

Source: Laidlaw & Company estimates

Figure 12b: Thyroid-β agonists in X-linked adrenoleukodystrophy revenue model



Source: Laidlaw & Company estimates

Yale Jen, Ph.D.
yjen@laidlawltd.com

VK2809 Also Has the Potential to Treat Dyslipidemias and NASH

VK2809 could potentially be used to treat dyslipidemias and NASH

Like VK5211, thyroid-β agonists such as VK2809 were also in-licensed from Ligand Pharmaceuticals. VK2809 is part of the Thyroid Receptor β Agonist Program initially developed by Metabasis Therapeutics, which was later acquired by Ligand in 4Q09. One of the objectives of the thyroid receptor β agonist program was to use its HepDirect prodrug technology and other structural characteristics to develop liver-targeted therapeutics that could reduce LDL, triglycerides and LP(a) while avoiding dose-limiting side effects due to activation in extra-hepatic tissues.

By modulating TRß receptor in liver tissue selectively, a thyroid-β agonist could generate a favorable cholesterol and lipoprotein profile via multiple mechanisms, including increasing the expression of low-density lipoprotein receptors and increasing mitochondrial fatty acid oxidation.

VK2809 has potential to be a hyperlipidemia treatment agent. It is known that by selectively modulating TRß receptor in liver tissue, a thyroid-β agonist could generate a favorable cholesterol and lipoprotein profile via multiple mechanisms, including increasing the expression of low-density lipoprotein receptors and increasing mitochondrial fatty acid oxidation. As such, a highly tissue specific thyroid-β agonist, such as VK2809, could potentially act as an effective therapy to reduce LDL cholesterol, plasma and liver triglycerides.

In prior Phase Ib studies, VK2809 demonstrated dose-related reductions in fasting LDL-C and fasting triglyceride levels at day 14.

In prior Phase Ib studies conducted by Metabasis Therapeutics, VK2809 demonstrated dose-related reductions in fasting LDL-C and fasting triglyceride levels at day 14. At dose 5 mg and above, VK2809 showed significant placebo-adjusted LDL-C reductions from baseline ranging from 15% – 41% (Figure 13). At doses of 2.5 mg or greater, a placebo-adjusted triglyceride levels were reduced by >30%. Further, the study also showed statistically significant reductions of lipoprotein a (Lp(a)) and apolipoprotein (Apo(B)) in certain cohorts. On the safety side, VK2809 was safe and well-tolerated across doses ranging from 0.25 mg to 40 mg per day. No serious adverse events were identified and CV side effect regarding heart rate, heart rhythm and blood pressure, were similar between the treatment and placebo groups.

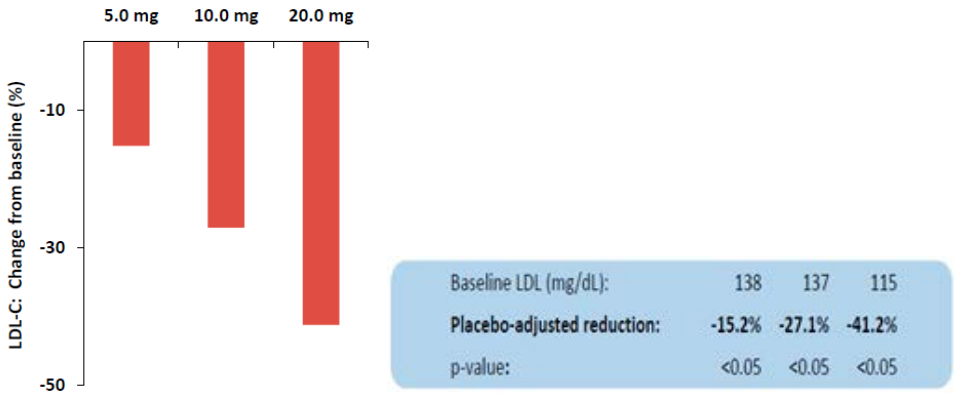
It was a placebo-controlled, dose ranging Phase Ib trial that enrolled 56 mild hypercholesterolemia patients. Patients were dosed from 0.25 mg to 40 mg with once a day treatment. The LDL criterion for enrolled patient was $\geq$ 100 mg /dL.

From pre-clinical analyses, VK2809 has shown to be very potent in binding to TRß receptor with Ki of 2.2nM; and high specificity with 16:1 selectivity for the beta receptor over the alpha receptor. VK2809 has demonstrated an additive

Yale Jen, Ph.D.
yjen@laidlawltd.com

effect in combination with atorvastatin (Lipitor) in cholesterol lowering without drug/drug interactions.

Figure 13: VK2809 Phase Ib study results of LDL-C reductions



Source: Company presentation

Together, VK2809 has demonstrated promising preliminary results from its Phase I studies. Given its preliminary but favorable profile, VK2809 could compare favorably with the lipid lowering activities of existing oral agents for hyperlipidemia (Figure 14).

Figure 14: VK2809 could compare favoralbly against other oral hyperlipidemia agents

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	-18	-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	-23	-36	-	-28	Gallstones, risk of kidney dysfunction, skeletal muscle toxicity

(1) All data presented as placebo-adjusted % change from baseline.

Source: Company presentation

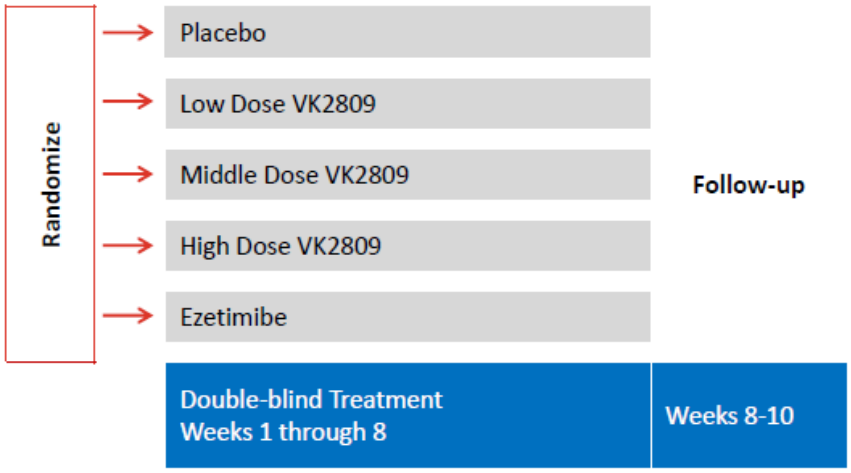
Given its preliminary but favorable profile, VK2809 could compare favorably with the lipid lowering activities of existing oral agents for hyperlipidemia

Next step. VKTX is scheduled to start a VK2809 in hypercholesterolemia Phase IIa study, possibly in 4Q15 if sufficient funding is available. The planned Phase IIa trial is a double-blind, multi-arm, dose ranging study that intends to enroll ~150 patients. The treatment duration is eight weeks with two weeks of post-treatment follow-up. The primary endpoint is the change of LDL-C level at

Yale Jen, Ph.D.
yjen@laidlawltd.com

eight weeks comparing to baseline. Secondary endpoints include changes of Lp(a) and change s in liver fat measured by MRI (Figure 15).

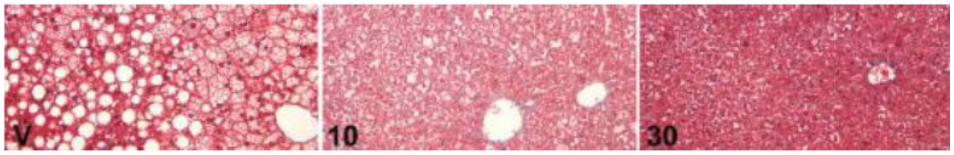
Figure 15: Potential VK2809 in hypercholesterolemia Phase II study design



Source: Company presentation

VK2809 has potential as a treatment for NASH. In pre-clinical studies in rodents, VK2809 also exhibited potential benefits in reducing fat content in the liver (Figure 16). As such, it is possible that VK2809 could have the potential as a treatment in disorders characterized with excessive accumulation of lipids in liver tissue, such as NASH. VK2809 treatment also did not show any impact on transaminase activities.

Figure 16: VK2809 showed to reduce fat content in animal hepatic steatosis model (from left to right: Vehicle, 10, 30 mg/kg/day)



Source: Company presentation

Yale Jen, Ph.D.
yjen@laidlawltd.com

Multiple Assets with Large Market Potential Could Advance With Additional Financial or Partnership Support

In addition to the key assets, VK5211 and the two thyroid-β agonists, VK2809 and VK0214, VKTX also has several clinical assets that have larger market potential: VK0612 (a Phase IIb-ready drug candidate for type 2 diabetes), EPO receptor (EPOR) agonist program (pre-clinical as a potential treatment of anemia), and diacylglycerol acyltransferase-1 (DGAT-1) inhibitor program (pre-clinical as a potential treatment of lipid disorders such as obesity and dyslipidemia).

Three products are: 1) VK0612 (a Phase IIb-ready drug candidate for type 2 diabetes); 2) EPO receptor (EPOR) agonist program (pre-clinical as a potential treatment of anemia); and 3) diacylglycerol acyltransferase-1 (DGAT-1) inhibitor program (pre-clinical as a potential treatment of lipid disorders such as obesity and dyslipidemia).

VK0612, a selective fructose-1,6-bisphosphatase (FBPase) inhibitor, is a first-in-class, orally available drug candidate for type 2 diabetes (T2D). Given that FBPase plays an important role in endogenous glucose production, its inhibition could potentially control blood glucose levels in diabetes patients. FBPase might have a novel mechanism of action in lowering blood glucose levels since it is independent from the pancreatic function. Prior Phase I and IIa studies have indicated that estimated plasma HbA1c reduction by VK0612 treatment of >1%, which exceeds the typical anti-glycemic effects of newer drug classes. In addition, VK0612 is well-tolerated and has not demonstrated hypoglycemia, lacticemia, or other drug-related safety issues. Additional data suggested that VK0612 could maintain a weight and lipid neutral profile; while the drug potentially can be dosed once daily. The company currently has 12 issued and three pending patents for VK0612 with both the U.S. and international coverages, while the earliest expiration is in 2019-2020. The company currently also has 12 issued and one pending patents for FBPase inhibitor combinations with both the U.S. and international coverages, while the earliest expiration is in 2019-2021.

VKTX plans to start a Phase IIb clinical trial to evaluate VK0612 (possibly at a dose $\leq$ 300mg) in T2D in the future if funding is available.

EPOR agonist program. VKTX is scheduled to develop small molecule agonists of the erythropoietin (EPO) receptor as a potential treatment of anemia. It is an orally administrated treatment, which could potentially circumvent the inconveniences of injection administrated current therapies. all are human EPO and other erythropoiesis-stimulating agents, or ESAs. The company currently has 12 issued and one pending patents for EPOR inhibitors with both the U.S. and international coverages, while the earliest expiration is in 2030-2031. VKTX plans to conduct further preclinical studies and file an IND at a future date.

Yale Jen, Ph.D.
yjen@laidlawltd.com

DGAT-1 inhibitor program. VKTX is developing small molecule inhibitors of the enzyme DGAT-1 for the potential treatment of lipid disorders such as obesity and dyslipidemia. DGAT-1 can reduce triglyceride levels in the circulation and fat accumulation in adipose tissues. Pre-clinical animal model studies have suggested that if without DGAT-1, post-meal plasma triglyceride levels could reduce while energy expenditure increases. It also does not impact on normal levels of circulating free fatty acids. If an animal with excess DGAT-1, their adipose tissue are predisposed to obesity when fed a high-fat diet and have elevated levels of circulating free fatty acids. The company currently has two issued and one pending patents for DGAT-1 inhibitors with both the U.S. and European coverages, while earliest expiration is in 2030. VKTX plans to conduct further preclinical studies and file an IND at a future date.

Agreements with Ligand Pharmaceuticals. VKTX entered into a Master License Agreement with Ligand Pharmaceuticals on May 21, 2014 (amended on April 8, 2015) to license the exclusive worldwide rights of VK5211, VK2809, VK0214, VK0612 and two preclinical programs. VKTX will pay Ligand an upfront fee of $29MM, commercial milestone payments of up to $1.54 billion, and single-digit royalties based on future worldwide net product sales. . Ligand received ~3.4 million shares of stock as an equity milestone for license programs. In addition, Ligand also purchased 1.125 million shares at $8 per share for a total of $9MM during VKTX's IPO. Ligand currently owns ~49% of VKTX shares.

Yale Jen, Ph.D.
yjen@laidlawltd.com

Financial Projections and Valuation

The company recently (May 5, 2015) completed an initial public offering (Laidlaw and Co. was the sole book-running manager) by issuing 3.45 million shares at $8 per share with gross proceeds of $27.6MM. After deducting the expenses, we estimate the company has cash of ~$24MM. Together, we believe the cash should support the company's operations into 2017, by our estimate.

Our probability-adjusted DCF analysis suggested a one-year target value for VKTX of $20.16 based on cash flow until 2025 with an assumed terminal value multiple of two and a conservative probability adjustment of 24%.

Probability-adjusted DCF analysis

Cash driven NPV	2016	2017	2018	2019	2020	2021	2022	2023	2024	2025
Revenue	0	0	0	88,989	297,528	626,498	935,093	1,202,202	1,407,990	1,559,876
R&D	(8,957)	(15,495)	(21,538)	(23,476)	(25,355)	(27,129)	(29,028)	(31,060)	(33,235)	(35,561)
SG&A	(3,568)	(4,781)	(5,020)	(36,271)	(59,784)	(65,486)	(68,761)	(72,199)	(75,808)	(79,599)
Operating income	(12,524)	(20,276)	(26,558)	29,241	212,389	533,883	837,304	1,098,943	1,298,947	1,517,251
Net income	(12,932)	(19,776)	(26,058)	18,737	134,120	336,661	527,817	692,649	818,652	956,183
Period	0.6	1.6	2.6	3.6	4.6	5.6	6.6	7.6	8.6	9.6
NPV	(11,360)	(13,952)	(14,767)	8,529	49,034	98,861	124,494	131,223	124,573	116,869

Total DCF	613,503
Terminal value	175,303
Cash (4Q16)	9,000
Total valuation ($ '000)	797,806
Probability adjustment	24%
Value per share	**$20.16**
Share outstanding (2015)	9,654
Discount rate	25%
Terminal value multiple	2

Source: Laidlaw & Company estimates

Given the limited competition for the indications for which VKTX is developing therapeutics, we do not find many perfectly matched comparable peers. Although Bluebird and Morphosys are developing therapeutics of the similar indications as VKTX, the relevant program is just a portion, or not the leading value driver of respective company's pipeline (Morphosys: 23 and Bluebird: 4). As such, we do not consider these companies are fair peers in our comparable analysis.

For the peer comparable analysis, we have chosen a group with a mix of endocrine and rare disease developing companies given these are the two indications that VKTX are actively developing near term. As such, our peer comparable analysis suggested a 12-month target price for VKTX of $20.45.

We note that two clinical top-line results reporting (VK5211 in post hip fracture surgery rehabilitation Phase II trial and VK2809/VK0214 in X-ALD with focus on adrenomyeloneuropathy (AMN) Phase I trial) will be available in the next 12 to 15 months as in mid-2016 to 3Q16. Positive outcomes, in our opinion, could represent an inflection point to VKTX share value.

Yale Jen, Ph.D.
yjen@laidlawltd.com

Comparable analysis

Company	Ticker	Rating	Target Price ($)	Price ($) (5/29/15)	Shares Outstanding (MM)	Market Cap ($ MM)	Cash ($ MM)	Debt ($ MM)	Tech Value ($ MM)	Most Advanced Development Stage	Disease Group	Major Indication
Protalix BioTherapeutics	PLX	NR	NA	1.94	93	181	48	68	200	Marketed	Rare	Gaucher and, Fabry diseases
Corcept Therapeutics	CORT	NR	NA	6.12	108	659	38	0	622	Marketed	Rare	Cushing's syndrome
GlycoMimetics	GLYC	NR	NA	7.97	19	152	47	0	105	Phase II	Rare	SCD
Galmed Pharmaceuticals	GLMD	NR	NA	10.57	11	117	30	0	87	Phase II	Endocrine	NASH
Ampio Pharmaceuticals	AMPE	NR	NA	2.40	52	125	43	0	82	Phase III	Endocrine	Osteoarthritis of the knee
Palatin Technologies	PLT	NR	NA	0.88	42	37	37	0	0	Phase II	Endocrine	FSD, obesity
Versartis	VSAR	NR	NA	15.34	29	449	230	0	219	Phase III	Endocrine	Growth hormone deficiency
Conatus Pharmaceuticals	CNAT	NR	NA	5.45	20	108	31	1	78	Phase II	Endocrine	Liver diseases
Average						325	*73*	*8*	174			
Viking Therapeutics	VKTX	Buy	20.00	8.23	10	79	24	0	56	Phase II		Hip fracture, X-ALD
RNN share fair value matching its Phase I/II oncology peers =									$20.45			
Potential upside =									149%			

Source: Company reports and Laidlaw & Company estimates

In addition, our probability-adjusted-PV-driven, sum-of-the-parts analysis illustrates a breakdown of value for each potential value driver, with VK5211 in post hip fracture surgery rehabilitation accounting for 60% of the total value, while VK2809/VK0214 in X-ALD with focus on adrenomyeloneuropathy (AMN), thyroid-β agonists for future indications and other programs account for 23%, 8% and 6%, respectively. As such, our supplemented probability-adjusted-PV-driven, sum-of-the-part analysis suggested a 12-month target price of $20.

NPV driven sum-of-the-parts analysis

VK5211	Post hip fracture surgery rehabilitation			
		Adjusted NPV =	$162.9	
		PV per share =	$11.93	60%
VK2809/VK0214	X-ALD (AMN)			
		Adjusted NPV =	$61.9	
		PV per share =	$4.53	23%
Thyroid-β agonists	Multiple indications			
		Adjusted NPV =	$22.0	
		PV per share =	$1.61	8%
Other programs	T2D and others			
		Adjusted NPV =	$17.3	
		PV per share =	$1.27	6%
Cash				
		Adjusted NVP =	$9.0	
		NVP per share =	$0.66	3%
		Total =	$20.00	100%

Source: Laidlaw & Company estimates

Together, we assigned our blended 12-month target price for VKTX of **$20**.

Yale Jen, Ph.D.
yjen@laidlawltd.com

Major risks

Risks of clinical study failure could have a major impact on VKTX share value. Despite promising aspects of the company's lead products, VK5211 in the post hip fracture surgery rehabilitation and thyroid-β agonists (VK2809 / VK0214) in X-ALD, it remains too early to predict the safety and efficacy from the two upcoming Phase I and Phase II studies. Given that clinical validation or POC for these programs has not been established, it would be critical for these studies to demonstrate a positive outcome in order to increase the asset and shareholder value. Negative results of either clinical study could potentially impair their value and have a materially negative impact on shareholder value, especially since success of each study could illustrate the value of VK5211 in hip fracture rehabilitation and thyroid-β agonists in X-ALD. Further, it remains too early to predict any potential future success of clinical trials should these programs further advance into next stage clinical stage development. In thyroid-β agonists in X-ALD, although it is possible that the drug could reduce or eliminate VLCFA, it remains too early to forecast that the drug could slow and stop the progression of symptoms to provide clinical benefits.

Product may not be approved or reach anticipated sales. Although Viking's current pipeline products have exhibited the potential to generate positive clinical outcomes from current and future trials; it remains too early to project whether any of these products would be approved by regulatory agencies. Even if the products were to enter the market, sales could be significantly below projections due to the specific product label under approval, physician consensus for prescribing the drug, changes of treatment paradigms, entrance of competitors, and possibly the changes in pricing flexibility and payer reimbursement. A revenue outlook below expectations could also negatively affect VKTX shareholder value.

Positive relationship with Ligand is important. Given that Viking is substantially dependent on technologies and drug candidates licensed from Ligand for further development, it would be important for the company to maintain a positive relationship with Ligand. If Viking loses the right to license these technologies and drug candidates or the Master License Agreement with Ligand is terminated for any reason, VKTX's ability to develop existing and new drug candidates would be harmed.

Additional financings could dilute shareholder value. Although the company currently has ~$23MM cash after recent IPO financing, VKTX could need more financial resources going forward if they want to expand and further develop its pipeline. Should the product not receive FDA approval, or product revenue does not reach expectations; the company might need to issue new equity to raise additional cash. Under such a scenario, the share value of existing shareholders could be diluted.

Limited trading liquidity limits shareholder options. Given VKTX shares only entered the public market recently; daily trading volume and name recognition are relatively modest. With relatively illiquid trading volume, shareholders wanting to increase or reduce their positions in a volatile stock market may face constraints.

Yale Jen, Ph.D.
yjen@laidlawltd.com

Management

Brian Lian, Ph.D. is Viking Therapeutics CEO since 2012 and he joined the company as Chief Commercial Officer since 2010 of its inception. Prior joining Viking from 2012 to 2013, he was a managing director and senior research analyst at SunTrust Robinson Humphrey. Prior to SunTrust Robinson Humphrey from 2011 to 2012, he was managing director and senior research analyst at Global Hunter Securities. Prior from 2008 to 2011, he was senior healthcare analyst at the Agave Group, LLC. Prior to The Agave Group from 2006 to 2008, he was an executive director and senior biotechnology analyst at CIBC World Markets. Prior to CIBC, he was a research scientist at Amgen, a biotechnology company; and before that, he was a research scientist at Microcide Pharmaceuticals. Dr. Lian holds a Ph.D. degree in organic chemistry from the University of Michigan and an MBA in accounting and finance from Indiana University.

Michael Morneau has served as CFO of Viking Therapeutics since May 2014 Prior to Viking, from 2009 to 2014, he was VP of Finance and Chief Accounting Officer at Trius Therapeutics. Prior to Trius, from 2008 to 2009, he was Director of Lilly Research Labs Finance at Eli Lilly and company. Prior to Eli Lilly, from 2006 to 2008, he was Director of Finance and Accounting at SGX Pharmaceuticals. Prior to SGX from 2004 to 2006, he was Controller at Momenta Pharmaceuticals. Mr. Fischer holds a MBA and MA degree in accounting from New Hampshire College.

Michael Dinerman, M.D. has served as Chief Operating Officer of Viking since 2012 and he also served as a member of board of directors from inception until May 2014. Prior joining Viking from 2009 to 2013, he was a research analyst with Piper Jaffray & Co. covering the Specialty Pharmaceuticals and Medical Device industries. Prior to Piper Jaffray from 2005 to 2008, he was an associate and, later a director at CIBC World Markets. Dr. Dinerman holds a M.D. degree from University of Cincinnati College of Medicine and an MBA from the Freeman School of Business at Tulane University.

Rochelle Hanley M.D. has served as Chief Medical Officer of Viking Therapeutics since 2013. Prior joining Viking From 2011 to 2013, Dr. Hanley was an independent consultant to the pharmaceutical industry. Prior to that from 2008 to 2011, she was Medical Director, Cardiovascular, Metabolic and Musculoskeletal Diseases at GlaxoSmithKline. Prior to GSK from 2006 to 2008, she served as Chief Medical Officer for Quatrx Pharmaceuticals. Prior to Quatrx, she was VP and Clinical Site Head, and before that, as VP and Therapeutic Area Development Leader, Cardiovascular and Metabolic Diseases at Pfizer. Prior, she was Senior Director, Endocrine and Diabetes Clinical Development, Parke Davis Pharmaceutical Research. Prior to Parke Davis, she was International Therapeutic Head, Metabolic Diseases, Glaxo Wellcome. Prior to Glaxo Wellcome, Dr. Hanley was an Assistant Professor of Duke University Medical Center. Dr. Hanley holds a M.D. from the University of Michigan.

Yale Jen, Ph.D.
yjen@laidlawltd.com

Viking Therapeutics – Income Statement

($',000)	2012	2013	2014	1Q15E	2Q15E	3Q15E	4Q15E	2015E	2016E	2017E	2018E	2019E	2020E	2021E
Revenue														
Product revenue	0.0	0.0	0.0	-	-	-	-	0	0	0	0	88,989	297,528	626,498
Other revenue	0.2	0.0	0.0	-	-	-	-	0	0	0	0	0	0	0
Total revenue	0.2	0.0	0.0	-	-	-	-	0	0	0	0	88,989	297,528	626,498
Costs of goods												10,679	35,703	75,180
Gross sales												78,310	261,825	551,318
Research and development	(69)	(12)	(22,223)	(320)	(701)	(1,507)	(2,019)	(4,547)	(8,957)	(15,495)	(21,538)	(23,476)	(25,355)	(27,129)
General and administrative	(41)	(89)	(1,245)	(420)	(567)	(658)	(750)	(2,395)	(3,568)	(4,781)	(5,020)	(5,271)	(5,534)	(5,811)
Marketing and sales												(31,000)	(54,250)	(59,675)
Total Operating Expenses	(110)	(101)	(23,468)	(740)	(1,268)	(2,164)	(2,769)	(6,941)	(12,524)	(20,276)	(26,558)	(59,747)	(85,139)	(92,616)
Operating Incomes (losses)	(109)	(101)	(23,468)	(740)	(1,268)	(2,164)	(2,769)	(6,941)	(12,524)	(20,276)	(26,558)	29,241	212,389	533,883
Change in fair value of accrued license fees	0	0	(1,822)	(2,400)	0	0	0	(2,400)	0	0	0	0	0	0
Change in fair value of debt conversion features	0	21	(391)	(500)	(200)	(100)	100	(700)	(200)	(500)	(500)	(500)	(500)	(500)
Amortization of debt discount	0	18	558	420	420	420	420	1,680	840	0	0	0	0	0
Interest expense	1	6	71	34	34	34	34	136	68	0	0	0	0	0
Total other (income) expenses	1	45	(1,584)	(2,446)	254	354	554	(1,284)	708	(500)	(500)	(500)	(500)	(500)
Loss before tax	(111)	(146)	(21,884)	1,706	(1,522)	(2,518)	(3,323)	(5,657)	(13,232)	(19,776)	(26,058)	29,741	212,889	534,383
Tax	0	0	0	-	-	-	-	0	0	0	0	(11,004)	(78,769)	(197,722)
Net Income (Loss)	(111)	(146)	(21,884)	1,706	(1,522)	(2,518)	(3,323)	(5,657)	(13,232)	(19,776)	(26,058)	18,737	134,120	336,661
Net Income (Loss) Applicable to Common Shareholders	(111)	(146)	(21,884)	1,706	(1,522)	(2,518)	(3,323)	(5,657)	(13,232)	(19,776)	(26,058)	18,737	134,120	336,661
Net Earnings (Losses) Per Share—Basic	($0.07)	($0.07)	($5.23)	$0.28	($0.16)	($0.26)	($0.34)	($0.64)	($0.97)	($1.26)	($1.48)	$0.83	$5.92	$14.86
Net Earnings (Losses) Per Share—Diluted	($0.07)	($0.07)	($5.23)	$0.28	($0.16)	($0.26)	($0.34)	($0.64)	($0.97)	($1.26)	($1.48)	$0.83	$5.92	$14.86
Shares outstanding—basic	1,483	2,043	4,187	6,150	9,650	9,652	9,654	8,777	13,654	15,654	17,654	22,654	22,657	22,659
Shares outstanding—diluted	1,483	2,043	4,187	6,150	9,650	9,652	9,654	8,777	13,654	15,654	17,654	22,654	22,657	22,659
Margin Analysis (% of Sales/Revenue)														
Costs of goods												12%	12%	12%
R&D	-33433%	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	-26%	-9%	-4%
SG&A	-19791%	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	-6%	-2%	-1%
Operating Income (loss)	-53124%	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	33%	71%	85%
Pretax	-537.9223	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	33%	72%	85%
Tax Rate										0%	37%	37%	37%	37%
Net Income	-53792%	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	21%	45%	54%
Financial Indicator Growth Analysis (YoY%)														
Total Revenue	NA	-100%	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	234%	111%
R&D	NA	-83%	191264%	540%	NA	NA	-91%	-80%	97%	73%	39%	9%	8%	7%
SG&A	NA	119%	1292%	163%	NA	NA	-21%	92%	49%	34%	5%	5%	5%	5%
Marketing and sales					NA	NA							75%	10%
Operating Income (Losses)	NA	-8%	23118%	253%	NA	NA	-88%	-70%	80%	62%	31%	-210%	626%	151%
Pretax Income	NA	32%	14864%	-845%	NA	NA	-84%	-74%	134%	49%	32%	-214%	616%	151%
Net Income	NA	32%	14864%	-845%	NA	NA	-84%	-74%	134%	49%	32%	-172%	616%	151%
EPS	NA	-4%	7202%	-487%	NA	NA	-83%	-88%	50%	30%	17%	-156%	616%	151%

Yale Jen, Ph.D. 212-953-4978

Source: Bloomberg LP; Company reports; Laidlaw & Company estimates

Viking Therapeutics – Balance Sheet

($'000)	2012	2013	2014	1Q15E	2Q15E	3Q15E	4Q15E	2015E	2016E
Assets									
Cash and cash equivalents	0	180	756	1,134	23,609	21,816	19,638	19,638	6,104
Short term investments	0	0	0					0	0
Liqid assets	***0***	***180***	***756***	***1,134***	***23,609***	***21,816***	***19,638***	***19,638***	***6,104***
Prepaids and other current assets	0	0	18	19	20	24	26	26	29
Total Current Assets	0	180	774	1,153	23,629	21,840	19,664	19,664	6,133
Deferred IPO financing costs	0	0	2,269	2,700	-	-	0	0	0
Deposits	0	1	1	1	1	1	1	1	1
Total Assets	**0**	**180**	**3,043**	**3,854**	**23,630**	**21,841**	**19,665**	**19,665**	**6,134**
Liabilities and Stockholders' Equity									
Accounts payable	49	73	1,831	1,886	1,704	1,715	1,943	1,943	2,010
Accounts payable – related party	5	1	0	-	-	-	0	0	0
Accrued license fees	0	0	19,866	19,311	17,442	17,412	17,910	17,910	19,690
Other accrued liabilities	0	0	380	284	314	297	357	357	342
Accrued interest	0	7	77	47	39	43	67	67	0
Convertible notes payable, current portion	0	47	304	347	320	298	390	390	0
Debt conversion feature liability	0	0	59	47	52	50	78	78	0
Total current liabilities	54	127	22,517	21,922	19,871	19,815	20,746	20,278	22,042
Convertible notes payable	43	232	1,264	1,164	1,151	1,890	1,994	1,994	300
Debt conversion feature liability	8	72	1,390	1,190	1,051	1,099	1,191	1,191	1,191
Total Liabilities	105	431	25,172	24,277	22,074	22,804	23,932	23,932	23,533
Common stock	0	0	0	0	0	0	0	0	0
Preferred stock	0	0	0	-	-	-	0	0	-
Additional paid-in capital	6	11	13	13	23,513	23,513	23,532	23,532	23,632
Notes receivable from stockholders	0	(4)	0	-	-	-	0		
Accumulated Deficit	(111)	(257)	(22,141)	(20,435)	(21,957)	(24,476)	(27,799)	(27,799)	(41,031)
Total Stockholders' Equity	(105)	(251)	(22,129)	(20,423)	1,556	(963)	(4,266)	(4,266)	(17,399)
Total Liabilities and Stockholders' Equity	**-**	**180**	**3,043**	**3,854**	**23,630**	**21,841**	**19,665**	**19,665**	**6,134**

Yale Jen, Ph.D. 212-953-4978

Source: Bloomberg LP; Company reports; Laidlaw & Company estimates

Yale Jen, Ph.D.
yjen@laidlawltd.com

Viking Therapeutics – Cash Flow Statement

($'000) Cash Flows From Operating Activities:	2012	2013	2014	1Q15E	2Q15E	3Q15E	4Q15E	2015E	2016E
Net income (loss)	(111)	(146)	(21,884)	1,706	(1,522)	(2,518)	(3,323)	(5,657)	(13,232)
Adjustments to reconcile net loss to net cash used in operating activities:									
Amortization of discount charged to interest expense on convertible notes	1	18	558	410	290	310	790	1,800	500
Change in fair value of debt conversion feature	0	21	(391)	500	200	100	(100)	700	200
Change in fair value of accrued license fees	0	0	(1,822)	(2,400)	0	0	0	(2,400)	0
Stock-based compensation	6	4	6	109	189	309	209	816	830
Changes in operating assets and liabilities:									
Prepaids and other current assets	0	0	(18)	(1)	(1)	(4)	(2)	(8)	(3)
Deposits	0	(1)	0	(0)	0	0	0	(0)	0
Accounts payable	49	24	191	55	(182)	11	229	113	67
Accounts payable – related party	5	(4)	(1)	0	0	0	0	0	0
Accrued expenses	0	6	81	0	0	0	0	0	0
Accrued license fees	0	0	21,688	0	0	0	0	0	0
Net Cash from Operating Activities	**(50)**	**(78)**	**(1,591)**	**379**	**(1,026)**	**(1,793)**	**(2,197)**	**(4,637)**	**(11,639)**
Cash flows from investing activities:									
	0	0	0	0	0	0	0	0	(1,995)
Net Cash from Investing Activities	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**(1,995)**
Cash Flows From Financing Activities:									
Proceeds from issuance of common stock	0	0	0	0	23,500	0	19	23,519	100
Repurchase of common stock	0	(3)	(0)	0	0	0	0	0	0
Proceeds from convertible notes payable	50	260	2,500	0	0	0	0	0	0
Deferred IPO financing costs paid	0	0	(332)	0	0	0	0	0	0
Net Cash Provided by Financing Activities	**50**	**258**	**2,168**	**0**	**23,500**	**0**	**19**	**23,519**	**100**
Net increase (decrease) in cash	**0**	**180**	**576**	**379**	**22,474**	**(1,793)**	**(2,178)**	**18,882**	**(13,534)**
Cash at beginning of period	0	0	180	756	1,134	23,609	21,816	756	19,638
Cash at end of period	**0**	**180**	**756**	**1,134**	**23,609**	**21,816**	**19,638**	**19,638**	**6,104**

Yale Jen, Ph.D. 212-953-4978

Source: Bloomberg LP; Company reports; Laidlaw & Company estimate

Yale Jen, Ph.D.
yjen@laidlawltd.com

DISCLOSURES:

ANALYST CERTIFICATION

The analyst responsible for the content of this report hereby certifies that the views expressed regarding the company or companies and their securities accurately represent his personal views and that no direct or indirect compensation is to be received by the analyst for any specific recommendation or views contained in this report. Neither the author of this report nor any member of his immediate family or household maintains a position in the securities mentioned in this report.

EQUITY DISCLOSURES

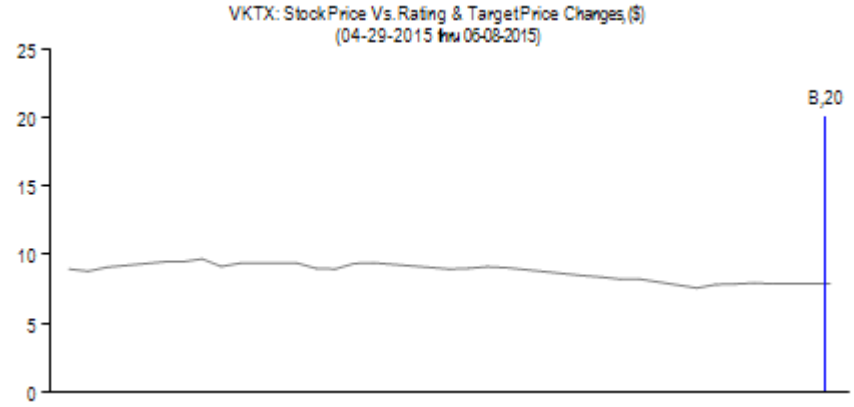
For the purpose of ratings distributions, regulatory rules require the firm to assign ratings to one of three rating categories (i.e. Strong Buy/Buy-Overweight, Hold, or Underweight/Sell) regardless of a firm's own rating categories. Although the firm's ratings of Buy/Overweight, Hold, or Underweight/Sell most closely correspond to Buy, Hold and Sell, respectively, the meanings are not the same because our ratings are determined on a relative basis against the analyst sector universe of stocks. An analyst's coverage sector is comprised of companies that are engaged in similar business or share similar operating characteristics as the subject company. The analysis sector universe is a sub-sector to the analyst's coverage sector, and is compiled to assist the analyst in determining relative valuations of subject companies. The composition of an analyst's sector universe is subject to change over time as various factors, including changing market conditions occur. Accordingly, the rating assigned to a particular stock represents solely the analyst's view of how that stock will perform over the next 12-months relative to the analyst's sector universe.

Additional information available upon request.

‡ Laidlaw & Company has received compensation from the subject company for investment banking services in the past 12 months and expects to receive or intends to seek compensation for investment banking services from the company in the next three months.

RATINGS INFORMATION

Rating and Price Target Change History



3 Year Rating Change History

Date	Rating	Closing Price ($)
06/08/2015	Buy (B)	7.89*

3 Year Price Change History

Date	Target Price ($)	Closing Price, ($)
06/08/2015	20.00	7.89*

** Previous Close6/5/2015*

Source: Laidlaw & Company **Created by: Blue-Compass.net**

Laidlaw & Company Rating System*		% of Companies Under Coverage With This Rating	% of Companies for which Laidlaw & Company has performed services for in the last 12 months	
			Investment Banking	Brokerage
Strong Buy (SB)	Expected to significantly outperform the sector over 12 months.	0.00%	0.00%	0.00%
Buy (B)	Expected to outperform the sector average over 12 months.	75.00%	32.14%	7.14%
Hold (H)	Expected returns to be in line with the sector average over 12 months.	3.57%	0.00%	0.00%
Sell (S)	Returns expected to significantly underperform the sector average over 12 months.	0.00%	0.00%	0.00%

ADDITIONAL COMPANIES MENTIONED

Ligand Pharmaceuticals (LGND – Not Rated)
Valeant Pharmaceuticals (VRX – Not Rated)
Novartis (NVS – Not Rated
Morphosys AG (MOR GY – Not Rated)
Eli Lilly (LLY – Not Rated)
Bluebird Bio (BLUE – Not Rated)

ADDITIONAL DISCLOSURES

As of the date of this report, neither the author of this report nor any member of his immediate family or household maintains an ownership position in the securities of the company (ies) mentioned in this report.

This report does not provide individually tailored investment advice and has been prepared without regard to the individual financial circumstances and objectives of persons who receive it. Laidlaw & Co (UK), Ltd. recommends that investors independently evaluate particular investments and strategies, and encourages investors to seek the advice of a financial adviser. The appropriateness of a particular investment or strategy will depend on an investor's individual circumstances and objectives. The securities, instruments, or strategies discussed in this report may not be suitable for all investors, and certain investors may not be eligible to purchase or participate in some or all of them. This report is not an offer to buy or sell or the solicitation of an offer to buy or sell any security/instrument or to participate in any particular trading strategy.

Associated persons of Laidlaw & Co (UK), Ltd not involved in the preparation of this report may have investments in securities/instruments or derivatives of securities/instruments of companies mentioned herein and may trade them in ways different from those discussed in this report. While Laidlaw & Co (UK), Ltd., prohibits analysts from receiving any compensation. Bonus or incentive based on specific recommendations for, or view of, a particular company, investors should be aware that any or all of the foregoing, among other things, may give rise to real or potential conflicts of interest.

With the exception of information regarding Laidlaw & Co (UK), Ltd. this report is based on public information. Laidlaw & Co (UK), Ltd makes every effort to use reliable, comprehensive information, but we make no representation that it is accurate or complete and it should not be relied upon as such. Any opinions expressed are subject to change and Laidlaw & Co (UK), Ltd disclaims any obligation to advise you of changes in opinions or information or any discontinuation of coverage of a subject company. Facts and views presented in this report have not been reviewed by, and may not reflect information known to, professionals in other Laidlaw & Co (UK), Ltd business areas. Laidlaw & Co (UK), Ltd associated persons conduct site visits from time to time but are prohibited from accepting payment or reimbursement by the company of travel expenses for such visits. The value of and income from your investments may vary because of changes in interest rates, foreign exchange rates, default rates, prepayment rates, securities/instruments prices. market indexes, operational or financial conditions of companies or other factors. There may be time limitations on the exercise of options or other rights in securities/instruments transactions. Past performance is not necessarily a guide to future performance. Estimates of future performance are based on assumptions that may not be realized. If provided, and unless otherwise stated, the closing price on the cover page is that of the primary exchange for the subject company's securities/instruments.

Any trademarks and service marks contained in this report are the property of their respective owners. Third-party data providers make no warranties or representations of any kind relating to the accuracy, completeness, or timeliness of the data they provide and shall not have liability for any damages of any kind relating to such data. This report or any portion thereof may not be reprinted, sold or redistributed without the written consent of Laidlaw & Co (UK), Ltd. This report is disseminated and available primarily electronically, and, in some cases, in printed form.

The information and opinions in this report were prepared by Laidlaw & Co (UK), Ltd. For important disclosures, please see Laidlaw & Co (UK), Ltd.'s disclosure website at www.Laidlawltd.com, or contact your investment representative or Laidlaw & Co (UK), Ltd at 546 Fifth Ave, 5th Floor, New York, NY 10036 USA.

Yale Jen, Ph.D.
yjen@laidlawltd.com

NOTES:

Yale Jen, Ph.D.
yjen@laidlawltd.com