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Company Report

December 17, 2018

Tiziana Life Sciences (TLSA - $8.25)

Healthcare / Biotechnology

Initiating Coverage – Novel Mechanisms to Attack Large Unmet Medical Needs in Immunology and Oncology

Ticker:	TSLA
Rating:	**Buy**
Price Target:	**$17.00**

We are initiating coverage of Tiziana Life Sciences (TLSA) with a Buy rating and a $17 price target. TLSA is a biotechnology company focused on discovery and development of novel molecules in immunology and oncology. Their main value driver consists of Foralumab (fully human anti-CD3 oral mAb) in development as a potential treatment for large unmet medical needs such as NASH. TLSA may seem very early in their development of Foralumab as they have only initiated one of their two Phase 1 trials. However, we consider the program relatively de-risked as a previous orally administered anti-CD3 mAb (OKT3) in NASH and T2D patients showed encouraging immunological trends such as increase in Treg markers and anti-inflammatory markers. We believe Foralumab's fully human nature as opposed to OKT3 (fully murine) could enhance efficacy and especially decrease toxicity concerns. TLSA expects to initiate their oral Phase 1 for NASH in 1Q19 and recently announced (11/28/19) the start of their second Phase 1 (intranasal delivery) for patients with MS. Their next value driver is Milciclib (orally bioavailable, small molecule broad spectrum CDK inhibitor) for the treatment of Hepatocellular Carcinoma (HCC). With its novel ability to reduce levels of microRNAs (miR-221 and miR-222), which may be linked to blood supply in cancer tumors and resistance to standard of care Sorafenib, we believe Milciclib could help treat a patient population in dire need of a new treatment options. Having met its primary endpoints in two previous Phase 2 clinical trials in patients with Thymic Carcinoma and Thymoma, we also see Milciclib as relatively de-risked. TLSA has initiated a Phase 2a trial as a monotherapy (interim look: well tolerated) in patients with HCC in 4Q17 and anticipate initiating a Phase 2b trial in combination with Sorafenib in 1H19. With a relatively de-risked pipeline in large unmet medical needs, we are initiating coverage with a Buy rating and a $17 price target.

- **Foralumab, a novel relatively de-risked approach to fulfill a large unmet medical need.** While Foralumab remains in Phase 1, we see their fully human oral anti-CD3 mAb as relatively de-risked as fully murine OKT3 showed encouraging immunological trends in a previous Phase 2 trial in NASH.
- **Milciclib's differentiated MOA builds on strong data.** That Milciclib hit its primary endpoint in two previous Phase 2 trials helps de-risk the program as their novel MOA (decrease in microRNAs) could benefit HCC patients.
- **Initiate with a Buy rating, $17 PT.** Our PT is based on Foralumab royalties at $12.5/share; Milciclib royalties at $3/share; cash (end'19) and tech value at $1.5/share.

Trading Data:

Last Price (12/14/2018)	$8.25
52–Week High (11/21/2018)	$12.17
52–Week Low (12/06/2018)	$6.61
Market Cap. (MM)	$126.1
Shares Out. (MM)	118.0

Earnings Estimates: (per share)

(Dec)	1Q	2Q	3Q	4Q	FY	P/E
FY19E	(0.18)	(0.18)	(0.20)	(0.21)	(0.77)	NA
FY18E	(x.xx)	(x.xx)	(x.xx)	(0.07)E	(0.54)	NA
FY17A	(x.xx)	(x.xx)	(x.xx)	(x.xx)	(0.09)	NA
FY16A	(x.xx)	(x.xx)	(x.xx)	(x.xx)	(0.11)	NA

Source: Laidlaw & Company estimates

Analyst
Francois Brisebois, MSc/ Specialty Pharma & Biotech
(857) 317-5362
fbrisebois@laidlawltd.com

5 Key Reasons to own Tiziana Life Sciences

1. **Foralumab, a relatively de-risked approach for a large unmet medical need.** Although TLSA remains in its first stages of Phase 1 clinical trials having yet to initiate their oral formulation for NASH and only very recently (11/28/19) started their intranasal dosing in patients for MS, we view Foralumab as relatively de-risked as similar MOA has shown efficacy in the past. A Phase 2 clinical trial evaluating OKT3 (fully-murine anti-CD3 mAb) demonstrated immunological benefits (increase in T reg and anti-inflammatory biomarkers). AST, glucose, as well as insulin levels were also decreased, which we see as a significant positive as this trial wasn't powered for efficacy.

2. **With a novel MOA, Foralumab seems differentiated in this increasingly competitive field.** As the first fully human anti-CD3 mAb, we are particularly encouraged by TLSA's novel approach to attacking the underlying cause of NASH. Although NASH is far from fully understood as a disease state, most would agree that combination therapy represents the most likely therapeutic path forward and we believe this potentially safer immunological approach could fit nicely in the treatment paradigm.

3. **Milciclib's differentiated MOA builds on strong data.** We see Milciclib's ability to reduce levels of microRNAs (miR-221 and miR-222), which seem related to blood supply in cancer tumors as a real positive. We are particularly encouraged that Milciclib met its primary endpoints in two Phase 2 clinical trials in patients with thymic carcinoma and thymoma as this helps de-risk their Phase 2 clinical trial in HCC, in our opinion.

4. **Whether it be by monotherapy or combination therapy, HCC patients are in need of new treatment options.** Since combination of Sorafenib+Milciclib could have a combined anti-proliferative effect on tumor cells as they attack different targets, TLSA has opted to test Milciclib in HCC as monotherapy and combination therapy. Regardless, current standard of care Sorafenib's ability to extend survival probability from 7.9 months to 10.7 months indicates a clear unmet medical need in this patient population.

5. **2019 is setting up to be a catalysts filled year.** With potential data readouts in both their oral and intranasal Foralumab trials as well as their Phase 2a monotherapy Milciclib trial in HCC, it is hard to overstate the importance of catalysts at TLSA in 2019.

Francois Brisebois
fbrisebois@laidlawltd.com

Figure 1: Upcoming Potential Catalysts

Event	Expecting Timing
Foralumab Phase 1 oral IND	1Q19
Foralumab Phase 1 oral data	2H19
Foralumab Phase 1 intranasal data	2Q19
Milciclib Phase 2a monotherapy data	2Q19

Source: Company Reports; Laidlaw and Company estimates

Valuation

We value TLSA at $17/share based on a sum-of-the-parts valuation. TLSA Foralumab US royalties are valued at $11/share based on a 8x multiple of 2028 US royalties of $590M, discounted back 9 years at a 45% discount rate. Foralumab EU royalties are valued at $1.50/share based on 8x multiple of 2030 EU royalties of $153M, discounted back 11 years at a 45% discount rate. Milciclib WW royalties are valued at $3.00/share based on 8x multiple of 2026 WW royalties of $54M, discounted back 7 years at a 37.5% discount rate. We value net cash (end 2019) and technology at $1.50/share.

Figure 2: Sum-of-the-Parts Analysis

Sum-of-the-parts valuation		
Segment	Valuation (000's)	Per share value
Foralumab US royalties	$166,623	$11.00
Foralumab EY royalties	$20,585	$1.50
Milciclib WW royalties	$46,491	$3.00
Cash (end '19) & tech value	$22,739	$1.50
	$235,853	**$17.00**
2019 fully diluted shares out (000)		14,991

Source: Company Reports; Laidlaw and Company estimates

Francois Brisebois
fbrisebois@laidlawltd.com

Company Description

TLSA is a biotechnology company focused on the discovery and development of novel molecules to treat high unmet medical needs in immunology and oncology. Their lead product candidate in immunology is Foralumab (TZLS-401), which is the only fully human anti-CD3 monoclonal antibody (mAb), in clinical development. MAbs represent a single pure antibody produced by single clones and are an important class of human therapeutics for treating cancers and autoimmune diseases. Generation of antibodies for use in humans developed in animals can lead to strong immune responses limiting their effectiveness and potentially leading to severe side effects. Humanization removes most of the animal components of the antibody and lowers the immune response from the human immune system. Their lead product candidate in oncology is Milciclib (TZLS-201), which is an orally bioavailable, small molecule broad spectrum inhibitor of cyclin-dependent kinases (CDKs), and Src family kinases. CDKs are a highly conserved family of enzymes that phosphorylate a specific group of proteins that are involved in regulating the cell cycle. They also have a drug discovery pipeline of small molecule new chemical entities (NCEs), and biologics.

Francois Brisebois
fbrisebois@laidlawltd.com

Foralumab for NASH, CD & MS

TLSA is developing Foralumab (in-licensed from Novimmune in December 2014) as a potential treatment for non-alcoholic steatohepatitis (NASH), Crohn's Disease (CD) as well as neurodegenerative diseases such as multiple sclerosis (MS). They believe that oral or intranasal administration of Foralumab has the potential to reduce inflammation while minimizing side effects.

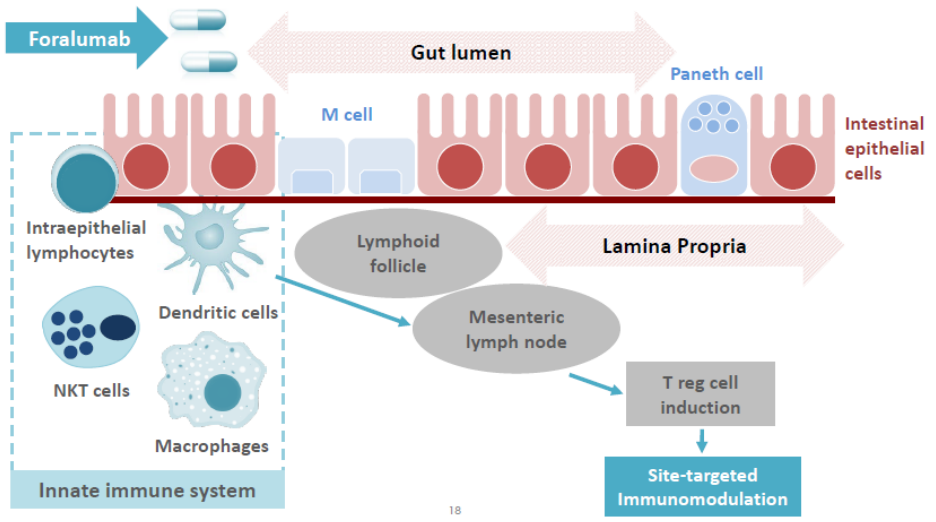
Foralumab has been studied in one Phase 1 and two Phase 2a clinical trials conducted by Novimmune (n=68) dosed by the IV route of administration. Foralumab was well-tolerated and produced immunologic effects consistent with potential clinical benefit while demonstrating mild to moderate infusion related reactions (IRR). TLSA plans to initially investigate Foralumab for safety and its immunomodulatory activity in healthy volunteers in two Phase 1 trials. In one of them, subjects will be dosed in a newly developed oral formulation with an intent to treat patients with NASH and/or CD (expected to initiate 1Q19) and in the other trial, Foralumab will be dosed via intranasal administration (hand held nasal device) for patients with MS (initiated on 11/28/18).

An IND for the first-in human evaluation of Foralumab was filed on 6/1/18 and accepted by the FDA on 8/20/18. On 4/16/18 they entered into an exclusive license agreement with The Brigham and Women's Hospital (BWH), relating to a novel formulation of Foralumab in a medical device for intranasal administration. Foralumab consists of the only fully human anti-CD3 mAb in clinical development, in contrast to the previous non-human or humanized anti-CD3 mAbs. It targets the CD3 epsilon (CD3ε) receptor, which is a recognized approach for modulating T-Cell response and achieving immunosuppression. Additionally, Foralumab could have broad application to autoimmune and inflammatory diseases, such as NASH, CD, MS, T1D, inflammatory bowel disease, psoriasis and RA, where modulation of a T-cell response is desirable. In July 2017, TLSA announced a publication demonstrating for the first time the potential of oral therapy with Foralumab for inflammatory diseases such as NASH. In fact the article showed oral Foralumab's ability to prevent skin xenograft rejection in humanized mice (Clinical Immunology, 2017).

Francois Brisebois
fbrisebois@laidlawltd.com

Autoimmune diseases are primarily due to a malfunction in the immune system leading it to attack certain cells in the body as foreign invaders. The CD3ε molecule, along with 4 other membrane-bound polypeptides (CD3γ, CD3δ, CD3ζ, CD3ϟ) form the CD3 complex, which is associated to the T-cell receptor. Upon antigen bindings, the CD3 complex sends signals through the cell membrane to the cytoplasm inside the T-cell, which leads to activation of the T-cell that divides to produce new T-cells to fight the particular antigen to which the TCR was exposed. While T-cell activation is critical for the human immune system to properly fight bacterial, viral or parasitic infections, abnormal T-cell induction can cause and worsen human diseases such as T-cell lymphoma and leukemia, human malignancies, autoimmune disorders, cardiovascular disease (CVD) and transplant rejection.

Figure 3: MOA Foralumab



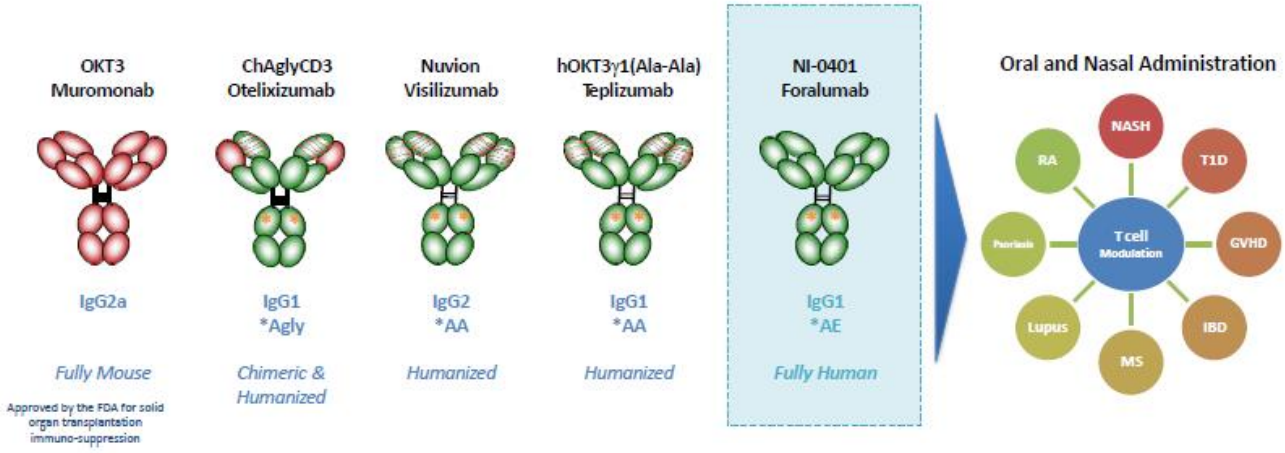
Source: Company Reports

Since the discovery of the hybridoma technology, a method to generate large quantities of a single (monoclonal) antibody, the production and manufacture of mAbs has shown benefits in several autoimmune and inflammatory disease clinical trials and therapeutic utility in animal models. The first murine anti-CD3 mAb (IgG2a) was developed and approved by the FDA in 1985 under the name of muromonab, OKT3 to treat allograft rejection in kidney, liver and heart transplantation by exerting its immunosuppressive effects, mainly due to depletion of T-cells in tissues helping prevent rejection of the allografts. OKT3 was administered in clinical trials to patients with MS, T1D, IBD, RA and NASH. Although showing promise to alleviate the disease process, the mAb being of murine origin and extremely immunogenic in humans, was associated with a wide range of side effects that include the typical CRS (flu-like syndrome). The side effect profile of OKT3 is a consequence of T-cell activation resulting in the release of numerous cytokines into the systemic circulation. These side effects of the murine OKT3 led to the development of a new generation of anti-CD3 mAbs using genetic engineering of the mAb structure. Foralumab dosed IV has been observed to alter T-cell function via antigenic modulation, that is, removal of the CD3/TCR complex from the T-cell surface. Modulation has two therapeutic

Francois Brisebois
fbrisebois@laidlawltd.com

benefits such as it transiently renders the T-cells incapable of recognizing an antigen and thus unable to have an immune response such as an allograft rejection and it has a favorable long-term effect on generation and maintenance of regulatory T-cells, a specialized subset of T-cells that promote immunological tolerance.

Figure 4: CD3-specific monoclonal antibodies in clinical development



Source: Company reports

In comparison with the two other anti-CD3 mAbs evaluated in patients with T1D, otelixizumab by Tolerx and teplizumab by (MGNX), Foralumab was less mitogenic, therefore allowing re-treatment, and had a better risk/benefit profile. Recent clinical studies with oral administration of anti-CD3 (OKT3, murine mAb) in HCV infected patients (non-respondents) and in NASH patients suggested that the treatment was well-tolerated and produced immunologic effects. In addition, increasing appreciation for the gut-liver cross-talk and of its role in the initiation of NASH-associated inflammation and fibrogenesis led to the understanding that systemic inflammatory processes can be alleviated by modulating the gut immune system, without inducing generalized immunosuppression. This has been achieved in many approaches, including oral administration of fatty liver-derived proteins, anti-CD3 antibodies, TNF, fusion protein, anti-lipopolysaccharide antibodies, glucosylceramide, delayed-release mercaptopurine, and soy-derived extracts. Many of these compounds were shown to be effective in patients with NASH.

Orally administered OKT3 was evaluated in a Phase 2 trial (n=36) with NASH and T2D and was found to be well tolerated. Increases in regulatory T-cell markers consistent with induction of regulatory T-cells was observed as well as increases in other anti-inflammatory markers. Although not powered sufficiently to evaluate efficacy endpoints, positive trends were observed including lowering of liver enzymes and lowering of glucose levels (J. Clin. Immunol., 2015). However, OKT3 was withdrawn from the market due to severe side effects being a murine mAb.

Francois Brisebois
fbrisebois@laidlawltd.com

Figure 5: Clinical Trial Design

Phase 2a OKT3 Mab in Patients with NASH and T2 Diabetes	
Aim	Evaluate safety/efficacy of oral administration of Anti-CD3 mAb in patients with metabolic syndrome
Design	randomized, single-blinded, 9/group, not powered for stat sig. follow up: days 0, 14, 30, 60. At Hadassah Medical Center, Jerusalem Israel
Dosing	0.2, 1.0, 5.0 mg or placebo daily for 30 days
Endpoints	1) safety and trends in immunomodulation. 2) indication or trend of efficacy through biomarkers
Patients	n=36
Safety	no treatment related adverse events, well tolerated, no change in CD3+ ymphocyte count, normal blood chemistry and blood cell counts
Results	Immunological: increase in T reg markers consistent with induction of Tregs, anti-inflammatory markers increase, CD4+CD25+LAP+ Treg cells, TGFβ increase. Efficacy Biomarkers: positive trends, some of which were stat sig, AST decrease liver enzyme indicating reduced liver inflammation, glucose decrease favorable for subjects with T2 diabetes, insuline decrease favorable for subjects with T2 diabetes.

Source: Company reports and Laidlaw estimates

Although still early, the following Figure depicts TLSA's plans for their Phase 2 Foralumab trial in NASH patients. While the primary endpoints will be mostly geared around safety/tolerability, efficacy/immunomodulatory activity will also be established using certain criteria.

Figure 6: Clinical Trial Design

Phase 2 Foralumab in NASH	
Aim	Evaluate safety and tolerability of Foralumab in NASH patients
Design	randomized, placebo-controlled, 4-arm, double-blind study. 1:1:1:1. follow-up of 30 days post-completion of treatment. Study visits on days 14, 30 and 60.
Dosing	once daily oral placebo or Foralumab dose of 0.5mg, 2.5mg or 5.0mg for 30 consecutive days
Endpoints	monitor metabolic parameters (BMI and waist circumference), serum lipid profiles, immunological markers (C-reactive protein and array of cytokines), hepatic enzymes and functions (13C-methacetin breath test and liver steatosis/fibrosis). 1) safety and tolerability of treatment regimen and determined by monitoring vital signs, laboratory values, AEs and physical findings. efficacy/immunomodulatory activity will be established using 1) reduced day 30 ALT levels; 2) reduced hemoglobin A1c; 3) improved homeostasis model assessment (HOMA); 4) HOMA of insulin-resistance (HOMA-IR) as well as 5) levels of T-cells and cytokines
Patients	n=48
Safety	many of the primary endpoints are focused on safety and tolerability
Results	waiting on guidance

Source: Company reports and Laidlaw estimates

TLSA plans to begin to evaluate Foralumab in their newly developed oral formulation for patients with NASH and/or CD in a Phase 1 trial. In another Phase 1 trial, they plan to evaluate Foralumab via intranasal administration in patients with MS.

Francois Brisebois
fbrisebois@laidlawltd.com

Figure 7: Clinical Trial Design

Phase 1 Foralumab in Healthy Volunteers	
Aim	Evaluate safety and tolerability of Foralumab in healthy volunteers
Design	Single Center Single arm ascending study
Dosing	low doses (1.0, 2.5 and 5.0 mg/dose) of Foralumab orally admistered for 5 consecutive days followed by monitoring for 10 days. At each dose, if resultant data indicates drug is well tolerated and free from significant Aes, next higher dose will be administered orally further for 5 consecutive days followed by 10 days of monitoring for tolerance and AEs.
Endpoints	1) safety and tolerability of Foralumab in humans
Patients	
Safety	
Results	If P1 well tolerated, p2a expected to be initiated in 2019 with intent to treat patients with NASH and/or CD

Phase 1 Foralumab for MS	
Aim	Evaluate safety and tolerability of Foralumab in patients with MS
Design	single center single arm ascending study
Dosing	nasally administered low doses (10, 50 and 250 ug/dose) of administered for 5 consecutive days followed by monitoring for tolerance and adverse events for 10 days. If low dose well tolerated, move on to next higher dose for 5 consecutive days followed by 10 days of monitoring
Endpoints	1) safety and tolerability 2)evaluation of unique biomarkers of immunomodulation, induction of T regs and anti-inflammation
Patients	
Safety	
Results	waiting on guidance

Source: Company reports and Laidlaw estimates

Two of Novimmune's clinical trials were in patients with CD and the third clinical trial was conducted in patients undergoing kidney transplantation and suffering with renal allograft rejection. Sixty-eight subjects with active CD and 11 subjects with acute cellular renal allograft rejection were treated with Foralumab (IV).

Figure 8: Clinical Trial Design

Study NI-0401-01	
Aim	Evaluate safety and tolerability of Foralumab in moderate to severe CD
Design	phase 1/2a randomized, double-blind, placebo-controlled and dose escalation study in patients with moderate to severe active CD.
Dosing	Single and repeat IV doses of 1.0, 2.0 and 10.0mg Foralumab we re administered and serum PK evaluated for up to 5 days
Endpoints	designed for tolerability and not powered for efficacy (proportion of patients achieving, clinical response and change from baseline of CD Endoscopy Index of Severity) .
Patients	n=33
Safety	main adverse events were infusion related reactions to limited release of cytokines at doses > 1mg.
Results	Trend (NS). Limited PK data but at doses > 1.0mg, Foralumab accumulated over the 5 day dosing period. CD3 modulation on CD4+ and CD8+ T cells was related to Foralumab dose. Dose response for reduction of peripheral T-cell (CD2+) count.

Source: Company reports and Laidlaw estimates

Francois Brisebois
fbrisebois@laidlawltd.com

Figure 9: Clinical Trial Design

Study NI-0401-02	
Aim	Evaluate safety and tolerability of Foralumab in subjects with cellular renal allograft rejection
Design	open-label dose titration, multicenter Phase 1 for treatment of subjects with biopsy-proven acute cellular renal allograft rejection (BpACR).
Dosing	1.0mg, 1.5mg, 2.0mg and 2.5mg of Foralumab daily for 5 days and most were pre-treated with methylprednisolone.
Endpoints	Nature, frequency, intensity, causality and seriousness of adverse events (1-6 weeks)
Patients	n=11
Safety	main Aes were infusion related reaction in patients that were not premedication with prednisolone.
Results	confirme dose response in terms of CD3 modulation and reduction of peripheral T-cell count. A CD3 modulation of up to 90% was achieved at day 5 with a daily dose of 2.5mg during 5 days of treatment. Conclusions about effectiveness in reversing acute renal rejection could not be drawn.

Source: Company reports and Laidlaw estimates

Figure 10: Clinical Trial Design

Study NI-0401-03	
Aim	Evaluate safety and efficacy of Foralumab in patients with morerate to severe CD.
Design	Phase 2a, open-label dose escalation followed by a double-blind phase for safety and effocacy patients with moderate to severe active CD.
Dosing	
Endpoints	
Patients	n=24
Safety	
Results	74% achieved clinical response at week2 and 87% at week 4. At weeks 6,8 and 12, proportion of patients with a clinical response decreased to 75%, 70% and 67%, respectively. 30% of patients had achieved clinical remission at week 2, 42% by week 4, 38% by week 6, 43% at week 8 and 46% week 12.Treatment failures were 12.5%. no dose-response due to variability between patients. Half-life was about 180 hours. rapid and almost complete disapearance of CD45+ lymphocytes, CD+3+ T-cells, CD3+ and CD4+ helper T-cells and CD3+ and CD8+ cytotoxic T-cells from circulation within 24 hours of infusion of all dose cohorts. Pre-medication with prednisolone reduced severerity and frequency of infusion related reactions.

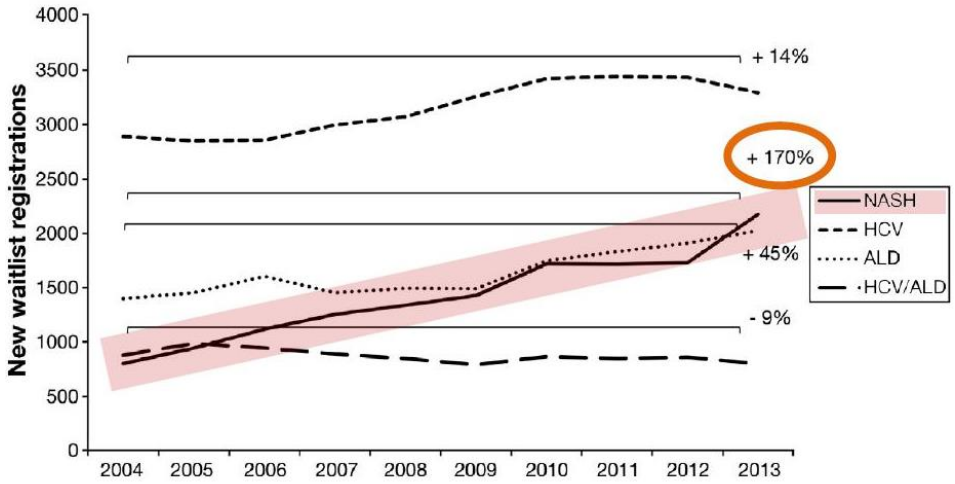
Source: Company reports and Laidlaw estimates

NASH

Non-alcoholic fatty liver disease (NAFLD), comprises a spectrum of progressive liver diseases, which currently affects ~1/3 of the western world. It is associated with liver-related morbidity and mortality and with increased risk of CVD, T2DM, hyperlipidemia, and abdominal obesity. NASH, one of the manifestations of NAFLD, leads to inflammation, fat and fibrous tissue buildup in the liver as well as elevated liver enzymes levels, and can lead to liver cirrhosis, end-stage liver disease, and primary liver cancer (HCC). NASH is predicted to become the leading cause of liver transplantation in the US by 2020. Both genetic predisposition and environmental factors have been implicated in its onset, and inflammation and associated fibrogenesis contribute to its progression. Chronic inflammatory processes involve an imbalance in pro versus anti-inflammatory cytokines, altered insulin responses due to inflammation and fat and fibrous tissue buildup.

Francois Brisebois
fbrisebois@laidlawltd.com

Figure 11: NASH as one of the leading causes of liver disease among adults awaiting liver transplant in US



Source: Gastroenterology, 2015

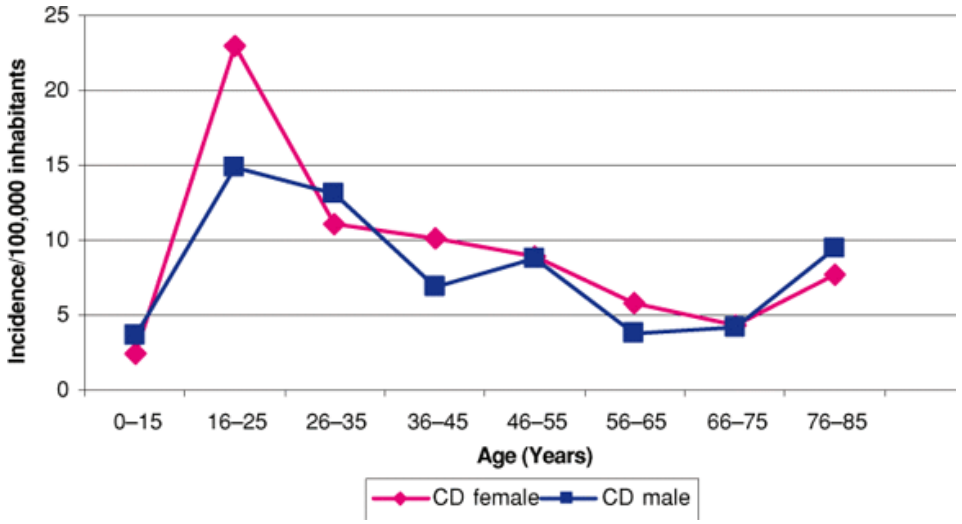
Therapeutics provide multifactorial effects, with focus on reduced insulin resistance and inflammatory processes. Mild inflammation imparts a hepatoprotective effect, while excessive inflammation triggers hepatocyte damage and irreversible liver damage, fibrosis, and carcinogenesis. Such detrimental effects are associated with overexpression of inflammatory genes and increased activity of Kupffer cells, natural killer T-cells, hepatic stellate cells, sinusoidal endothelial cells, dendritic cells, monocytes and lymphocytes, which secrete a range of proinflammatory factors, including cytokines, chemokines, lipid messengers and reactive oxygen species (ROS). We do believe NASH treatment will consists of combination therapy as the disease is better understood.

Crohn's Disease

CD is a relapsing, transmural inflammatory disease of the GI mucosa that can affect all parts of the intestinal tract as well as extra-intestinal organs. CD has a prevalence of 201/100,000 adults and an incidence rate of 3.1-14.6/100,000 persons/year, thus affecting ~500,000 people in the US (CDC, 2018). Differences in incidence across age, time, and geographic region suggest that environmental factors (cigarette smoking, appendectomy) significantly modify the expression of CD. The disease affects slightly more females than males and is most commonly diagnosed in young adults to the third decade of life (Gastroenterology, 2004).

Francois Brisebois
fbrisebois@laidlawltd.com

Figure 12: Crohn's Disease Incidence by gender



Source: Am J Gastroenterol, 2006

Although the exact etiology remains unknown, the occurrence of CD is strongly associated with mutations of a receptor for microbial pathogens (NOD2) that lead to increased activation of antigen presenting cells and a defect in the release of antimicrobial defensins. It is now widely accepted that as a result of this altered balance of immune homeostasis, exposure to commensal bacterial antigens causes increased stimulation and proliferation of mucosal T-lymphocytes, leading to immune inflammation. Additional pathogenic mechanisms may include a defect in T-cell programmed death (apoptosis) and possibly a defect in regulatory T-cell function. CD usually presents as acute or chronic bowel inflammation then the inflammatory process evolves toward one of two patterns of the disease 1) a fibrostenotic-obstructing pattern or 2) a penetrating-fistulous pattern, each with different treatments and prognoses. The site of disease influences the clinical manifestations and can include diarrhea, abdominal pain, fever, clinical signs of bowel obstruction, as well as passage of blood or mucus or both.

The pharmacological management of CD is based on the control of the inflammatory process. Current treatment regimens include anti-inflammatory drugs such as corticosteroids and aminosalicylates (1st-line treatment) to induce remission in acute active disease. Other include immunosuppressants such as azathioprine, 6-mercaptopurine, and methotrexate which are used to maintain remission or treating chronic active disease. Biologic immunotherapies (anti-TNF mAbs including infliximab and adalimumab) are also sometimes used to induce and maintain remission (Feagan, 2000). All of these treatments have limited long-term efficacy and potential for SAEs (Targan, 1997; Present, 1999). Previously reported studies using anti-CD4, and TNF, binding mAbs provide a strong rationale for targeting T-cells in CD (Intl. J. Clin. Pharm, 1997). It is now known that TNF targeting mAbs in CD and IBD are effective because of the bringing about of programmed cell death (apoptosis) of activated T-lymphocytes rather

Francois Brisebois
fbrisebois@laidlawltd.com

than neutralization of soluble TNF (Current Drug Targets, 2010; Gastroenterology, 2011).

Additionally, there are few published clinical data on the use of anti-CD3 mAbs in subjects with CD. One product in development, visilizumab (Nuvion) of PDL Biopharma (PDLI) has been tested in the clinical setting. Two studies with visilizumab in patients with severe CD have been performed: an open-label study in patients with CD having peri-anal fistulas and an open-label study in patients with moderate-to-severe inflammatory, non-structuring, non-penetrating CD. Eighteen patients were expected to be enrolled in each study. Preliminary results from the 2nd study suggested that two 10 μg/kg doses of visilizumab administered by IV bolus injection on consecutive days appeared to have clinical activity. Ten of the 14 patients reported a clinical response by day 59, as determined by a drop in the CD Activity Index, (CDAI) score, of 100 points. Five patients achieved a complete remission, as defined by a CDAI score of <150 during the 59 days. Of note, two patients who never responded to infliximab, as well as seven patients who lost their response to infliximab, responded to visilizumab.

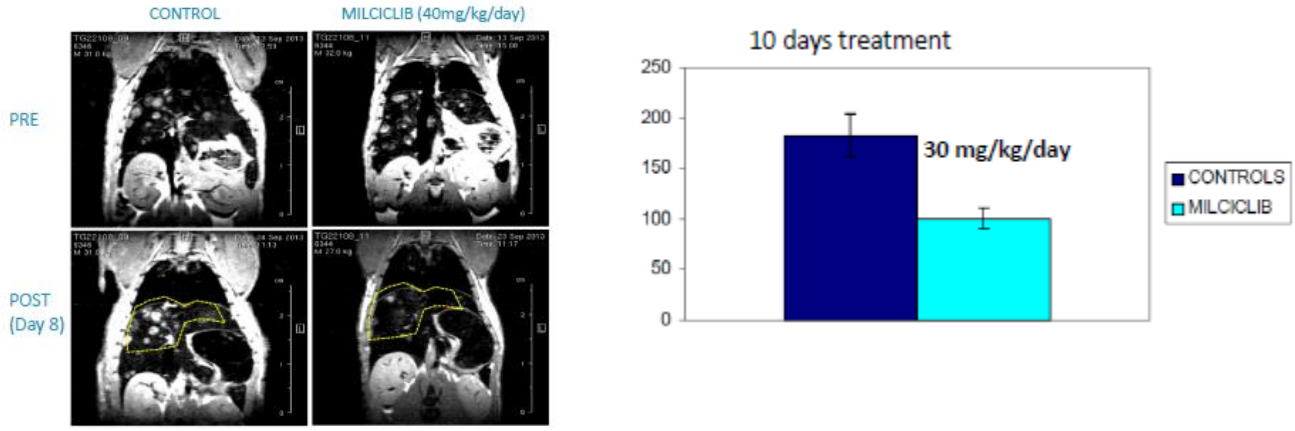
Multiple Sclerosis

MS is an inflammatory-mediated demyelinating disease of the human CNS. The disease develops in young adults with a complex predisposing genetic trait and most likely involves an environmental insult such as a viral infection to trigger the disease. The activation of CD4+ autoreactive T-cells and their differentiation are crucial initial steps in the progression of this disease. The therapeutic use of mAbs was first viewed with great skepticism owing to the high rates of sensitization against mouse proteins, their PK properties, and the difficulties in their production. However, most of these problems have been overcome, and mAbs are now among the most promising therapies for MS.

Milciclib for Hepatocellular Carcinoma (HCC)

TLSA is developing Milciclib (in-licensed from Nerviano Medical Sciences in January 2015) as a potential treatment for HCC. A novel feature of Milciclib is its ability to reduce levels of microRNAs, miR-221, and miR-222. MicroRNAs are small RNA molecules that play an important role in the regulation of gene expression. miR-221 and miR-222 are believed to be linked to the development of blood supply in cancer tumors. Levels of these microRNAs are elevated in HCC patients and may contribute towards resistance to treatment with Sorafenib (a multikinase inhibitor for HCC patients). Milciclib has been studied in a total of seven completed and ongoing Phase 1 and Phase 2 clinical trials in a total n=296 conducted by Nerviano. In these trials, Milciclib was well-tolerated with minimal AEs. They initiated a Phase 2a trial for Milciclib as a monotherapy in patients with HCC in the 4Q17 and expect to initiate a Phase 2b trial for Milciclib in combination with Sorafenib in patients with HCC in the 1H19.

Figure 13: Milciclib inhibits MIR221/222 to suppress HCC tumor growth in mice – reduction in # and volume of lesions



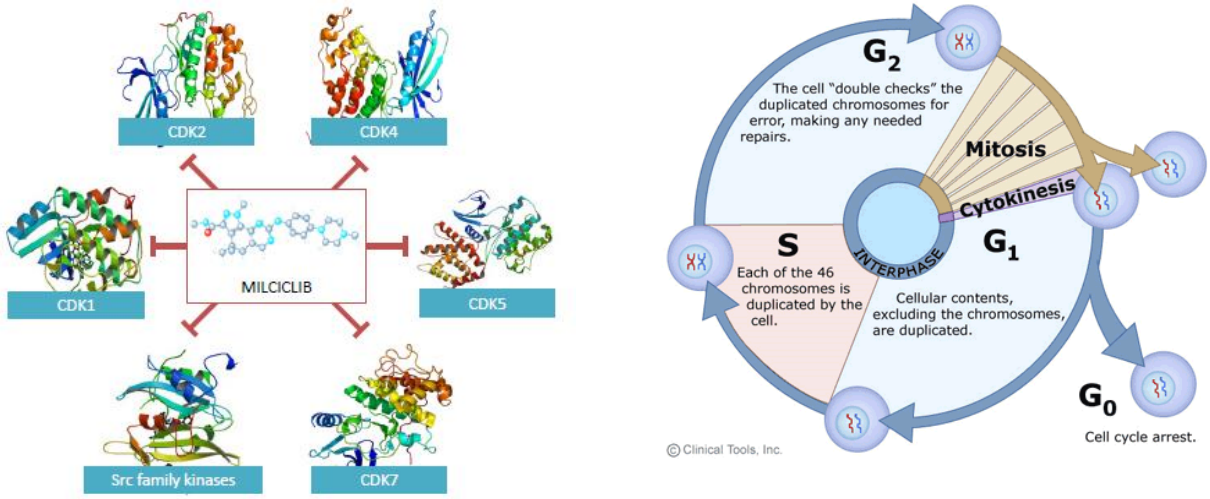
Source: Company Reports

Milciclib is an orally bioavailable, small molecule broad spectrum inhibitor of CDKs (CDKs): 1, 2, 4, 5 and 7 and Src family kinases. CDKs are a family of highly conserved enzymes that are involved in regulating the cell cycle, which is a series of events that takes place in cells leading to division and duplication of its DNA to produce two daughter cells. Src family kinases regulate cell growth and potential transformation of normal cells to cancer cells. As mentioned previously, a novel feature of Milciclib is its ability to reduce microRNAs, miR-221 and miR-222, that silence gene expression. In tumor cells exposed to

Francois Brisebois
fbrisebois@laidlawltd.com

Milciclib, a block in G1 phase (first phase of the growth cycle where the cell synthesizes messenger RNA and proteins before cell division) of the cell cycle was observed, supporting the hypothesized MOA of the compound as determined in biochemical assays. Additionally, Milciclib was able to modulate the phosphorylation of the Retinoblastoma protein, a substrate of the CDK/cyclin complex as well as to reduce phosphorylation status of proteins of the TRKa signaling pathway in cells expressing the tyrosine kinase receptor. These results supported that Milciclib was active against several families of protein kinases that actively controlled cell growth and transformation from normal to cancerous cell types. This is important because many chemotherapeutic agents are effective at only a single point in the cell cycle, allowing cells to not be blocked. Significant anti-tumor activity was observed in all tested preclinical animal models with different oral treatment schedules of Milciclib. The first FDA approval in March 2015 of a CDK inhibitor for palbociclib by Pfizer (PFE), and more recently in 2017, ribociclibby Novartis (NVS), for a type of breast cancer, has led to great interest in the development of this class of drugs as oncology therapeutics.

Figure 14: Milciclib MOA



Source: University of Leicester

In January 2017, TLSA initiated a single-arm, multicenter, Phase 2a clinical trial in adults with unresectable or metastatic HCC and good liver function to test for tolerability and safety of Milciclib in adult patients.

Francois Brisebois
fbrisebois@laidlawltd.com

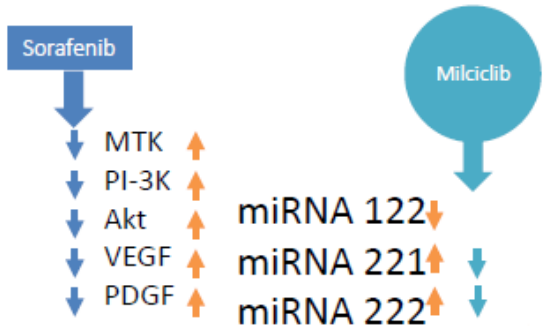
Figure 15: Clinical Trial Design

Phase 2a Milciclib as a monotherapy for treatment of HCC	
Aim	Evaluate safety and tolerability of Milciclib for HCC
Design	multicenter, phase 2a, in adult patients with unresectable or metastic HCC and good liver function. Sites in Italy, Greece and
Dosing	orally, 100mg/day for 4 consecutive days/week followed by 3 days off for total of 12 weeks. Dose reduced to 80mg/day on subsequent cycles if more than 2 of the first 6 treated patients exhibit poor tolerability
Endpoints	1) overall tolerability profile, based on laboratory findings and Aes. 2) Objective Tumor Response Rate, based on modified response evaluation criteria in solid tumors (mRECIST). Decrease in alpha-fetoprotein (AFP) vs baseline in patients with high AFP at baseline will also be considered. As exploratory endpoint, expression of miRNAs and their possible association to Milciclib will be investigated.
Patients	n=10 but additional enrollment allowed after positive safety evaluation of first 10 patients by IDMC. Study actively enrolling 20 more patients
Safety	well-tolerated with no drug-related SAEs.
Results	interim evaluation of tolerability and Aes was undertaken as soon as 10th patient completed first cycle. On 12/8/17, announced treatment was well-tolerated with no drug-related SAEs. Second IDMC meeting on 5/9/18 for all 11 patients and IDMC concluded safety and tolerability in first cohort is acceptable. 4 patients completed completed protocol mandated treatment (6 cycles, 6 months). 3 of these patients ad health care-provider/investigator opted to continue by compassionate use.

Source: Company reports and Laidlaw estimates

Sorafenib (Nexavar) is the standard of care for treatment of HCC, yet treatment extends survival probability from 7.9 months (placebo control) to 10.7 months (N Engl J Med, 2008). There is a need for improvement which may be realized by combination of Milciclib with Sorafenib. Sorafenib is a multikinase inhibitor which has demonstrated both anti-proliferative and anti-angiogenic properties in vitro and in vivo. Sorafenib+Milciclib could exert a combined anti-proliferative effect on tumor cells, involving targets different from the ones modulated by Milciclib, together with antiangiogenesis properties.

Figure 16: Synergistic effect on HCC with Sorafenib expected



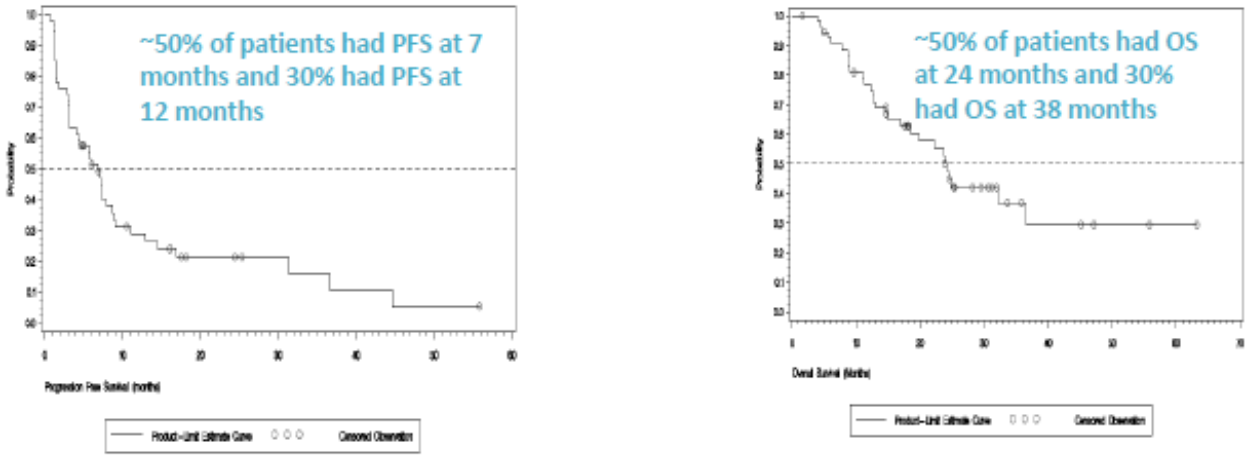
Source: Company reports

By 1H19, TLSA intends to initiate a Phase 2b randomized, multicenter study to explore tolerability and antitumor activity of Milciclib in combination with Sorafenib, administered as 1st-line systemic therapy in adults with recurrent, unresectable or metastatic HCC and good liver function.

Francois Brisebois
fbrisebois@laidlawltd.com

Thymomas and thymic carcinomas originate in epithelial cells of the thymus gland. Thymoma usually does not spread beyond the thymus, while thymic carcinoma, represents an aggressive cancer that metastasizes rapidly and poses treatment challenges. Both cancers are rare, and it is estimated that together they account for ~400 cases per year in the US (~1.5 persons per million diagnosed with thymoma/thymic carcinoma). Patients more often present with advanced disease, with a 5-year survival of 30%-50%. Standard primary treatment for patients with these types of tumors is surgical resection. Depending on tumor stage, treatment options include the use of radiation therapy and chemotherapy with or without surgery. First line of chemotherapy treatment is the combination of cisplatin, doxorubicin, and cyclophosphamide for thymoma. For thymic carcinoma the first line of treatment is the combination of paclitaxel and carboplatin. Milciclib met its primary endpoints in two Phase 2 clinical trials in patients with thymic carcinoma and thymoma.

Figure 17: CDkO-125a-006 Demonstrated Milciclib's clinical activity in Thymic Carcinoma and Thymoma mixed population



Source: Company Reports

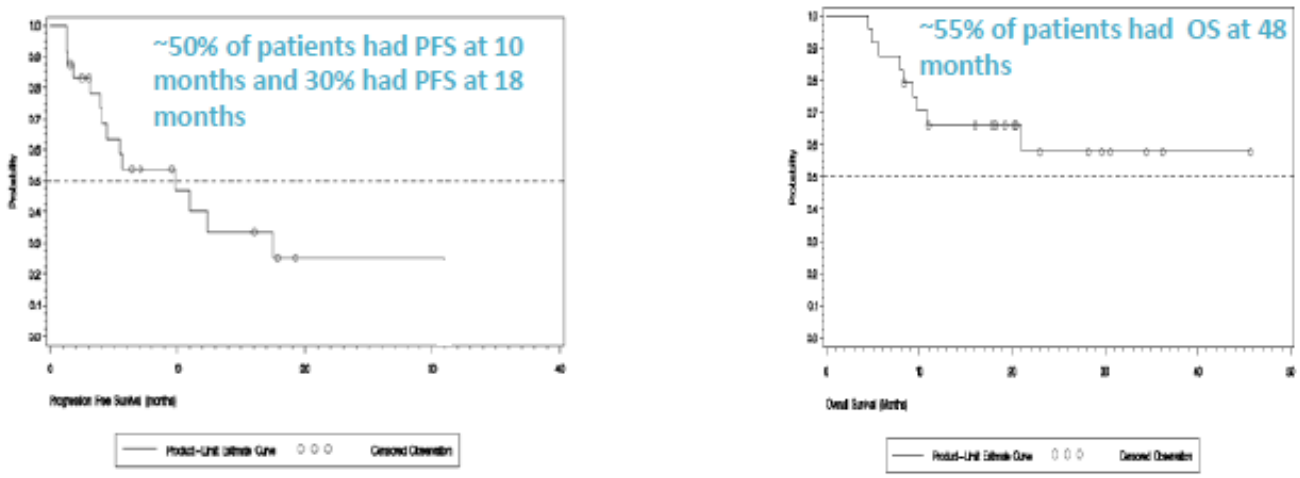
Figure 18: Clinical Trial Design

Phase 2 Milciclib CDKO-125A-006 in thymoma/thymic carcinoma	
Aim	Evaluate safety/efficacy of Milciclib in thymoma/thymic carcinoma
Design	Conducted in US, France and Italy
Dosing	monotherapy 150mg/day; 7 days on /7 days off
Endpoints	1)PFS 2) OS
Patients	n=72
Safety	well tolerated. N=5 continuing treatment for > 2 years with excellent tolerance profile
Results	1): n=56/72 treated had median PFS of 5.78 months with upper and lower 95% confidence limits of 3.48 months and X, respectively. Exceeded median PFS > 10.2 weeks established for monotherapy with permetrexed; 2) N=36/72 treated patients had median OS of 24.44 months with upper and lower 95% confidence limits of 22.05 and 53.55 months, respectively

Source: Company reports and Laidlaw Estimates

Francois Brisebois
fbrisebois@laidlawltd.com

Figure 19: CDkO-125a-007 Demonstrated Milciclib's clinical activity in Thymic Carcinoma and Thymoma mixed population



Source: Company Reports

Figure 20: Clinical Trial Design

Phase 2 Milciclib CDKO-125A-007 in thymoma/thymic carcinoma	
Aim	Evaluate safety/efficacy in thymoma/thymic carcinoma
Design	Conducted in US, France and Italy
Dosing	monotherapy 150mg/day; 7 days on /7 days off
Endpoints	1) PFS 2) OS
Patients	n=30
Safety	well tolerated. N=2 continuing treatment for > 2 years with excellent tolerance profile
Results	1): n=18/30 treated patients had median PFS of 5.65 months with upper and lower 95% confidence limits of 3.94 and 17.45 months, respectively. Exceeded median PFS > 10.2 weeks established for monotherapy with pemetrexed. 2) N=18/30 patients had an OS of 48 months. As a median was not reached, 95% confidence limits could not be calculated

Source: Company reports and Laidlaw estimates

Milciclib has been investigated in multiple Phase 1 open-label, multi-center, non-randomized, dose-escalation clinical trials and following are some of the main safety conclusions.

Francois Brisebois
fbrisebois@laidlawltd.com

Figure 21: Key Safety Conclusions from 5 Phase 1 trials

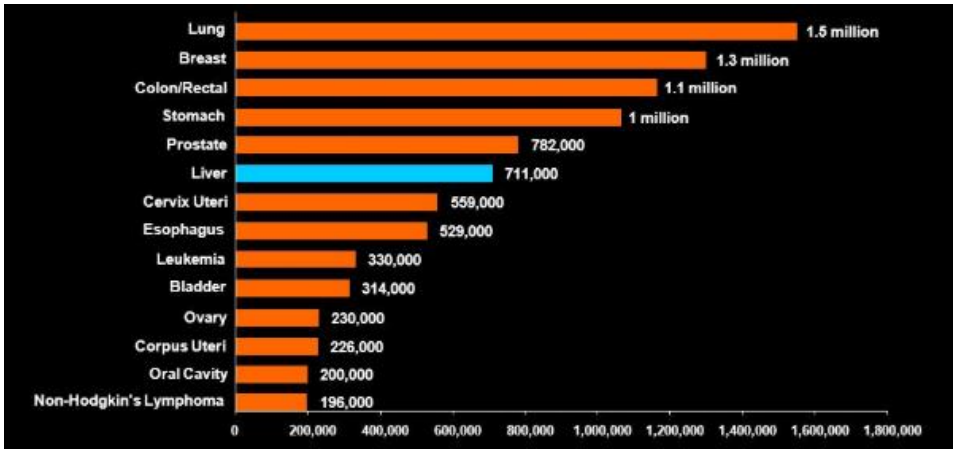
Phase 1 Safety Conlcusion
Overall similar pattern of toxicity across studies
Consistent with pre-clinical findings: safety profile of compounds in humans characterized by dose-limiting neurological toxicity (lesser extent: GI toxicity)
Weakness, fatigue and liver effects
Mild/moderate tremorsand ataxia generally reversible in up to 7-9 days, upon drug discontinuation or dose reduction
Grade 1, 2 dozziness also reported with only 1 Grade 3 occurrence
Mild dysgeusia and Diarrhea occasionally severe
Severe thrombocytopenia sporadically observed (especially at highest dose)
Liver effects dose dependent represented by transaminase elevation
1 case of retinal detachment reported as SAE

Source: Company Reports and Laidlaw estimates

Hepatocellular Cancer

As mentioned previously, TLSA is initially developing Milciclib for the treatment of HCC, which is the 6th most common cancer WW and 2nd most leading cause of death in the US. Liver cancer incidence and death rates are steadily rising. As of 2012, rates of new liver cancer cases went up 38% from 2003 to 2012 (CDC, 2012). Most HCC patients present with advanced disease and do not benefit from transplantation, surgical resection, or locoregional therapies, and there is only one chemotherapeutic agent, Sorafenib by Onyx (ONXX) approved in the US and EU for advanced HCC patients.

Figure 22: Most common cancers worldwide



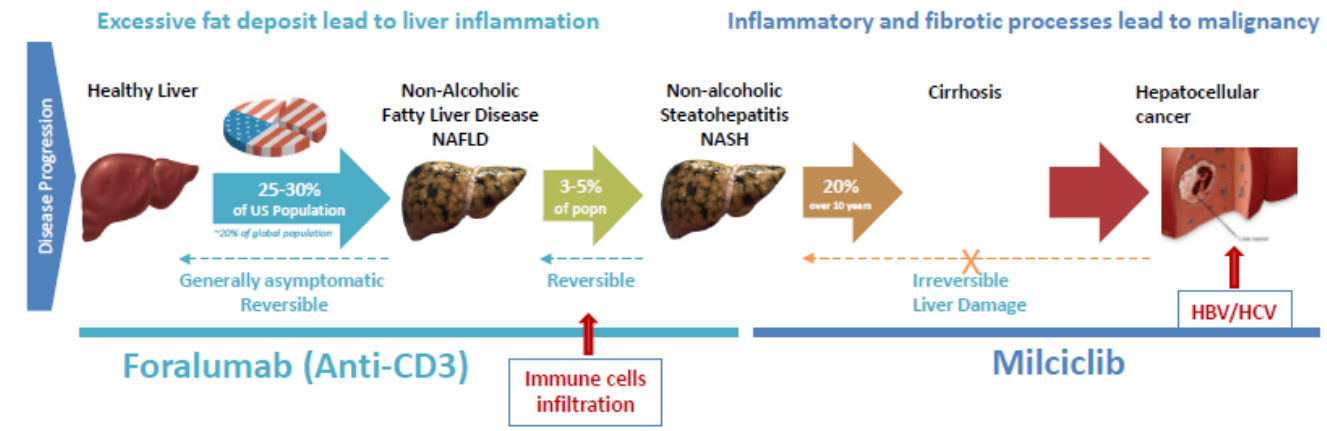
Source: American Cancer Society, 2007

Recently, the combination of insulin resistance, hypertension, dyslipidemia, and obesity (metabolic syndrome) has also been recognized as a cause of NAFLD.

Francois Brisebois
fbrisebois@laidlawltd.com

The following graphic represents the progression from a healthy liver to NAFLD, NASH and HCC.

Figure 23: Liver Disease progression from Healthy to Hepatocellular cancer



Source: Company Reports

Fully Human mAb targeting IL-6R for Autoimmune Diseases

Additionally, TLSA is developing a fully human mAb targeting the IL-6R (TZLS-501). They licensed the IP from Novimmune in January 2017. This fully human mAb has a novel MOA, binding to both the membrane-bound and soluble forms of the IL-6R and depleting circulating levels of the IL-6 in the blood. Excessive production of IL-6 is regarded as a key driver of chronic inflammation, associated with autoimmune diseases such as multiple myeloma, oncology indications and RA, and they believe that TZLS-501 may have potential therapeutic value for these indications.

Figure 24: TZLS-501 Product Description

Product	TZLS-501: Fully human anti-IL-6R mAb
MOA	IL-6 is potent cytokine regulating cell growth, differentiation and immune responses; Excessiv production of IL-6 and its receptor IL-6R are key drivers of chronic inflammation and inflammatory disease
Indications	Multiple myeloma, Maybe in combination with Foralumab for NASH and other inflammatory diseases like RA
Opportunity	Potential synergistic effect with Foralumab for inflammatory diseases
Competitive Edge	Acts not only on membrane-bound IL-6R, but also on soluble IL-6R, and able to deplete circulating levels of IL-6 in blood
IP	exclusive license from Novimmune, MOU in combo with anti-CD3 patent pending

Source: Company Reports and Laidlaw estimates

Francois Brisebois
fbrisebois@laidlawltd.com

In preclinical studies, TZLS-501 demonstrated the potential for overcoming the limitations of other IL-6 pathway drugs. Compared to Roche's tocilizumab and sarilumab by Regeneron (REGN), TZLS-501 has been observed to have a higher affinity for the soluble IL-6 receptor from antibody binding studies conducted in cell culture. TZLS-501 also demonstrated the potential to block or reduce IL-6 signaling in mouse models of inflammation. The soluble form of IL-6 has been implicated to have a larger role in disease progression compared to the receptor bound form (Biochimica et Biophysica Acta, 2002).

Major Risks

Exogenous events could impact our outlook. We believe pharmaceutical companies have the least control over competitive, political, and regulatory risks. Although we have incorporated competitive assumptions into our forecasts, there may be other risks beyond the scope of our analysis. Changes in the drug reimbursement system, as well as any political or regulatory amendments, may significantly influence the earnings power of these companies.

Actual clinical results and the FDA's conclusions may deviate from expectations. Many of our assumptions are based on a review of incomplete clinical trial data available in the public domain. Often, our conclusions are drawn from early stage data, which may not be reflected by pivotal studies. Furthermore, the FDA's conclusions may not coincide with our own, materially changing our revenue and earnings assumptions.

Compliance issues, product recalls, and other mandates by regulatory authorities could materially change our expectations. Regulatory compliance issues, ranging from accounting irregularities to defective manufacturing practices, could materially change our assumptions and earnings outlook. Unanticipated product recalls and labeling changes could also have adverse consequences on our earnings assumptions.

Legal risks could lead to additional liabilities and revenue loss. In addition to the expenses incurred by patent challenges, product liability and other legal suits could occur and lead to additional liabilities and revenue loss, which could substantially change our financial assumptions.

Francois Brisebois
fbrisebois@laidlawltd.com

Management

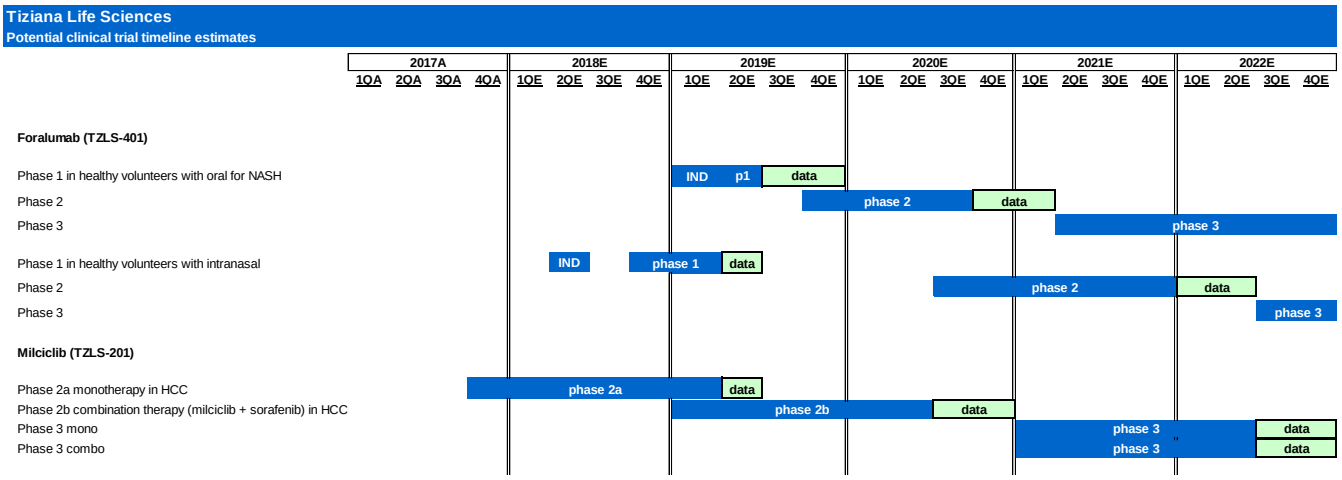
Kunwar Shalubhai, PhD, CEO and CSO. Dr. Shailubhai brings more than 25 years of experience within the life science industry, combined with a distinguished track record of success in translating drugs from concept through commercialization to market. He also currently serves as CEO of Rasna Therapeutics, a developer of therapeutics to address the high unmet need that exists for AML and other forms of leukemia. Dr. Shailubhai has been serving as a member of board of Tiziana Life Sciences since 2015. He actively played key roles in development of growth strategies through several key licensings of technologies and drug candidates. He steered the company through prioritization of projects to focus on novel drug candidates for treatment of autoimmune and inflammatory diseases and cancer. As co-founder, EVP and CSO of Synergy Pharmaceuticals (SGYP) he led the non-clinical, CMC and clinical development of Trulance from inception to approval by the FDA, having co-invented and pioneered Synergy's platform technology for functional GI disorders, inflammatory bowel disease, GI cancer and other human diseases. Dr. Shailubhai as the chief architect of the IP estate, directed all aspects of IP management, including timely submission of patent applications, directing office actions and coordinating with IP attorneys. Dr. Shailubhai received his Ph.D. in microbiology from the University of Baroda, India, and his MBA from the University of Missouri, St. Louis.

Tiziano Lazzaretti, CFO. Mr. Lazzaretti has extensive experience in the healthcare and pharmaceutical industry and joined Tiziana from Pharmentis, a spin-off from Teva Ratiopharm, where he served as Group Finance Director from 2011. Prior to this, Mr Lazzaretti was Executive Director at Alliance Boots Healthcare, and held senior positions at Accenture, SNIA Spa and Fiat Group. Mr Lazzaretti has a Bachelor of Science in Accounting and Finance from the University of Turin, Italy, was awarded an MBA from Bocconi University, Milan and studied Corporate Finance at the London Business School.

Fayez M. Hamzeh, SVP Clinical Development. Before joining Tiziana Life Sciences, Dr. Fayez Hamzeh served as Head of Clinical Trials Planning and Implementation Strategy of Immunology and Infectious Diseases at Roche Innovation Center, New York from 2015 to 2017. During this period Dr. Hamzeh's focus was Phase I and Phase II clinical trials in immunology, hepatology and infectious diseases. Dr. Hamzeh served as a Group Medical Director and Head of Infectious Disease, Transplantation and Hepatology at Genentech, South San Francisco from 2009 through 2011 and 2013 through 2015. Dr. Hamzeh served as a Group Medical Director in Genentech Medical Affairs and was responsible for glioblastoma and avastin pan tumor indications. Dr. Hamzeh served as Sr. Medical Director of Hepatology, Transplantation and Infectious Diseases at Roche Pharmaceutical, Nutley NJ from 2003 through 2009. During this period, Dr. Hamzeh was responsible for clinical trials in virology focusing on anti-HIV, anti-influenza, anti-cytomegalovirus, anti-HCV and anti-HBV drugs. Prior to joining the industry in 2003, Dr. Hamzeh served as an Assistant professor of Medicine and Pharmacology and Molecular Therapeutics at Johns Hopkins School of Medicine, Baltimore MD. During his academic and industry career, Dr. Hamzeh served on many NIHealth scientific steering committees including, AIDS Clinical Trials Group, AIDS Malignancy Consortium, Hepatitis-C (HALT-C) Scientific Steering Committee. Dr. Hamzeh received his MD degree in 1981 from the University of Jordan Medical School and his PhD from Johns Hopkins School of Medicine in 1990.

Francois Brisebois
fbrisebois@laidlawltd.com

Figure 25: Potential Clinical Trials Timeline



Source: Company Reports; Laidlaw & Company estimates

Figure 26: Quarterly Income Statement

Tiziana Life Sciences
Quarterly income statement

($000 except per share)	2017A Year	2018E 1QA	2018E 2QE	2018E 3QE	2018E 4QE	2018E Year
Revenues						
Total Revenue						
Expenses:						
COGS (% of US Revenue)						
Gross Margin						
G&A	(4,601)	(1,083)	(1,083)	(500)	(500)	(3,166)
R&D	(6,015)	(1,569)	(1,569)	(500)	(500)	(4,138)
Total operating expenses	**(10,616)**	**(2,652)**	**(2,652)**	**(1,000)**	**(1,000)**	**(7,304)**
Operating income						
other income (expense)	(12)					
Loss before income tax	**(10,628)**	**(2,652)**	**(2,652)**	**(1,000)**	**(1,000)**	**(7,304)**
Interest expense						
Provision (benefit) for income tax	1,912					
Net loss	**(8,716)**	**(2,652)**	**(2,652)**	**(1,000)**	**(1,000)**	**(7,304)**
Foreign currency	70					
Adj. NI/(loss)	**(8,646)**	**(2,652)**	**(2,652)**	**(1,000)**	**(1,000)**	**(7,304)**
NI/(loss) as reported	**(8,646)**					
Earning per Share (EPS)	**($0.09)**					
Adj EPS ex-1x & non-cash	**($0.09)**				**($0.07)**	**($0.54)**
Weighted avg. shares (000)	96,067				13,641	13,641
Fully diluted shares (000)	96,067	-	-	-	13,641	13,641

Source: Company Reports; Laidlaw & Company estimates

Francois Brisebois
fbrisebois@laidlawltd.com

Figure 27: Annual Income Statement

Tiziana Life Sciences
Annual income statement

($000's except per share)	2016A	2017A	2018E	2019E	2020E
Revenues					
Total sales	**$0**	**$0**	**$0**	**$0**	**$0**
COGS	0	0	0	0	0
Gross margin	**0**	**0**	**0**	**0**	**0**
R&D	(4,007)	(6,015)	(4,138)	(6,500)	(9,500)
G&A	(5,872)	(4,601)	(3,166)	(5,000)	(7,000)
Adj. Net Income	**(9,120)**	**(8,646)**	**(7,304)**	**(11,500)**	**(16,500)**
NI/(loss) as reported	**(9,770)**				
Adj-EPS ex-non-cash	**($0.11)**	**($0.09)**	**($0.54)**	**($0.77)**	**($0.99)**
EPS as reported	**($0.11)**	**($0.09)**			
Shares out (000)	82,909	96,067	13,641	14,991	16,724
Fully diluted shares (000)	82,909	96,067	13,641	14,991	16,724

Source: Company Reports; Laidlaw & Company estimates

Francois Brisebois
fbrisebois@laidlawltd.com

Figure 28: Balance Sheet Statement

Tiziana Life Sciences
Balance sheet

($000's except per share)	2017A	2018E	2019E	2020E
ASSETS:				
Current assets				
Cash and cash equivalents	$64	$1,302	$8,902	$20,802
trade and other receivables				
other				
Prepayments and other receivables	$2,383			
Total current assets	**2,447**	**1,302**	**8,902**	**20,802**
PP&E	23			
Total Assets	**2,470**	**1,302**	**8,902**	**20,802**
LIABILITIES				
Current liabilities:				
trade and other payables				
accounts payable and accrued exp	4,749	1,164	1,164	1,164
Total current liabilities	**4,749**	**1,164**	**1,164**	**1,164**
Total liabilities	**4,749**	**1,164**	**1,164**	**1,164**
Shareholder's equity				
called up share capital	8,141	10,000	12,000	14,000
share premium	31,284	35,000	40,000	45,000
share based payment reserve	3,213			
shares to be issued reverse (warrant	579			
convertible loan note reserve				
merger relief reserve				
other reserve	(46,171)	(50,000)	(50,000)	(50,000)
translation reserve	(2,207)			
capital reduction reserve	41,292	50,851	62,951	84,351
retained earnings	(38,409)	(45,713)	(57,213)	(73,713)
Total shareholders' equity	**(2,278)**	**138**	**7,738**	**19,638**
Total liabilites & net worth	**2,471**	**1,302**	**8,902**	**20,802**

Source: Company Reports; Laidlaw & Company estimates – Assets/Liabilities may differ due to rounding.

Francois Brisebois
fbrisebois@laidlawltd.com

Figure 29: Cash Flow Statement

Tiziana Life Sciences
Statement of cash flows

($000's except per share)	2017A	2018E	2019E	2020E
Operating Cash Flow				
Net Income/Loss	(10,628)	(7,304)	(11,500)	(16,500)
net increase/(decrease) in operating liabilities	2,304	111	500	500
depreciation	13			
loss on foreign exchange	45			
Cash from operations	**(7,533)**	**(7,193)**	**(11,000)**	**(16,000)**
Investing Activities				
PPE	(1)			
Acquisition of other investments				
Cash from investing	**(1)**			
Financing Activities				
Proceeds from issuance of ordinary shares/commo	1,542	7,267	18,600	27,900
Proceeds from issuance of fixed term loans		1,164		
Cash from financing	**1,542**	**8,431**	**18,600**	**27,900**
Change in cash	**(5,992)**	**1,238**	**7,600**	**11,900**
Cash, start of period	5,802	64	1,302	8,902
Exchange difference	254			
Cash, end of period	**64**	**1,302**	**8,902**	**20,802**

Source: Company Reports; Laidlaw & Company estimates – 2017 Net Income is pre-tax.

Francois Brisebois
fbrisebois@laidlawltd.com

DISCLOSURES:

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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.

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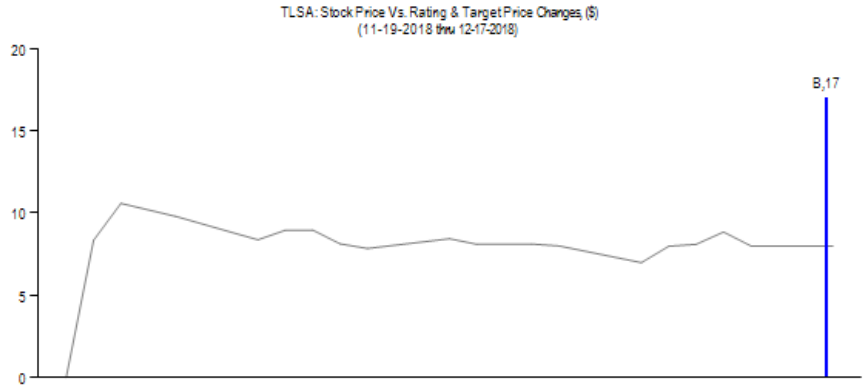
For the purpose of ratings distributions, regulatory rules require the firm to assign ratings to one of three rating categories (i.e. Strong Buy/Buy-Overweight, Hold, or Underweight/Sell) regardless of a firm's own rating categories. Although the firm's ratings of Buy/Overweight, Hold, or Underweight/Sell 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 ($)
12/17/2018	Buy (B)	8.00*

3 Year Price Change History

Date	Target Price ($)	Closing Price, ($)
12/17/2018	17.00	8.00*

** Previous Close12/14/2018*

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.	60.34%	24.14%	3.45%
Hold (H)	Expected returns to be in line with the sector average over 12 months.	6.90%	1.72%	0.00%
Sell (S)	Returns expected to significantly underperform the sector average over 12 months.	0.00%	0.00%	0.00%

ADDITIONAL COMPANIES MENTIONED

MacroGenics (MGNX – Not Rated)
PDL Biopharma (PDLI – Not Rated)
Pfizer (PFE – Not Rated)
Novartis (NVS – Not Rated)
Onyx (ONXX – Not Rated)
Regeneron (REGN – Not Rated)

ADDITIONAL DISCLOSURES

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Francois Brisebois
fbrisebois@laidlawltd.com

NOTES:

Francois Brisebois
fbrisebois@laidlawltd.com