ASX: VBS

VECTUS BIOSYSTEMS

A differentiated approach to pulmonary fibrosis

Phase 1A

Safety Completed

Phase 1B

Next Value Inflection

IPF

Lead Indication

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VECTUS BIOSYSTEMS

ASX: VBS

Forward-looking statements:
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The purpose of this presentation is to provide general information about Vectus and its subsidiary and business. The information in this presentation is current as at 13 May 2026. It is in summary form and is not necessarily complete. It should be read together with the Appendix 4D and Half-year report and other ASX announcements by VBS.

SHARE PRICE

$0.125

per share (AUD)

MARKET CAPITALISATION

$6.69M

AUD

CAPITAL STRUCTURE

Shares on issue	53.54M
Share price	A$0.125
Market capitalisation	A$6.69M
Options	1.35M

Idiopathic Pulmonary Fibrosis (IPF): A mostly fatal disease with no cure

Current therapies slow progression, none known to reverse fibrosis. Median survival: 2–5 years from diagnosis.

~3M

patients globally

2–5 yr

median survival

$5B+

global market by 2030

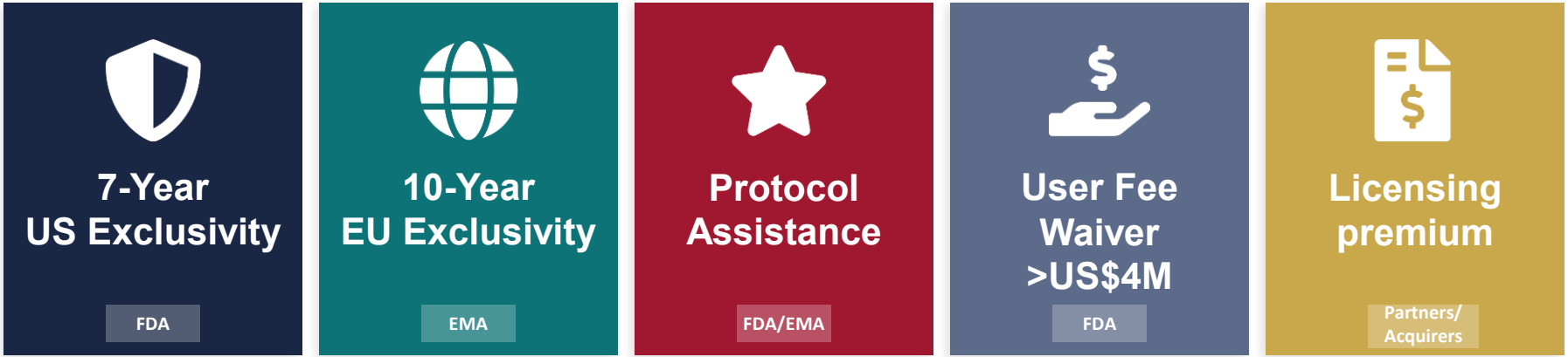
Significant orphan opportunity

IPF FACTS

- Incidence is rising; more common in men over 60
- Diagnosis often delayed 1–2 years after symptom onset
- Progressive, irreversible lung scarring (alveoli replaced by scar)
- Pirfenidone & nintedanib: ~50% slower decline; high discontinuation due to tolerability
- No approved therapy reverses established fibrosis
- Lung transplant is the only option for end-stage disease

IPF is a rare disease.

Vectus can target Orphan Drug Designation for VB0004 in IPF



IPF affects <200,000 Americans - meets FDA rare disease threshold

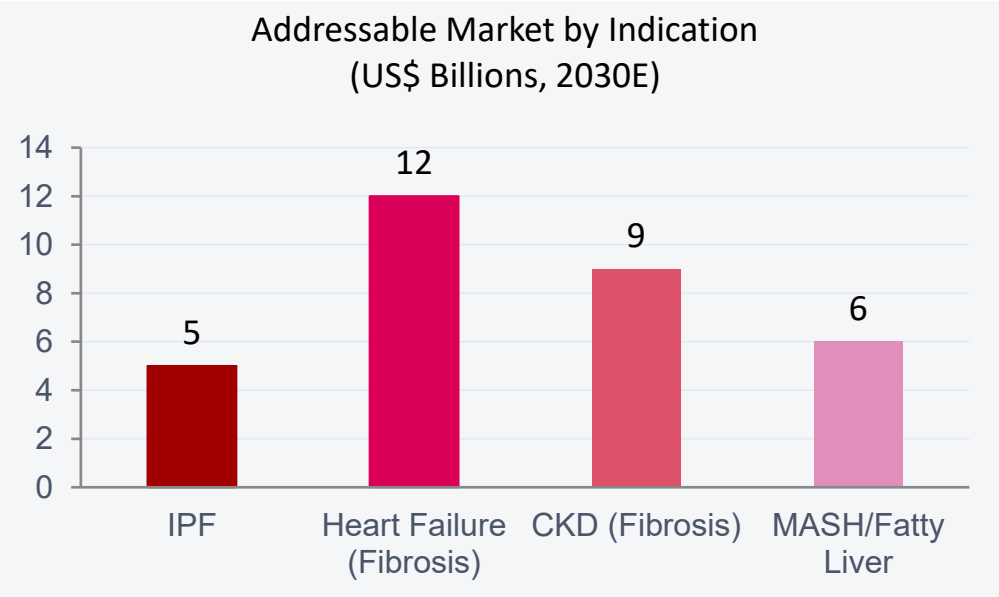
IPF as rare disease -precedent well established

Benefits begin at designation, which can occur at Phase 1 trial (not at approval)

Filing for ODD is a near-term, low-cost catalyst

A Platform Opportunity Across Fibrotic Disease

Fibrosis underlies >40% of deaths globally. Vectus' lead program targets IPF first - with platform expansion potential across heart, kidney, and liver.



IPF: Lead Indication
~3M patients globally. $5B+ market by 2030. No disease-reversing therapy approved. Rapid orphan drug pathway potential.

Cardiovascular Fibrosis
Heart failure largest US healthcare budget item. VB0004 shows reversal of cardiac fibrosis in preclinical models.

Chronic Kidney Disease
Dialysis & transplant costs $49B+ annually (US). Renal fibrosis reversal demonstrated at all doses in animal models.

Platform Value
One mechanism. Multiple organs. Early external validation: antifibrotic asset out-licensed to Canadian biotech.

VB0004: First-in-Class Antifibrotic with Human Safety Data

Differentiated Mechanism

- Targets VIP pathway via NPR-C receptor
- Increases nitric oxide & cGMP
- Downregulates TNF-α & pro-fibrotic mediators
- Dual: vasodilation + fibrolysis
- First-in-class VIP agonist platform

Favourable Drug Profile

- Small molecule — oral, once-daily dosing
- Tmax 6–8 hrs; half-life ~10–15 hrs
- No accumulation over 14 days
- Stable >2 years - long shelf life
- GI tolerable unlike current options

De-Risked Asset

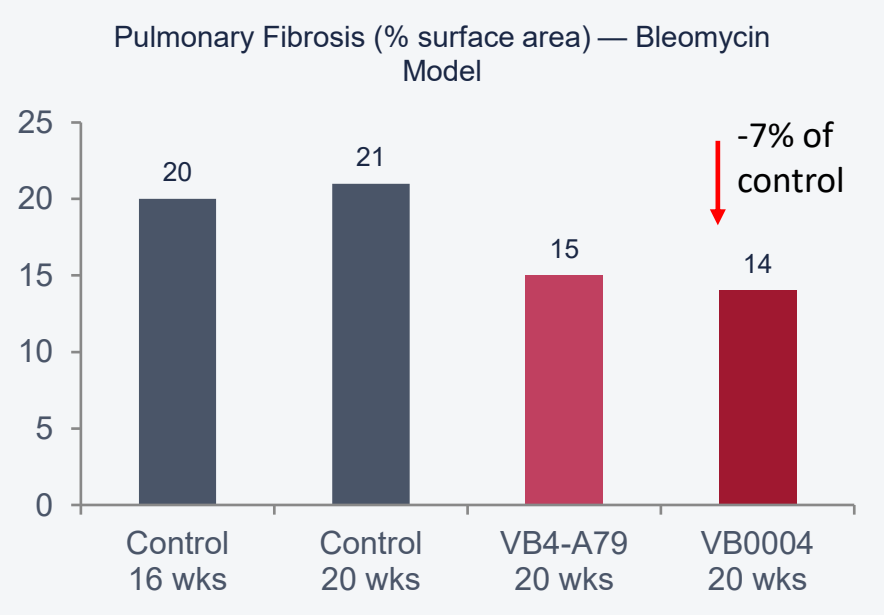
- Phase 1 SAD & MAD completed
- No significant adverse events
- GMP manufacturing validated at 2 centres
- Composition-of-matter IP in major jurisdictions
- Preclinical reversal: lung, heart, kidney

The only known antifibrotic to reverse established disease in preclinical models

VB0004 demonstrated reversal of established fibrosis in multiple preclinical organ models - a distinction no approved therapy has achieved.

	VB0004 (Vectus)	Nintedanib	Pirfenidone
Slows disease progression	✓	✓	✓
Reverses established fibrosis	✓ (preclinical)	X	X
Oral once-daily dosing	✓	X (2×/day)	X (3×/day)
Favourable tolerability	✓ (Phase 1)	Moderate	Moderate
Multi-organ activity	✓ Lung, Heart, Kidney	Lung only	Lung only
First-in-class mechanism	✓ NPR-C / VIP agonist	X	X

VB0004 Reverses Established Lung Fibrosis in Preclinical Model



16-week Control
Established pulmonary fibrosis — collagen deposition throughout lung parenchyma

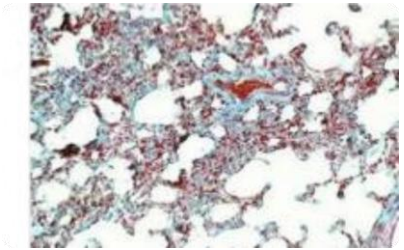
20-week Control (Vehicle)
Fibrosis has progressed — additional collagen deposition, reduced airspace

VB4-A79 (20 wks)
Significant reduction in fibrotic burden vs vehicle control — partial reversal

VB0004 (20 wks)
Marked reduction in fibrosis — near-normal lung architecture restored

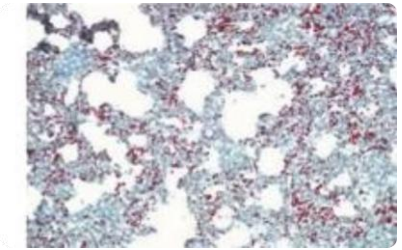
Note: Bleomycin-induced pulmonary fibrosis model. VB0004 administered after fibrosis established (2 weeks post-bleomycin).

VB0004 Reverses Established Lung Fibrosis in Preclinical Model



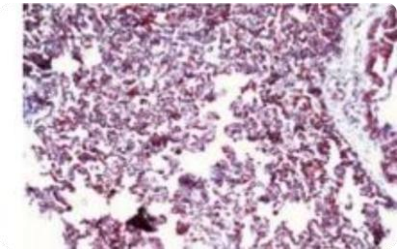
16-Week Fibrosis Model Control

Increased fibrosis (cyano), reduced capillaries



20-Week Fibrosis Model Control

Increasing fibrosis, decreasing capillaries



VB0004 in Fibrosis Model at 20 Weeks

Fibrosis removed, capillaries

Note: Bleomycin-induced pulmonary fibrosis model. VB0004 administered after fibrosis established (2 weeks post-bleomycin).
Histology: Masson's trichrome staining. Source: internal preclinical data.

Reversal of Established Fibrosis Across Multiple Organs

Published peer-reviewed data from European Journal of Pharmacology. Dose-dependent effects; normal tissue architecture restored.

LUNG

VB0004 reversed established pulmonary fibrosis 2 weeks after bleomycin induction

- Significant reduction in % surface area fibrosis
- Both VB0004 and VB4-A79 show activity
- Histology: near-normal lung architecture restored

Internal data

HEART

At highest dose (500 pmol/kg/min), pre-existing cardiac fibrosis fully reversed

- Dose-response relationship demonstrated
- Fibrous tissue (blue staining) reduced to minimal
- Normal cardiac architecture restored at 18 weeks

Eur J Pharmacol 802 (2019) 172029

KIDNEY

Renal interstitial fibrosis reversed at ALL doses — transformational finding

- Complete reversal at 500 pmol/kg/min
- Normal tubular architecture restored
- Blood pressure-independent mechanism confirmed

Eur J Pharmacol 873 (2020) 172979

Phase 1a Completed: Strong Safety & Predictable Pharmacokinetics

SAD — Single Ascending Dose

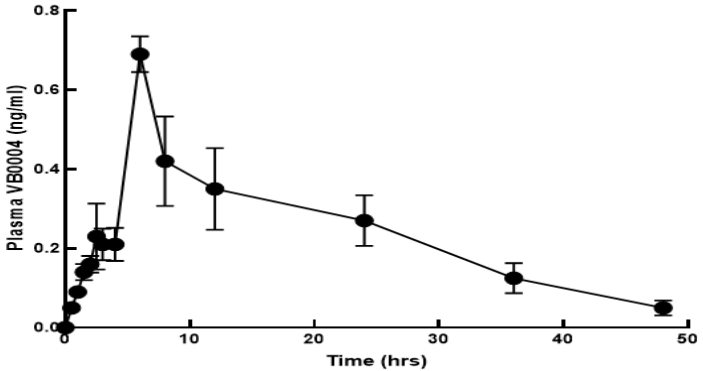
Max planned: 300mg
Max tolerated: 300mg
No significant adverse events

Dose ceiling not reached

MAD — 14-Day Multiple Ascending Dose

Max planned: 100mg/day
Max tolerated: 100mg/day
No significant adverse events

Healthy subjects & mild hypertensives



6–8 hr	Tmax
~10–15 hr	Half-life
None	Accumulation over 14 days
Once daily	Consistent dosing profile

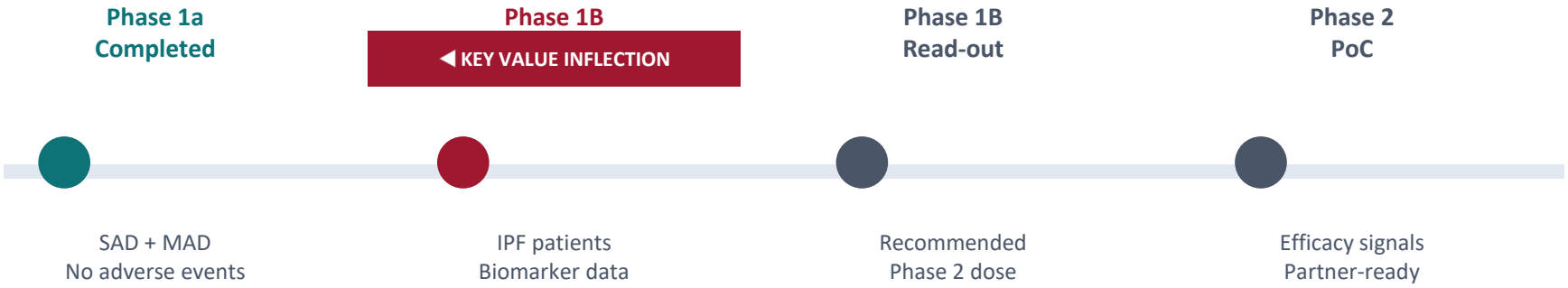
NEXT VALUE INFLECTION

Phase 1B: First Patient Data in Pulmonary Fibrosis

This is the milestone that de-risks the program, establishes antifibrotic activity in humans, and positions VBS for Phase 2 and strategic partnership discussions.

Population:	Mild pulmonary fibrosis / early ILD patients
Design:	Randomised, double-blind, placebo-controlled MAD
Duration:	21 days dosing — 2 doses, 2 groups
Primary Endpoint:	Safety, tolerability & dose-limiting toxicities
Biomarkers:	KL-6, SP-D, MMP-7, PRO-C3, FVC, DLCO
CRO:	CRO-led
Outcome:	Recommended Phase 2 dose + first antifibrotic signals in humans

Clear Path to Value Creation



Pipeline Overview

Program	Lead Indication	Stage	Status
VB0004	Pulmonary Fibrosis (IPF) (Cardiovascular & Renal benefits also demonstrated)	**Phase 1B Ready**	Lead Program
VB4-A79	Pulmonary Fibrosis (non-scleroderma)	Preclinical	Pipeline
VB4-A32	Liver Fibrosis (NASH/ASH)	Preclinical	Pipeline
VB4-P5	Renal Fibrosis	Preclinical	**Licensed — external validation**

Partnering-Led Model with Regulatory Acceleration Pathways

REGULATORY PATHWAY

Phase 1B → Phase 2 PoC

Standard pathway; IPF patient population; biomarker-driven design

Orphan Drug Designation

IPF qualifies — accelerated review, exclusivity benefits, fee waivers

FDA Engagement

Type C / End-of-Phase 2 meetings planned to align on design and endpoints

TGA / EMA Parallel

Australian and European regulatory alignment from Phase 2 design

COMMERCIALISATION MODEL

Out-licensing led

Advance to Phase 1B/2 PoC, then regional or global licensing to established pharma

Platform value

Multiple antifibrotic assets create flexibility for parallel partnering by organ or geography

External validation

VB4-P5 already out-licensed to Canadian biotech — proof of platform interest

Payer readiness

PBS, Medicare, private insurers — fibrotic disease is a reimbursed category globally

Capital efficiency

CRO-led model; leverage partner infrastructure for Phase 3 and global launch

Broad IP Protection + Validated GMP Manufacturing

PATENT PORTFOLIO

- VB0004 composition-of-matter: granted USA, Europe, Japan, China, Korea, Russia, Australia, Canada + others
- VB0004 library (~70 compounds): granted in all major jurisdictions
- VB4-A79 (pulmonary fibrosis): granted Australia, China; accepted USA, Europe
- VB4-A32 (liver fibrosis): granted US, Europe, Australia
- GMP synthesis method: granted USA, Australia, India
- >1,000 compound library encompassed within the patent estate

GMP MANUFACTURING

Two GMP centres validated
Process consistency confirmed

5kg scale demonstrated
Yield improves with scale; adequate for Phase 2 and beyond

Stability >2 years
Long shelf life — commercially viable; 3 validation batches underway

FDA Phase 1 & 2 ready
Manufacturing meets requirements for continued clinical development

Experienced Team Aligned to This Stage of Development

Dr Tara Speranza

CEO & CTO

20+ years across scientific research, commercial strategy & capital markets. Translational drug development at University of Sydney & University of Geneva. Led commercial partnership with Servier (Protelos). Extensive biotech investment and advisory roles.

Dr Ronald Shnier

Non-Executive Chairman

Radiologist; founder of one of Australia's first private MRI practices; former National Director of Diagnostic Imaging at Mayne Health; active in clinical research and international advisory boards. Dr Shnier was the Chief Medical Officer at I-MED Radiology Network for 7 years and continues to serve a Director on the Board of I-MED.

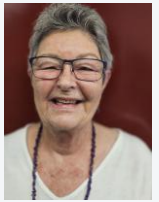
Mr Maurie Stang

Non-Executive Deputy Chairman

30+ years in healthcare and biotechnology. Strong track record in global IP commercialisation. Executive Chairman of Lumitron Technologies.

Ms Linda Walters

Non-Executive Director



25+ years in life sciences and healthcare. Deep familiarity with Vectus technology and IP. Expertise across commercialisation, finance, HR and IT.

De-Risking Milestones

Stage 1

Phase 1B initiation & execution

- CRO engagement & Phase 1B trial costs
- Biomarker panel & PK/PD analysis
- Regulatory preparation (IND, FDA & TGA)
- Working capital — operations

Stage 2

Phase 2 PoC & partnering readiness

- Phase 2 trial design & initiation
- GMP manufacturing scale-up
- IP maintenance & new filings
- Strategic advisory & BD resourcing

KEY GATING EVENTS FOR SCALE CAPITAL

- Phase 1B safety & tolerability data in mild IPF patients
- Recommended Phase 2 dose established
- Antifibrotic biomarker signals (KL-6, MMP-7, PRO-C3)
- Regulatory alignment on Phase 2 design

WHY INVEST NOW

Vectus Biosystems — The Investment Thesis

01

Unmet Need, Large Markets

IPF kills 2–5 years post-diagnosis. $5B+ global market. No approved therapy reverses fibrosis.
Vectus assets known to target substantial markets for cardiac, renal and hepatic diseases.

02

Differentiated & De-Risked

Phase 1 completed. Strong safety profile. Preclinical reversal of lung, heart, and kidney fibrosis. Published peer-reviewed data.

03

Clear Next Catalyst

Phase 1B in IPF patients is fully defined and ready. Biomarker-rich design maximises data value per dollar invested.

04

Broad IP & Platform

>1,000 compounds. Patents granted in all major markets. External out-licensing validates platform interest.

05

Partnering-Optimised

Strategy built around early licensing to global pharma. CRO-led model is capital-efficient and transaction-ready.

06

Strategic Reset Complete

New CEO. Refined clinical plan. Renewed focus on IPF. Positioned for capital raise and Phase 1B initiation.

VECTUS BIOSYSTEMS

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GET IN TOUCH

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CEO & CTO

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VIP - TARGET VALIDATION - APPENDIX

Treatment with VIP reversed 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

Vectus Biosystems, North Ryde, Australia

ARTICLE INFO

Keywords:
Heart failure
Myocardial fibrosis
Vasoactive intestinal peptide

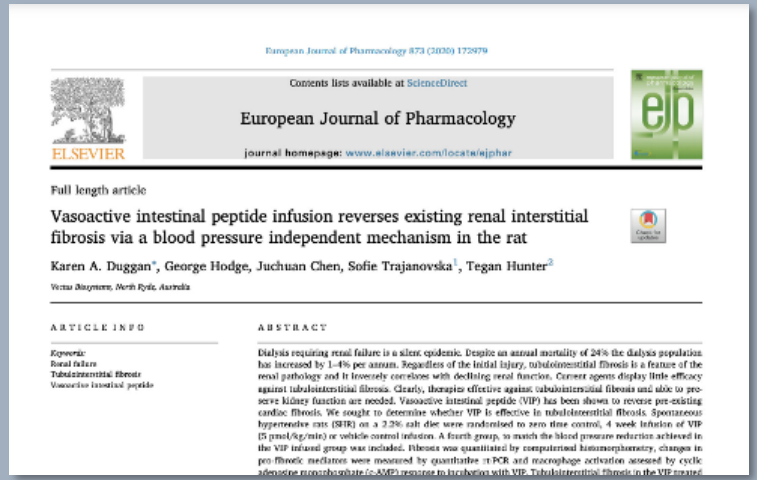
ABSTRACT

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

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"



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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 insecurely 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