Vectus Biosystems Limited
Chairman's Address to the 25 January 2022 Annual General Meeting

The 2021 year was very productive for Vectus as it continued with progress in its the Phase I human trial of its proprietary **VB0004** that addresses a significant unmet need for anti-fibrotic agents for patients with cardiovascular and/or kidney disease. We are pleased that the toxicology work done to-date gives every indication that there will be a good result from the Phase I trial. Whilst there have challenges recruiting patients for the phase I trial due to COVID, we have made significant progression.

Vectus continues to advance work on its library of over 1,000 compounds, derived from the platform underpinning VB0004. These emerging lead compounds address some of the most significant unmet needs in medicine today and include:

- VB4-A32 (liver fibrosis, including NASH and ASH);
- VB4-A79 (pulmonary fibrosis, including idiopathic fibrosis, asbestosis and coal dust pneumoconiosis (Black Lung Disease)); and
- VB4-P5 (renal tubular cell death consequent on cytotoxic therapy).

As a Radiologist and Clinical Physician, I emphasise the real need for this new class of drugs, providing significant social, patient and health economic outcomes. Fibrosis, or scar tissue, is the end point of a whole host of diseases including high blood pressure, injury, post infections (such as COVID-19), radiotherapy and silicosis. To have potential drugs, like those in the Company's stable, that can not only stop the growth of scar tissue, but also reverse the fibrosis, is a major development in medicine. To take the drug orally, in tablet form, also decreases the cost of production and, more importantly, increases the ease of use by patients. The use of Vectus' compounds to reduce blood pressure is also very significant.

Finance

The $7 million raised in the December 2020 placement of 7.78 million shares at $0.90 per share are being used to fund the human Phase I clinical trials for VB0004, and to fast-track work on the Company's additional compounds towards lead status and human trials, for the commercialisation of the Accugen technology, and for working capital.

It was pleasing to note that the shareholder value has progressed during the last year marked by Vectus' increased market capitalisation. The strong share price has encouraged several convertible note holders to convert at the conversion share price of $0.50.

Commercialisation Process

Vectus continues its dialogue with a cross-section of some of the world's leading pharmaceutical companies and regional mid-sized firms and feedback from these industry leaders remains very positive. The Company's objective is to partner with one or more companies via a licencing programme focusing initially on VB0004 as it completes its current trials. The additional compounds also present an attractive commercial opportunity for Vectus, and clinical success in any one of the Company's compounds is likely to generate increased interest by pharmaceutical companies with particular interest in the franchises and disease states that Vectus addresses.

Accugen

During the year Vectus has worked to enhance its technology aimed at improving the speed and accuracy of measuring the amount of DNA and RNA in samples tested in laboratories. The technology, consisting of AccuCal™ and RealCount™ software, offers a time, cost and accuracy benefit compared

with currently available systems. Recent activities in the commercialisation programme, which comprises a combination of direct sales, distribution partnerships and licensing opportunities, have broadened the potential market for the Accugen product. Opportunities are being worked on for applications related to food safety, which is a large and growing market. The Accugen reagent (AccuCal-D™) and software evaluation continue by internationally renowned research groups for possible utility in diagnostic tests.

The Vectus Team

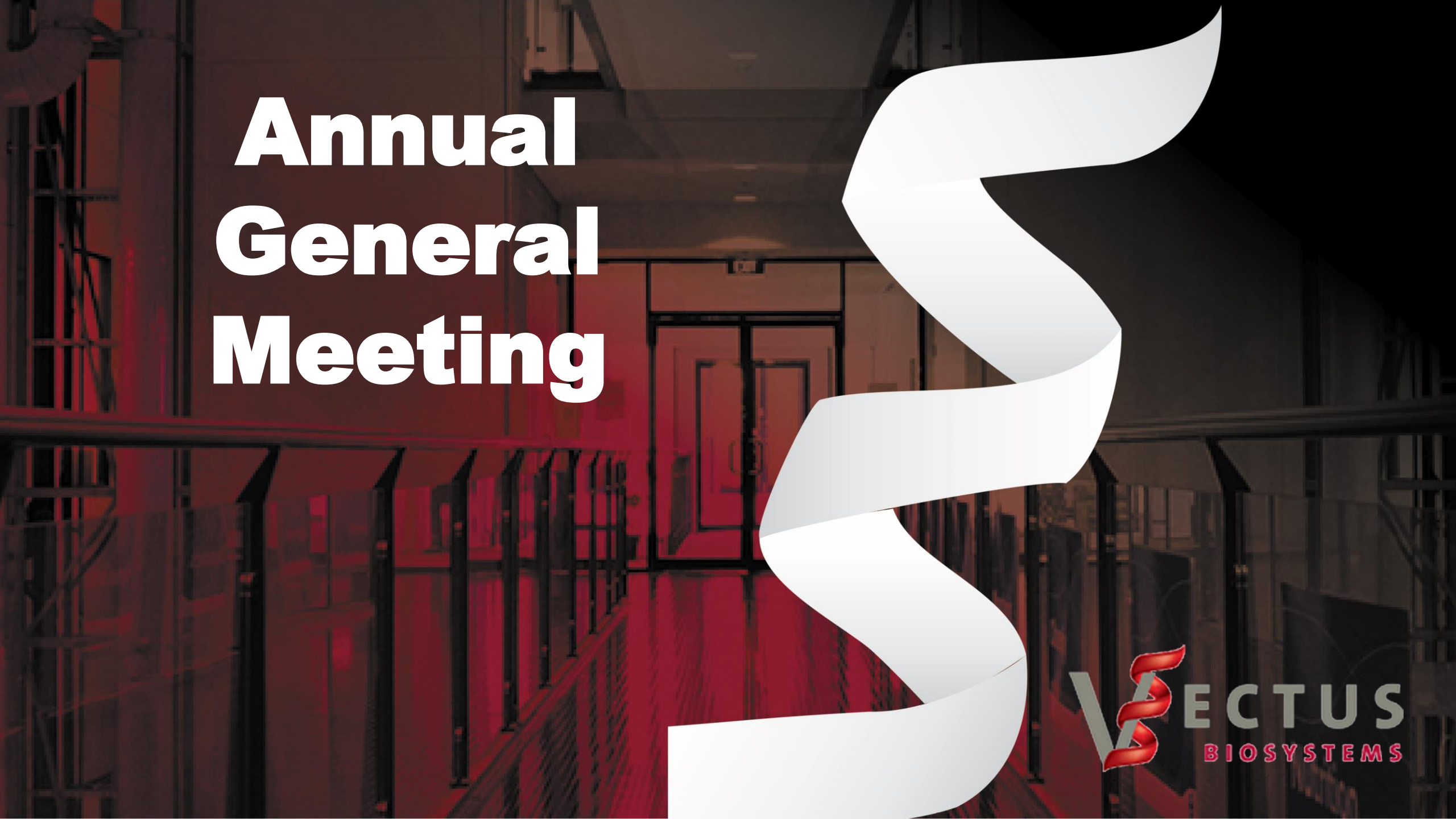
I thank the Vectus team, led by Dr Karen Duggan, for the success in getting VB0004 well into the important Phase I stage during a time of unprecedented challenges. I appreciate the very relevant expertise of the Company's Board, with the experience of Susan Pond in medical research and in large pharmaceutical companies being critical and the strong commercial skills in the medical field of my fellow Directors Maurie Stang, the Deputy Chairman, and Peter Bush.

I thank Vectus' shareholders for their very active support during this exciting phase of the Company's development. We look forward to progressing our activities and growth with the objective of contributing in a meaningful way to society, patients, our stakeholders, and the delivery of improved healthcare worldwide.

Vectus Biosystems Limited

Ron Shnier
Chairman

Annual General Meeting



VECTUS BIOSYSTEMS

VIP AGONISTS - The benchmark for Anti-fibrotics

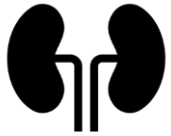
VECTUS BIOSYSTEMS

A SIGNIFICANT MARKET

Fibrosis is the thickening and scarring of connective tissue, usually as a result of injury, and is the pathology which underlies:

HEART FAILURE
(largest single item on US health care budget $US32b in 2013)

LIVER FAILURE
(40% of population of China, India and South East Asia are affected)



KIDNEY FAILURE
(Dialysis and renal transplant costs in the US reached $49.2b in 2011)

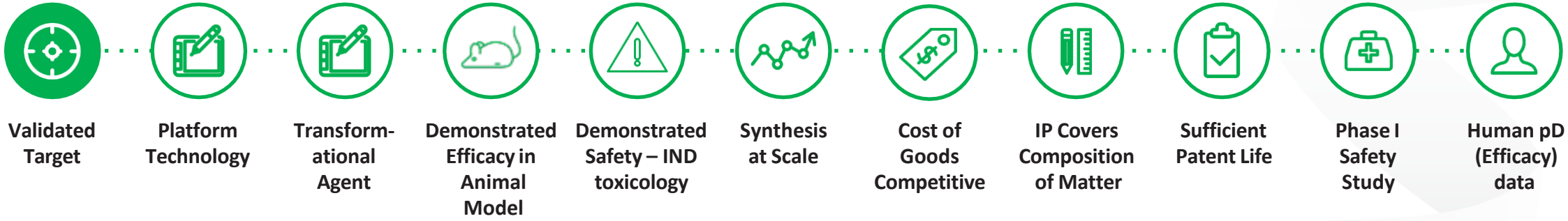
RESPIRATORY FAILURE
(pulmonary fibrosis)

Fibrosis Reversal, a MAJOR unmet need

Fibrotic disease contributes to more than 40% of all deaths worldwide

VB0004 - Heart, lung and kidney fibrosis

PATH TO CLINIC – VB0004



COMPLETED

In Progress

Potential Therapeutic Criteria

TARGET VALIDATION

Treatment with VIP reversed pre-existing cardiac fibrosis in multiple animal models data from one was published in the paper entitled "Vasoactive intestinal peptide reverses existing myocardial fibrosis in the rat"

European Journal of Pharmacology 862 (2019) 172629

Contents lists available at ScienceDirect

European Journal of Pharmacology

journal homepage: www.elsevier.com/locate/ejphar

Full length article

Vasoactive intestinal peptide infusion reverses existing myocardial fibrosis in the rat

Karen A. Duggan*, George Hodge, Juchuan Chen, Tegan Hunter[1]

Vectus Biosystems, North Ryde, Australia

ARTICLE INFO

Keywords:
Heart failure
Myocardial fibrosis
Vasoactive intestinal peptide

ABSTRACT

Congestive cardiac failure has become one of the major health challenges of the 21st century and new therapies are needed to address this problem. The concentration of vasoactive intestinal peptide (VIP) in the heart has been shown to decrease as fibrosis (the pathology leading to heart failure) increases and to become undetectable in end stage cardiomyopathy. We sought to determine whether replenishment of myocardial VIP might treat myocardial fibrosis and therefore represent a new therapeutic target.

Wistar Kyoto rats on a high (4.4%) salt diet were randomised to zero time control, 4 week infusion of VIP (5 pmol/kg/min) or vehicle control infusion. Myocardial VIP concentration was measured by radioimmunoassay, fibrosis was quantitated by computerised histomorphometry and changes in pro-fibrotic mediators were measured by quantitative rt-PCR.

Myocardial VIP increased significantly in VIP treated rats compared with vehicle treated controls ($P < 0.01$) while fibrosis in the VIP treated rats was significantly lower than in both the zero time control ($P < 0.05$) and the vehicle infused control ($P < 0.0005$). Although all six profibrotic mediators which were measured increased over the 4 week experimental period VIP infusion only affected angiotensinogen (Agt) and angiotensin receptor type 1a (AT_{1a}) expression. In both instances VIP caused a significant decrease in messenger rna expression (Agt $P < 0.01$ and At_{1a} $P < 0.01$) compared with vehicle infused controls.

We conclude that VIP infusion increased myocardial VIP concentration and was able to reverse existing myocardial fibrosis suggesting a possible therapeutic role for a VIP based therapy in cardiac failure.

European Journal of Pharmacology 873 (2020) 172979

Contents lists available at ScienceDirect

European Journal of Pharmacology

journal homepage: www.elsevier.com/locate/ejphar

Full length article

Vasoactive intestinal peptide infusion reverses existing renal interstitial fibrosis via a blood pressure independent mechanism in the rat

Karen A. Duggan*, George Hodge, Juchuan Chen, Sofie Trajanovska[1], Tegan Hunter[2]

Vectus Biosystems, North Ryde, Australia

ARTICLE INFO

Keywords:
Renal failure
Tubulointerstitial fibrosis
Vasoactive intestinal peptide

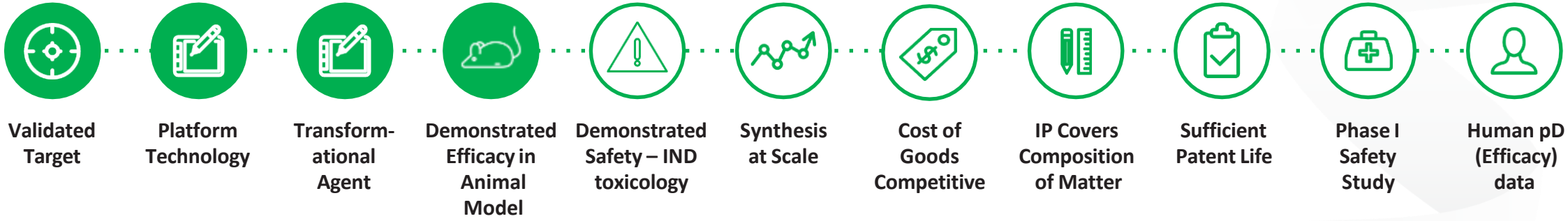
ABSTRACT

Dialysis requiring renal failure is a silent epidemic. Despite an annual mortality of 24% the dialysis population has increased by 1–4% per annum. Regardless of the initial injury, tubulointerstitial fibrosis is a feature of the renal pathology and it inversely correlates with declining renal function. Current agents display little efficacy against tubulointerstitial fibrosis. Clearly, therapies effective against tubulointerstitial fibrosis and able to preserve kidney function are needed. Vasoactive intestinal peptide (VIP) has been shown to reverse pre-existing cardiac fibrosis. We sought to determine whether VIP is effective in tubulointerstitial fibrosis. Spontaneous hypertensive rats (SHR) on a 2.2% salt diet were randomised to zero time control, 4 week infusion of VIP (5 pmol/kg/min) or vehicle control infusion. A fourth group, to match the blood pressure reduction achieved in the VIP infused group was included. Fibrosis was quantitated by computerised histomorphometry, changes in pro-fibrotic mediators were measured by quantitative rt-PCR and macrophage activation assessed by cyclic adenosine monophosphate (c-AMP) response to incubation with VIP. Tubulointerstitial fibrosis in the VIP treated rats was significantly lower than the zero time control ($P < 0.0005$), the vehicle infused control ($P < 0.0005$) and the blood pressure matched group ($P < 0.01$). Although all six profibrotic mediators increased over the 4 week experimental period VIP infusion only decreased tumour necrosis alpha (TNFα) expression significantly ($P < 0.001$). Incubation of RAW264 macrophages with VIP significantly increased c-AMP ($P < 0.01$).

We conclude that VIP infusion reversed existing tubulointerstitial fibrosis suggesting a possible therapeutic role for a VIP based therapy in chronic kidney disease.

Treatment with VIP was also found to reverse interstitial fibrosis in the kidney in multiple animal models. Data from one was published in the paper entitled "Vasoactive intestinal peptide infusion reverses existing renal interstitial fibrosis via a blood pressure independent mechanism in the rat"

PATH TO CLINIC – VB0004



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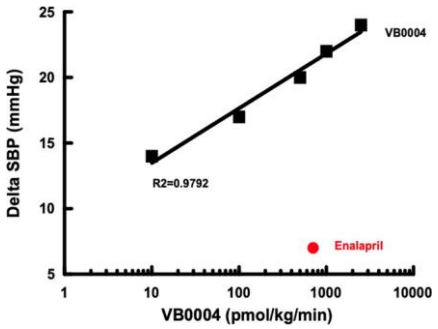
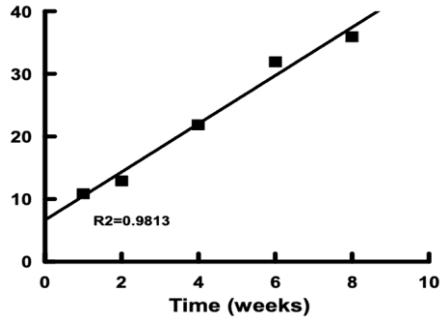
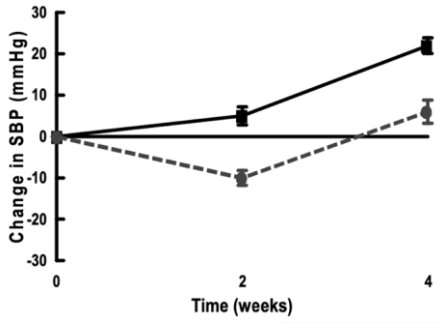
Potential Therapeutic Criteria

VB0004 & SYSTOLIC BLOOD PRESSURE







Left: Decrease in SBP from controls in 18 week old SHR treated with VB0004 at 10, 100, 500, 1,000 and 2,500 pmol/kg/min for 4 weeks. Enalapril dose to achieve a reduction of 7mmHg was 705 pmol/kg/min. SBP decreased with increasing dose to 2,500 pmol/kg/min.

Middle: Difference in SBP from control for SHR treated with VB0004 at 2,500 pmol/kg/min at 1, 2, 4, 6 and 8 weeks. The maximal effect of VB0004 in lowering SBP was not reached after 8 weeks treatment

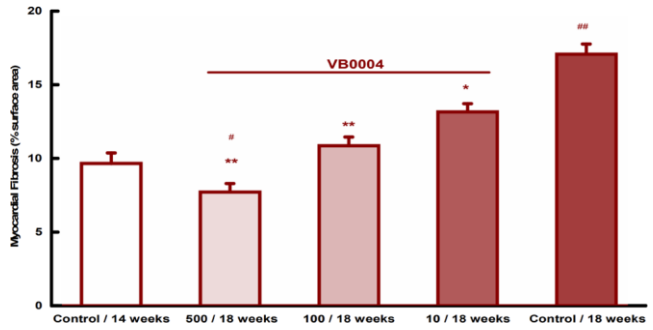
Right: Change in SBP from levels at the commencement of the experiment in Vehicle control for 4 weeks (solid line) SHR treated with VB0004 2,500pmol/kg/min for 2 weeks then vehicle for 2 weeks (dotted line) SBP in increased in parallel with vehicle control after cessation of VB0004

VB0004 & CARDIAC FIBROSIS

VB0004 has been shown to:

- Rescue cardiac tissue damaged by fibrosis
- Repair existing cardiac damage
- i.e. VB0004 is transformational

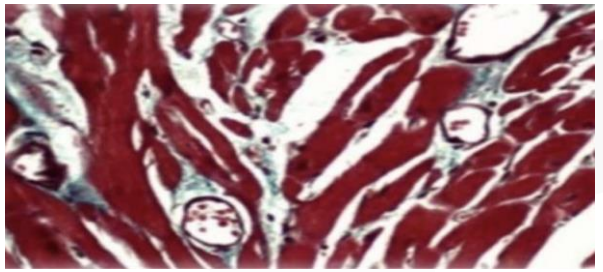
Treatment with VB0004 at 3 Doses



At the highest dose (500pmol/kg/min), VB0004 reversed pre-existing fibrosis, while a dose response effect on the level of fibrosis is apparent

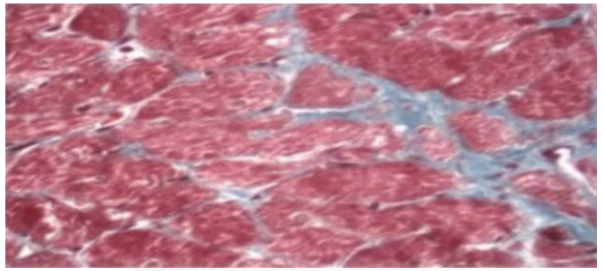
14-Week Control

Fibrous tissue (blue staining) is visible around blood vessels and extending between muscle fibres



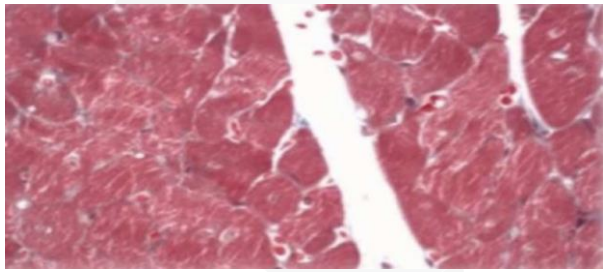
5% Ethanol 18-Week Control (Vehicle Control For VB0004)

Fibrosis visible as blue stained tissue is present throughout the section



Heart At 18 Weeks After 4-week Treatment with VB0004 (500 Pmol/Kg/Min)

Minimal fibrosis is visible; normal architecture has been restored



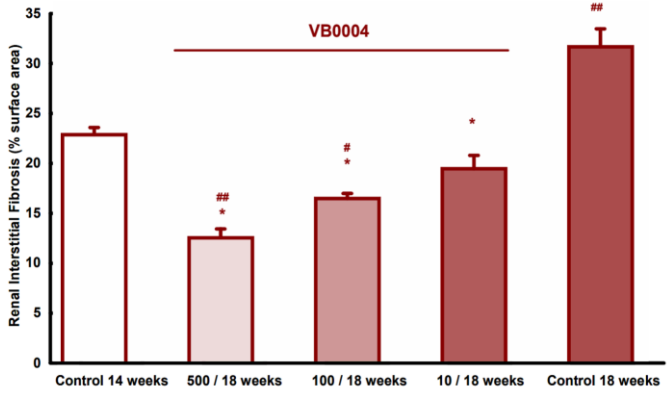
VB0004 & KIDNEY FIBROSIS

In the kidney VB0004 has been shown to:

- Reverse renal interstitial fibrosis at all doses
- Restore normal architecture at all doses

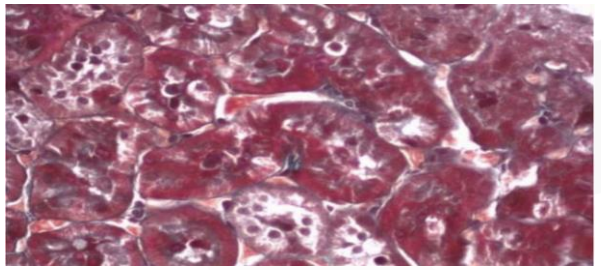
  (i.e. VB0004 is considered transformational)

Treatment with VB0004 at 3 Doses



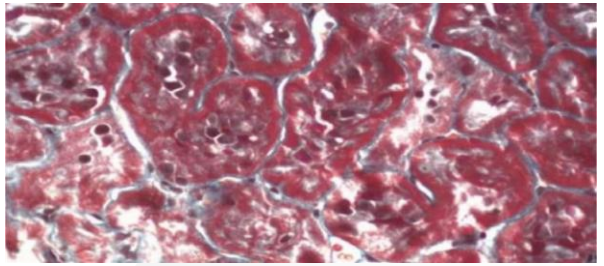
14-Week Control

Fibrosis (blue) partially surrounds some but not all tubukes



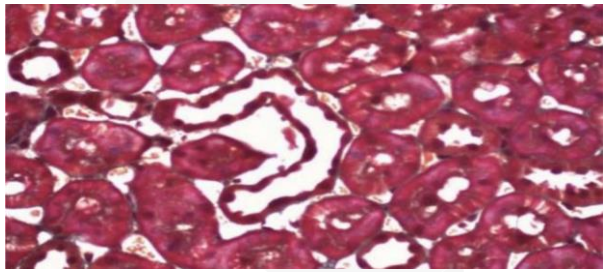
5% Ethanol 18-Week Control (Vehicle Control For VB0004)

Fibrosis has progressed to surround most tubules



Kidney At 18 Weeks After 4-week Treatment with VB0004 (500 Pmol/Kg/Min)

No fibrosis visible



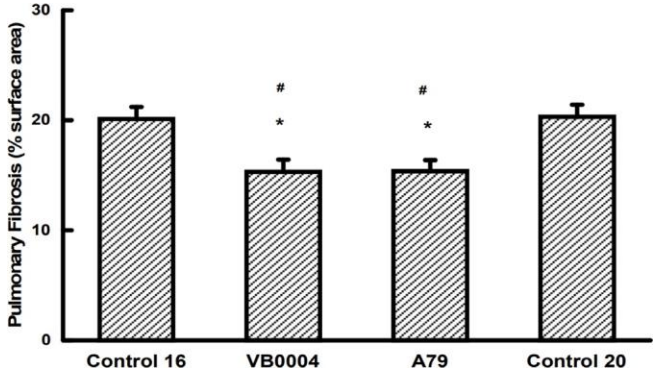
VB0004 & PULMONARY FIBROSIS

In the lung:

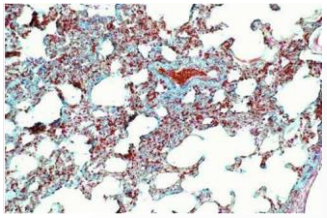
- VB0004 reversed fibrosis present 2 weeks after treatment with bleomycin (an anticancer drug)

  (i.e. VB0004 also transformational in the lung)

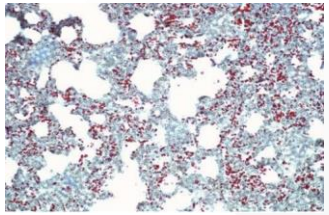
Treatment with VB0004 and VB4-A79



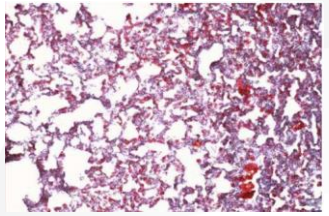
16-Week Control



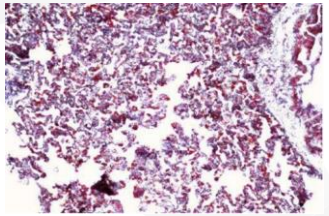
20-Week Control



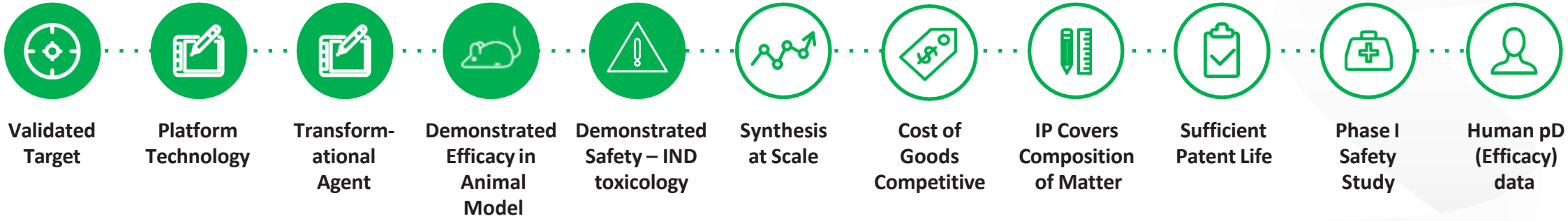
VB4-A79 at 20 weeks



VB0004 at 20 weeks



PATH TO CLINIC – VB0004



COMPLETED

In Progress

Potential Therapeutic Criteria

DEMONSTRATED SAFETY

SAD AND MAD (2 SPECIES)

- Single Ascending Dose (SAD) to 2,000mg/kg no adverse events
- 7 day Multiple Ascending Dose (MAD) to 2,000mg/kg no adverse events
- 28 day MAD to 500mg/kg no adverse

CARDIOVASCULAR SAFETY

- hERG studies - low arrhythmia potential
- Dog cardiovascular safety – No effects on cardiovascular function at maximum dose of 10 grams

RESPIRATORY SAFETY

- Rat study no adverse events

MUTAGENIC POTENTIAL

- In vivo and in vitro tests low to no mutagenic potential

METABOLISM

- Metabolites are the same in human, rat and dog

DRUG INTERACTIONS

- No Inhibition of major drug metabolising enzymes (drug interactions less likely)

PATH TO CLINIC – VB0004

COMPLETED

In Progress

Potential Therapeutic Criteria

SYNTHESIS AT SCALE & COST

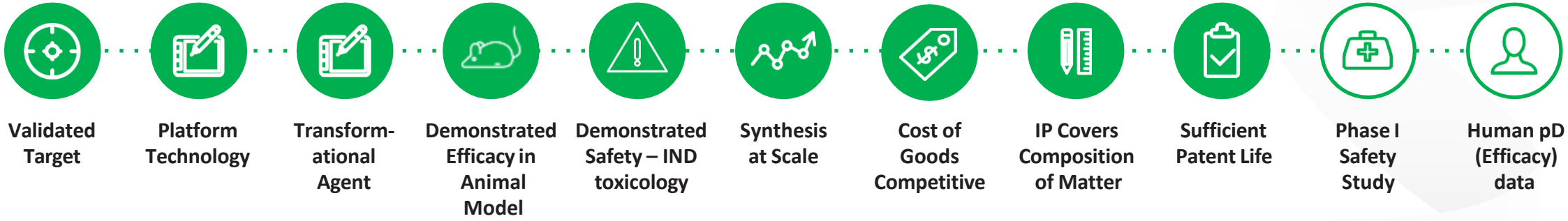
FIRST GMP SYNTHESIS BY GLYCOSYN

- Yield increased as scale increased
- VB0004 manufactured to 5kg scale
- Cost efficient at 5kg scale < $(US) 0.05 per mg
- Estimated dose 1-5mg
- Stability studies – stable at 2 yrs (long shelf-life)

SECOND GMP SYNTHESEIS ASSYCHEM

- Campaign planned to provide 3 validation batches
- Confirm consistency of the synthesis process
- Samples of all 3 will undergo 2 yr stability testing
- Meets FDA requirements for GMP manufacture for Phase 1 and 2 clinical trials

PATH TO CLINIC – VB0004



COMPLETED

In Progress

Potential Therapeutic Criteria

INTELLECTUAL PROPERTY

VB0004 PATENT COVERS

- Compositions of matter
- Methods of use

PATENT LIFE

- Priority date September 2014
- 13 years (+5 years on licensing)

VB0004 PATENT GRANTED IN ALL MAJOR JURISDICTIONS

- USA, Europe, Japan, Peoples Republic of China, Republic of South Korea, Russian Federation
- As well as Australia, Israel, Philippines, South Africa, Canada, ARIPO

VB0004 METHOD OF SYNTHESIS PATENT AT NATIONAL PHASE ENTRY STAGE

PATH TO CLINIC – VB0004

COMPLETED

In Progress

Pharma Criteria

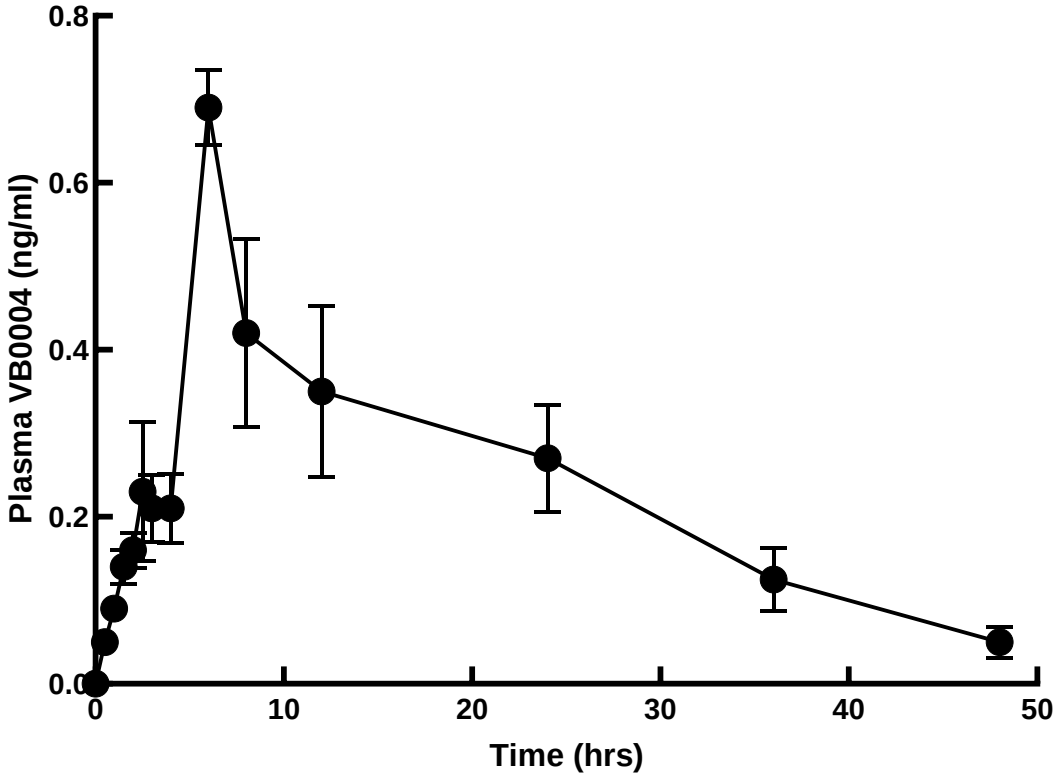
PHASE 1

- Syneos Health (Nasdaq SYNH) retained to write Investigator Brochure (IB), trial protocol and monitor Phase 1 trial
- Trial design conventional Single Ascending Dose (SAD) and Multiple Ascending Dose (MAD)
- Healthy subjects 14 day MAD
- Affected individuals 2 groups 28 days 2 doses
- Biomarkers identified
- Includes pharmacokinetic and pharmacodynamic studies
- Expected outcomes – maximum tolerated dose, dose limiting toxicity (if present), pharmacokinetic data and pharmacodynamic data
- First 2 cohorts of SAD completed no significant adverse events, cohort 3 in progress

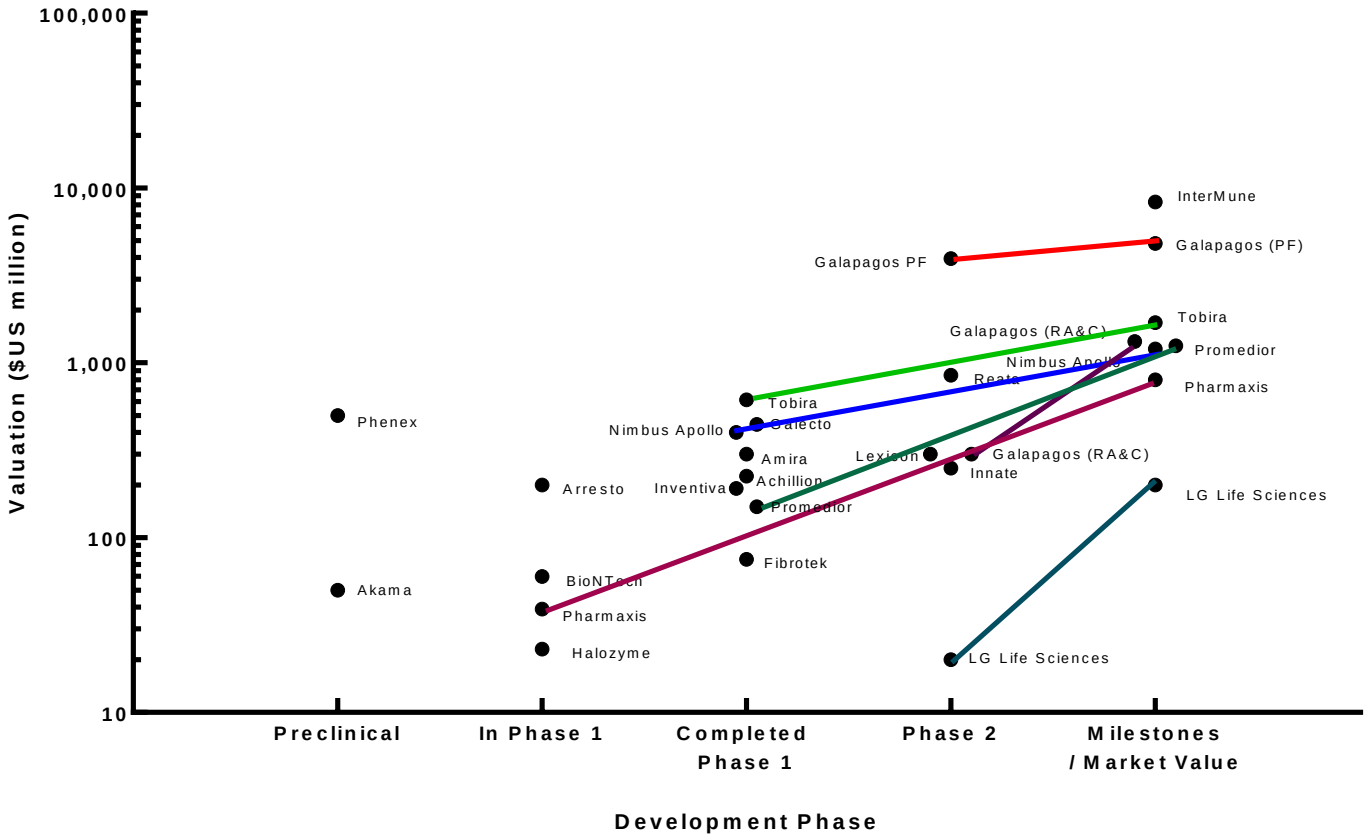
PHASE 1 – To Date

SAD – doses to 30mg completed no adverse events
100mg dose in progress

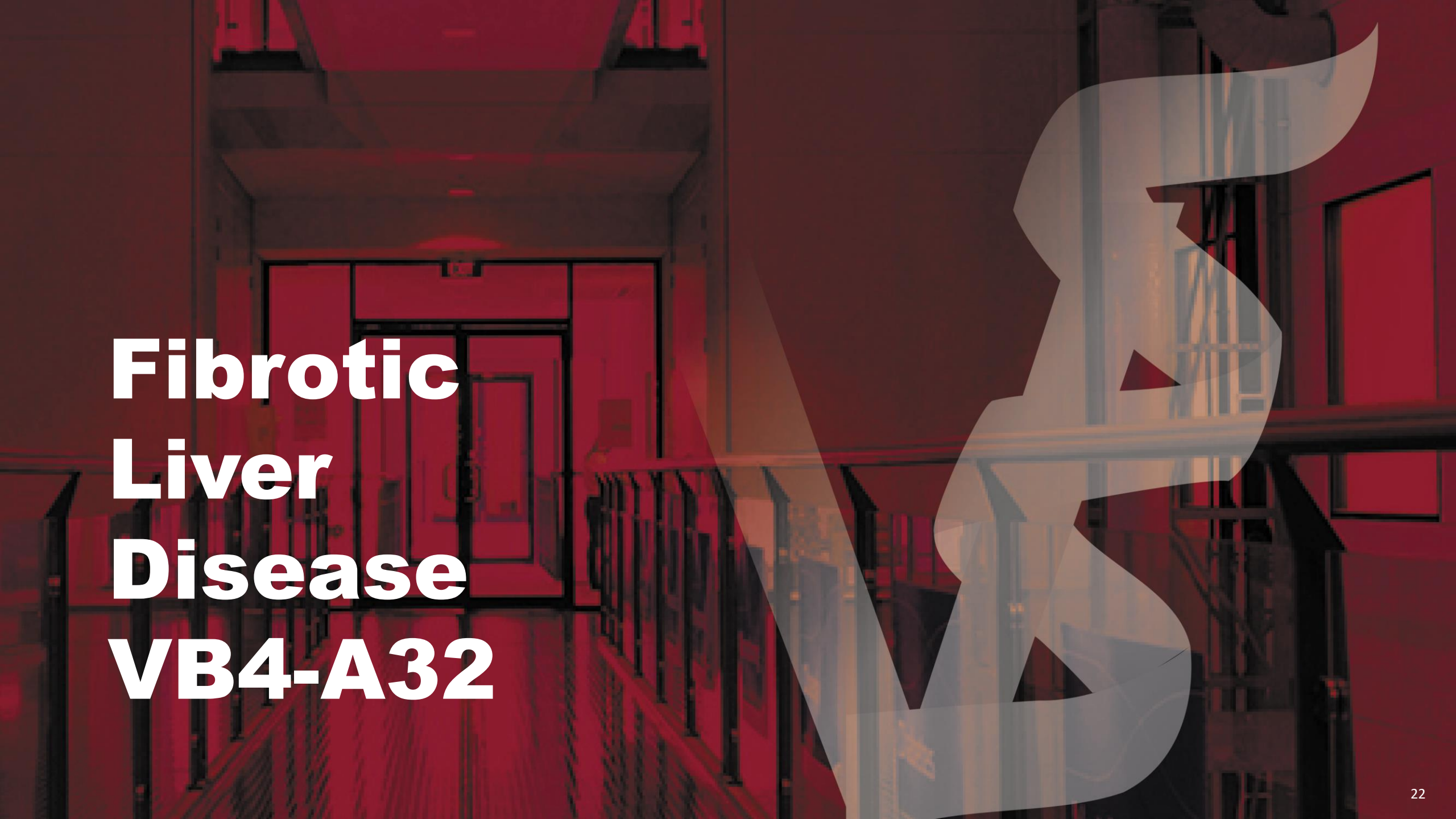
PK – Tmax 6-8 hrs
elimination half life 9.5-10hrs



Comparable Transactions



Successful Phase 1/1b would place Vectus at the point where many transactions have been completed



Fibrotic Liver Disease VB4-A32

HEPATIC CIRRHOSIS (LIVER FIBROSIS)

CAUSES

- Genetic
- Infectious (Hep A, B, C)
- Alcohol related
- Diabetic
- Due to obesity
- Cryptogenic (no discernible cause)

PREVALENCE

- Varies to >40% of the population in countries such as India, Cambodia, Vietnam and China due to endemic Hep B & C.

CURRENT THERAPIES

- Vaccination Hep A, Hep B for prevention
- Abstinence to prevent further damage (EtOH) weight loss
- Diabetes management
- Symptom relief (albumin infusion, ascites removal)
- Sofosbuvir and related agents for Hep C(note this treats the infection but does not prevent progression of the established fibrosis for which lifetime monitoring is required)
- Transplantation

POTENTIAL THERAPIES

FXR agonists

Phase 2 reduced liver fat at 6 months, Phase 3 no change in liver fat, decreased fibrosis at 18 months in 18-23% of patients, none achieved resolution of fibrosis. Side effects - itching moderate to severe in intensity in up to 50%

PPAR-α/δ agonists

Phase 2 decrease in fat and no progression in 19% at 6 months but a reversible loss in renal function. Phase 3 no progression in 20% at 12 months

Insulin sensitisers

Phase 2b no effect on liver disease, but improved insulin sensitivity.

FGF19 analogues

decrease in liver fat in 74-79% at 12 weeks. High incidence of side effects (93%) including injection site reaction, abdominal pain, diarrhoea, nausea

PPAR α & γ agonists

Phase 2 decrease in ALT at 16 weeks and reduced fat at the highest dose vs placebo. Well tolerated.

THR β agonist

Phase 2 decreased ALT, AST and liver fat vs placebo at 12 weeks. SCDI inhibitor- Phase 2b no effect at 12 weeks

ASK1 antagonists

Phase 2 open label decreased fibrosis at 6 months. Phase 3 discontinued as no decrease in fibrosis without worsening NASH at 12 months

Cardoso etal https://doi.org/10.1111/liv.14354

No current approved therapy

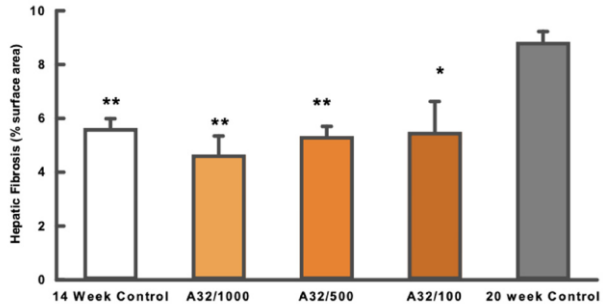
Potential therapies – ineffective and/or high incidence of side effects

Liver Fibrosis continues to represent an unmet therapeutic need

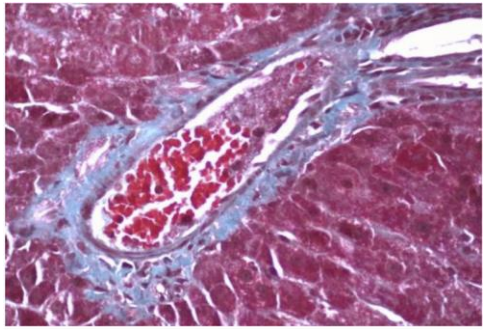
VB4-A32 & HEPATIC CIRRHOSIS

VB4-A32 demonstrated ability to:

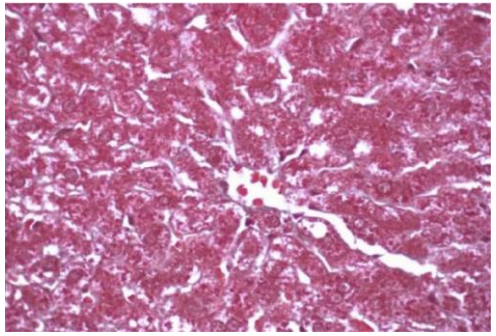
- Reduce peri-portal fibrosis in the liver in a dose dependent manner (right and below)
- Improve liver function tests (below right)

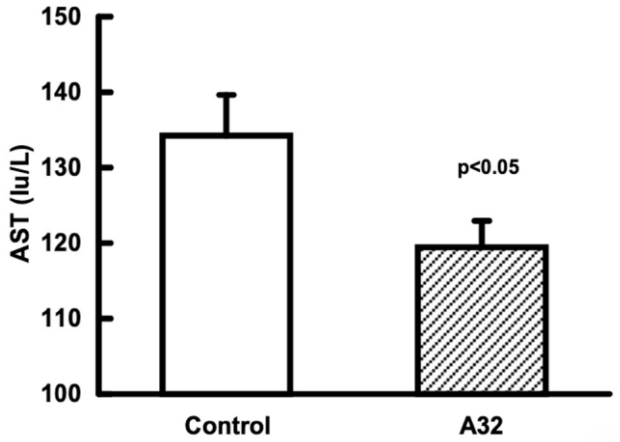


20-Week Control



A32 20 Weeks





Fibrotic Lung Disease VB4-A79

PULMONARY FIBROSIS

Causes

- Environmental (e.g. air pollution, diesel particles)
- Occupational (e.g. dusts such as silica, coal, asbestos, cotton dust)
- Infections (e.g. TB, psittacosis, Spanish flu, COVID-19)
- Drugs (e.g. bleomycin, methotrexate)
- Radiation
- Autoimmune diseases (e.g. sarcoid, SLE, scleroderma, Wegener Granulomatosis)

Current therapies

Pirfenidone

- Regulatory approval approximately 5 years ago
- Slows lung function (FVC and 6MWD) decline
- Approximately 50% discontinued or reduced dose due to side effects

Nintedanib

- Regulatory approval approximately 5 years ago also slows rate of lung function decline.
- High discontinuation rate due to side effects

Essentially, a triggering factor such as coal or silica dust accumulates in the lung which initiates a scarring (fibrotic) reaction to wall off the irritant. However, instead of then turning off once this is achieved the process becomes autonomous and continues to damage the lung even in the absence of continuing exposure.

This results in a reduction in the area available for oxygen to exchange across the lungs and manifests as increasing breathlessness.

POTENTIAL THERAPIES

Pentraxin 2 analogue

Phase 2 showed significant slowing of the decline in FVC and stabilisation of 6MWD at 6 months

Anti-CTGF antibodies

Phase 2 slowed decline in FVC and 6MWD (awaiting review)

Autoxin-LPA Inhibitors

Phase 2a ? Halted FVC decline at 12 weeks. Phase 3 underway

Medium Chain Fatty Acid Analogue (PBI4050)

Phase 2 PBI4050, alone or combined with Nintedanib slowed decline or stabilised FVC at 12 weeks. However, in combination with Pirfenidone the rate of decline increased.

Anti-LOXL.2 Antibodies

No beneficial effect at Phase 2

Anti-interleukin Antibodies

No efficacy

Leukotriene Antagonists

Phase 2, no interim results

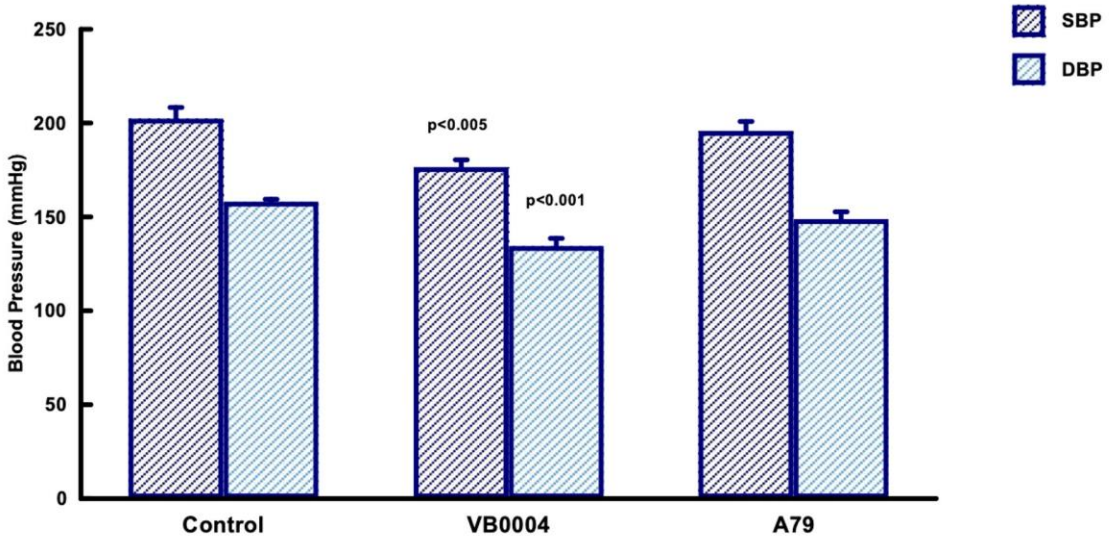
Anti-Integrin Antibodies

Phase 2 completed, awaiting data

Somogyi etal https://doi.org/10.1183/16000617.0021-2019

Current therapies – slow the decline in lung function compared with placebo, but have a high incidence of unacceptable side effects
Potential therapies – slow decline or at best stabilise lung function
Pulmonary Fibrosis continues to represent an unmet therapeutic need

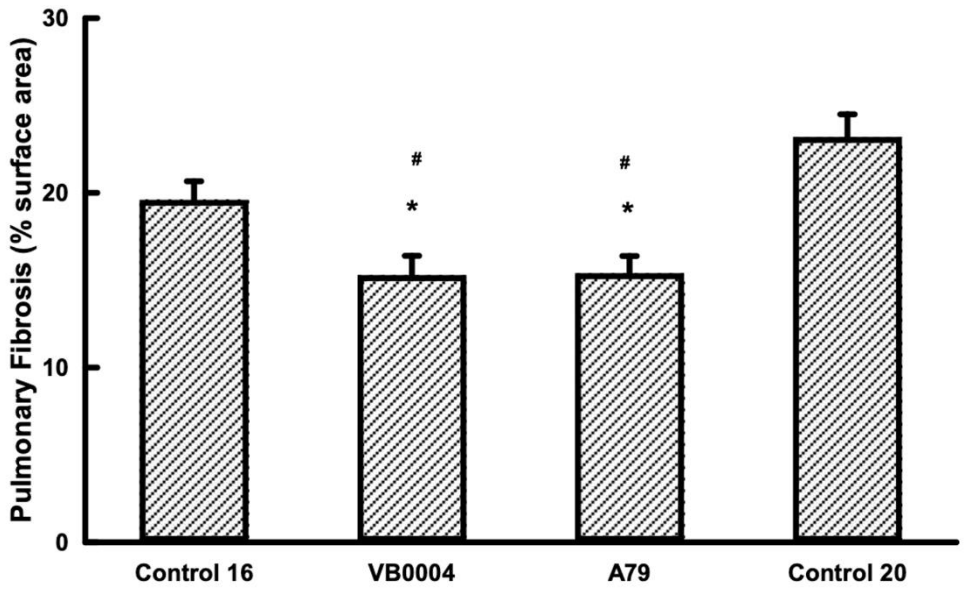
VB4-A79: BLOOD PRESSURE



Systolic and diastolic blood pressure in 20-week SHR following treatment with bleomycin at 14 weeks and randomisation to control, VB0004 or VB4-A79 at 16 weeks.

As previously VB0004 significantly decreases both systolic and diastolic pressure while VB4-A79 had no effect.

VB4-A79: PULMONARY FIBROSIS



Pulmonary fibrosis in 16-week controls (two weeks after Bleomycin administration) and at 20 weeks after 4 weeks treatment in VB0004, VB4-A79 and vehicle control rats. VB0004 and VB4-A79 were administered at 500pmol/kg/min in the drinking solution (5% ethanol) vehicle control is drinking solution alone.

* $p<0.001$ vs 20 week control, # $p<0.01$ vs 16-week control.

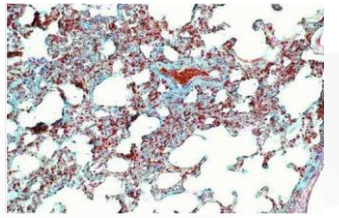
VB4-A79 HISTOLOGY

Lung sections in bleomycin treated rats after 2 weeks of control drinking solution (left), after 6 weeks of control drinking solution (centre) and after 2 weeks of control drinking solution followed by 4 weeks treatment with VB4-A79 or VB0004 (500pmolkg/min).

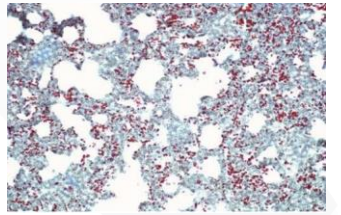
Scar or fibrous tissue appears blue / cyano in these sections. In the controls 2 weeks after bleomycin administration fibrous tissue has thickened many alveoli (air sac) walls but not yet obliterated small blood vessels (capillaries), which appear as red dots which are individual red blood cells.

By 6 weeks in the control rats fibrous tissue is evident causing thickening of all of the alveoli walls and replacing many of the thin walled blood vessels (capillaries) which would normally surround the alveoli allowing gas exchange. In VB4-A79 treated rats alveoli walls are thinner and capillaries are more numerous.

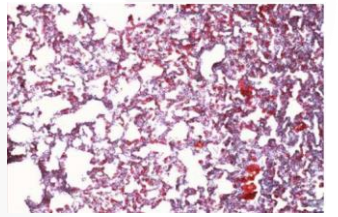
16-Week Control



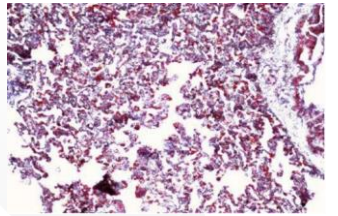
20-Week Control



VB4-A79 at 20 weeks



VB0004 at 20 weeks



PATENT PORFOLIO

VIP patents for heart, kidney and aortic fibrosis

– granted all jurisdictions

VIP fragment patents compositions and methods of use for hypertension, cardiac, renal and aortic fibrosis

– granted most jurisdictions

VB0004 compositions and methods of use for hypertension, cardiac and renal fibrosis

– granted Russian Federation, Israel, Singapore, ARIPO, Canada, Philippines, South Africa, Ukraine, Vietnam, Nigeria, Mexico, accepted in Indonesia

VB0004 library of approx. 70 related compounds compositions and methods of use for treatment of hypertension, cardiac and renal fibrosis

– granted US, Australia, China, Europe, Japan, Korea, Russia, Ukraine, Hong Kong, Vietnam, Singapore, accepted in South Africa, ARIPO, Brazil, accepted Mexico

VB4-A32 and library of related compounds compositions and methods of use for treatment of hepatic, cardiac and renal fibrosis

– granted US, Europe, Australia, South Africa

VB4-P5 and library of related compounds compositions and methods of use for treatment of renal cell death, renal fibrosis and hepatic fibrosis

– granted US, China, Australia, South Africa, accepted Europe, Japan, Russia, Israel

GMP method of synthesis VB0004

– granted USA, Australia, India, accepted Europe, China

VB4-A79 and related compounds compositions and use for treatment of pulmonary fibrosis

– granted Australia, China, accepted USA, Europe, Mexico

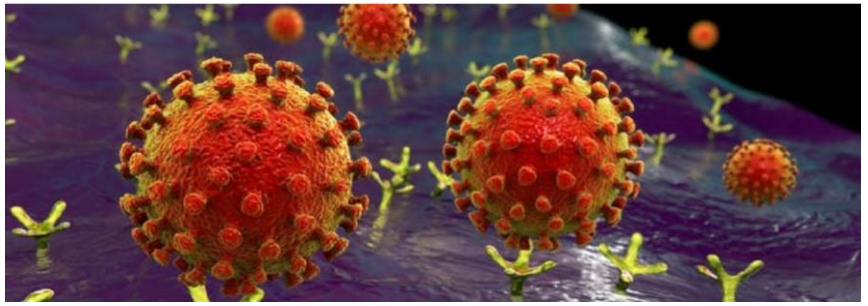
VB0001 and related compounds compositions and use for management of hypertension and fibrotic disease

– PCT application

VB0002, VB0003 and VB0005 and related compounds compositions and use for management of hypertension and fibrotic disease

– national phase

COVID AND FIBROSIS



Sars-Cov2 + ACE2 → ↑ Ang II

Cytokine Storm
↓
ARDS
↓
ICU ± Ventillation
↓
Fibrosis

↑ Agt
↓
↑Ang II
↙
↑TNFα → ↑AT1a
↓
Fibrosis

Fibrosis

Ang II = Angiotensin II
Agt = Angiotensinogen, the Ang II precursor

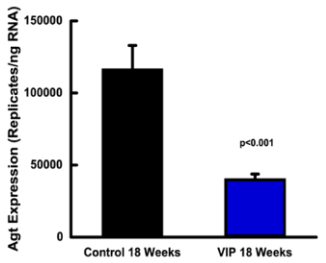
ARDS = Acute Respiratory Distress Syndrome
TNFα= Tumour Necrosis Factor alpha

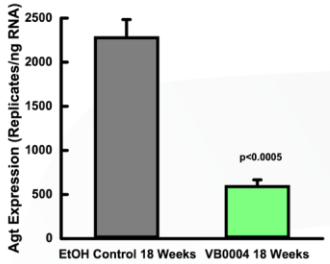
VIP, VB0004 AND POST COVID FIBROSIS

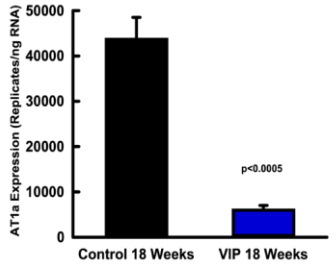
VIP and VB0004 downregulate expression of:

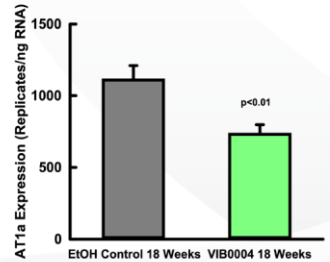
- **Agt (and therefore Ang II)**
- **TNFα**
- **AT1a**

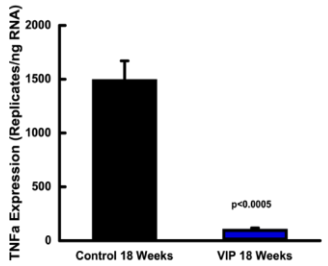
Possible role in treating post Covid Fibrosis

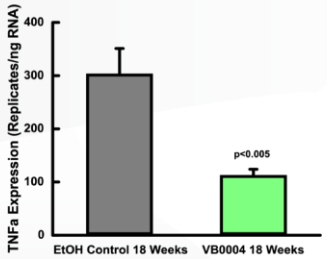




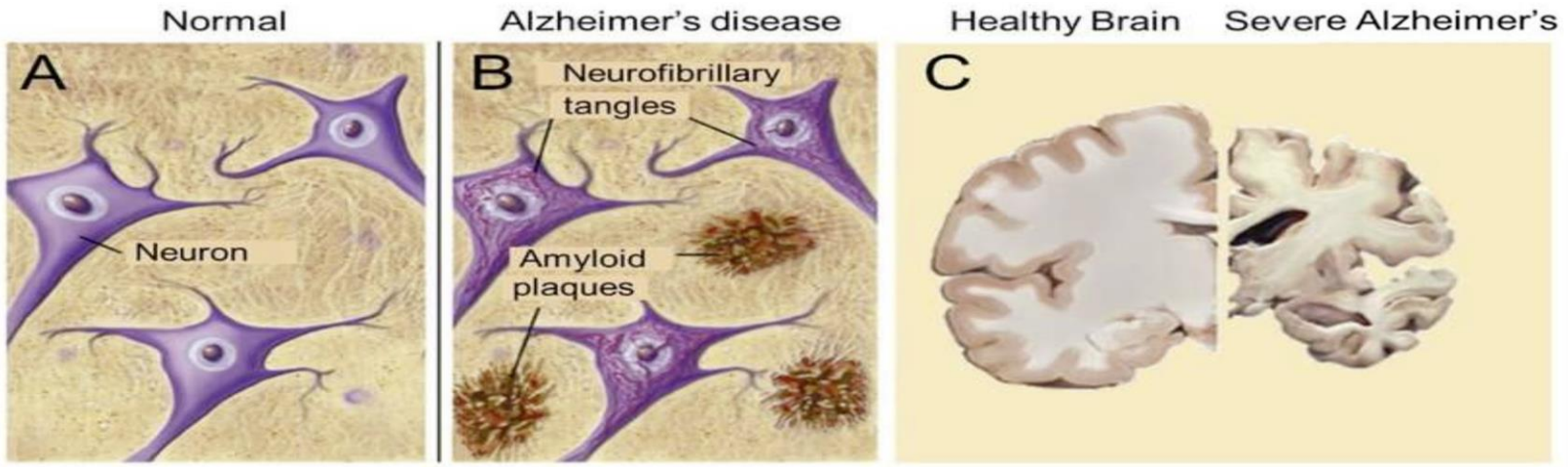








WHY ALZHEIMERS



Accumulation of B-amyloid in the brain causes formation of plaques, which disrupt neuronal connections and cause accumulation of Tau proteins, which are dissociated from microtubules within neurones causing tangle formation, a precursor to neuronal cell death.

VIP AGONISTS - the anti-fibrotic benchmark and a brighter therapeutic future

VECTUS BIOSYSTEMS