BENITEC BIOPHARMA
silencing genes for life™

Investor Presentation
June 2015

Forward Looking Statements

Today's presentation includes forward-looking statements intended to qualify for the Safe Harbor from liability established by the Private Securities Litigation Reform Act of 1995. These forward-looking statements, including statements regarding our planned pre-clinical studies and clinical trials, regulatory approval process and demand for our product candidates, are subject to risks, uncertainties and other factors that could cause actual results to differ materially from those suggested by our forward-looking statements.

These factors include, but are not limited to, the following: we have incurred significant net losses and anticipate that we will continue to incur significant net losses for the foreseeable future; we have never generated any revenue from product sales and may never be profitable; we will need to raise additional funding in the future, which may not be available on acceptable terms, or at all; no product candidates utilizing ddRNAi technology have been approved for commercial sale in the United States, and our approach to the development of ddRNAi technology may not result in safe, effective or marketable products; we are early in our product development efforts and may not be able to obtain regulatory approvals for the commercialization of some or all of our product candidates; our ability to develop and successfully commercialize product candidates may be compromised by other companies developing their technologies or product candidates for our target indications more rapidly than we do or if their technologies are more effective; we may not be able to obtain exclusivity or intellectual property rights for our product candidates or prevent others from developing similar competitive products; issues may arise that impact ddRNAi delivery into the cells and limit our ability to develop and commercialize product candidates.

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Investment Highlights

NOVEL GENE SILENCING PLATFORM	ddRNAi combines RNAi with gene therapy delivery to potentially provide "one shot" treatments and cures for a variety of diseases
BROAD PIPELINE	Programs in indications with high unmet clinical need or large patient populations such as hepatitis B and C, lung cancer, and macular degeneration. Key inflection points in 2015 and 2016
CLEAR STRATEGY	Maintain leadership in development of ddRNAi-based therapeutics through clinical proof-of-concept for a range of human diseases
MANAGEMENT EXPERTISE	Significant experience in designing and developing ddRNAi therapeutics; founding scientists in the ddRNAi field
STRONG INTELLECTUAL PROPERTY POSITION	Portfolio of patents, patent applications, and rights to intellectual property directed to our ddRNAi platform and each product candidate

Management Team and Investors

Management Team	
Dr Peter French *Managing Director and Chief Executive Officer*	Former Chief Scientific Officer, Benitec Biopharma Prior roles at CSIRO and St Vincent's Hospital Sydney
Greg West *Chief Financial Officer*	Former CFO, Immune Systems Therapeutics Prior roles at PriceWaterhouse, Bankers Trust, Deutsche Bank and NZI
Dr David Suhy *Chief Scientific Officer*	Former SVP of Research & Development, Benitec Biopharma Prior roles at Antara Biosciences and PPD Discovery
Carl Stubbings *Chief Business Officer*	Former VP of Sales & Marketing, Focus Diagnostics Prior role at PanBio Pty Ltd
Georgina Kilfoil *Chief Clinical Officer*	Former VP of Clinical Operations, Benitec Biopharma Prior roles at Anthera Pharmaceuticals, InClin and Peninsula Pharmaceuticals
Dr Michael Graham *Head of Discovery and Founding Scientist*	Discoverer of ddRNAi at CSIRO; Former Senior Research Fellow, University of Queensland Prior roles at Benitec, QDPI and CSIRO
Sakura Holloway *SVP, Corporate Development and IP Counsel*	Former Head of RNAi Commercialization, CSIRO Prior roles at Cephalon Australia (Arana Therapeutics) and Garvan Institute, Sydney

Investors

Ridgeback

RACAPITAL

AURIGA

Sabby

Sphera global health care

Perceptive

BLACKWELL GLOBAL

DAFNA Capital Management, LLC

Special Situations

Value, Investment & Opportunity

Company	Country Listed In	Technology	Stage	Market Cap (US $M)[1]
Alnylam	United States	siRNA	Phase III	10,100
Isis	United States	Antisense	Marketed	7,488
Tekmira	United States	RNAi	Phase II	759
Arrowhead	United States	siRNA	Phase II	374
Dicerna	United States	siRNA	Phase I	348
Benitec	**Australia**	**ddRNAi**	**Phase I/IIa**	**76[2]**

1. As of May 15, 2015
2. Converted into US$

Benitec: Leader in ddRNAi Technology

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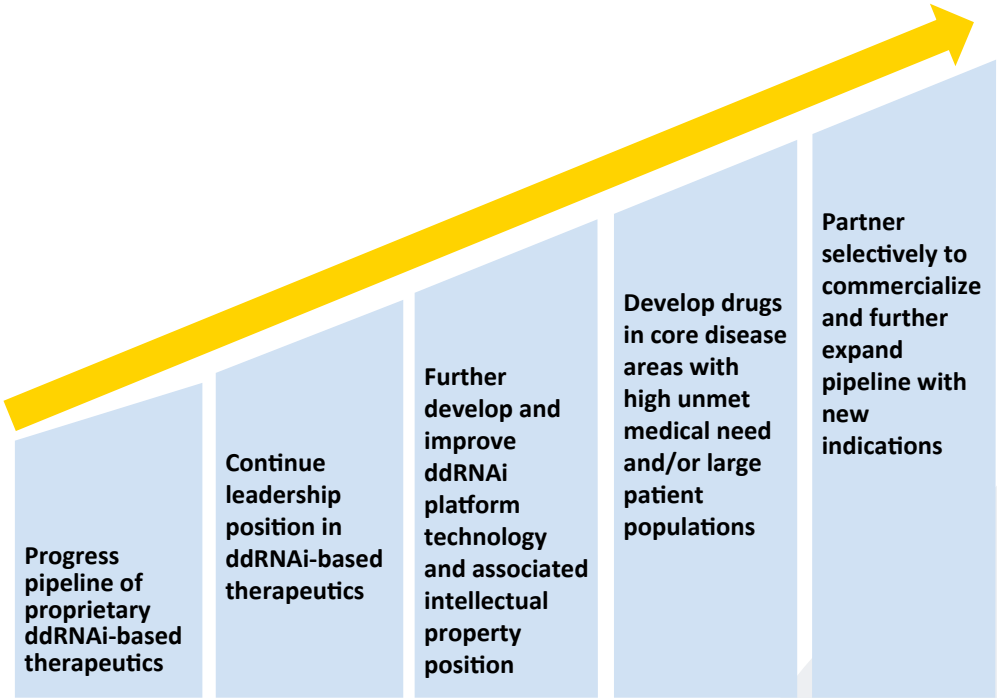
Company Overview

- Clinical-stage biotechnology company developing a novel, proprietary therapeutic technology platform called DNA-directed RNA interference ("ddRNAi")
- Combines gene silencing techniques of RNAi and gene therapy to deliver sustained, long-lasting silencing of disease-causing genes from a single administration
  - Designed to overcome many of the limitations associated with ongoing administration of siRNA
- Developing a pipeline of ddRNAi therapeutics for the treatment of chronic and life-threatening human diseases
  - Hepatitis C ("HCV")
  - Hepatitis B ("HBV")
  - Age-related macular degeneration ("AMD")
  - Drug-resistant non-small cell lung cancer ("NSCLC")
  - Oculopharyngeal muscular dystrophy ("OPMD")
- Out-licensing technology to a number of companies:

CaliMMune engineering immunity · Circuit Therapeutics · GENABLE · Regen BioPharma, Inc. · uniQure

Clear Strategy To Drive Significant Value



Pipeline: Multiple Shots on Goal

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			Preclinical Proof-of-Concept (PoC) Studies			Clinical Trials		
Focus	Indication	Product Candidate	Discovery	In Vitro	In Vivo	Phase I/IIa	Phase IIb/III	Anticipated Milestones
Infectious Disease	Hepatitis C (GT-1)	TT-034						Efficacy data Q4 2015 Completion of Phase I/IIa trial Q4 2016 Initiation of Phase IIb/III trial Q2 2017
	Hepatitis B	Hepbarna						Completion of *in vivo* PoC study Q2 2016 IND filing Q1 2017 Initiation of Phase I/IIa trial Q2 2017
Ocular Disease	AMD	TT-211						AAV vector developed Q4 2015 Completion of *in vivo* PoC studies Q2 2016 IND filing Q2 2017 Initiation of Phase I/IIa trial Q3 2017
Cancer	Drug-Resistant Non-Small Cell Lung Cancer	Tribetarna						Dose optimization Q4 2015 IND-enabling studies complete Q3 2016 IND filing Q3 2016
Genetic Disease	OPMD	Pabparna						Completion of pre-clinical PoC study Q3 2016

ddRNAi: A Quiet Revolution in Therapeutics

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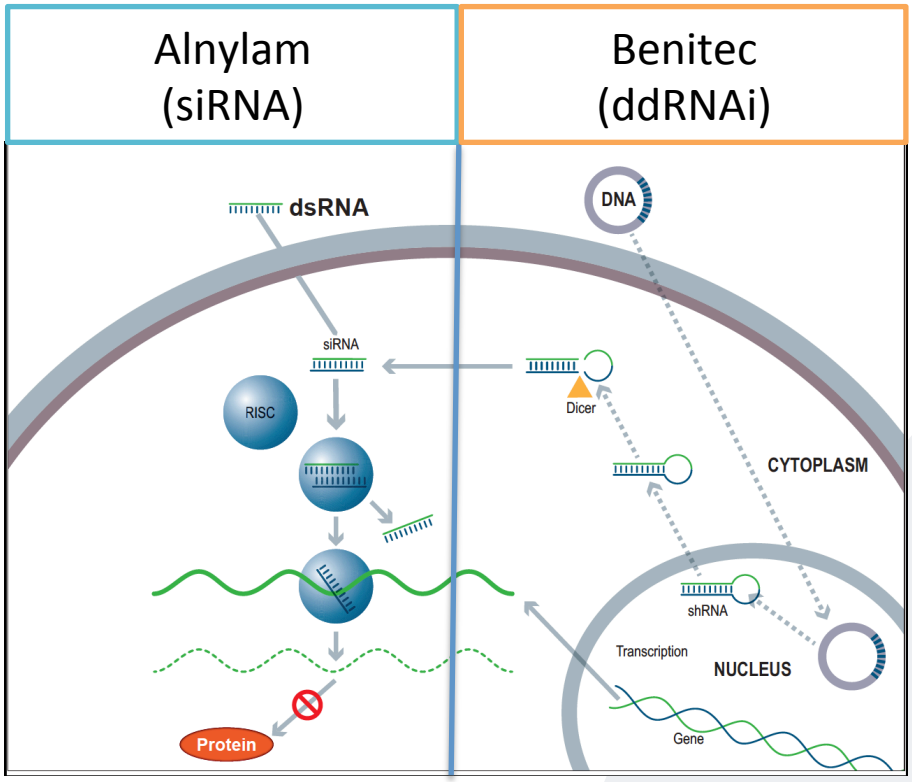
A specific and long-lasting method for turning off disease-associated genes

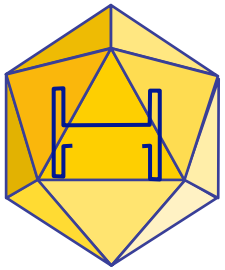
ddRNAi technology designed to utilize the power and specificity of RNAi while avoiding many of its limitations

- Specific delivery to target organs
- Lasting benefits – gene silenced potentially for the life of the cell from a single treatment
- Potential multiple therapeutic effects from a single molecule – can be designed to silence one specific gene, multiple sites on a gene or simultaneously target multiple genes

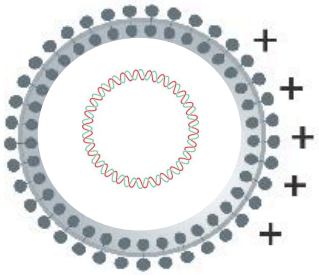
Protected by an international patent estate and rights to intellectual property covering ddRNAi, specific disease targets and product candidates



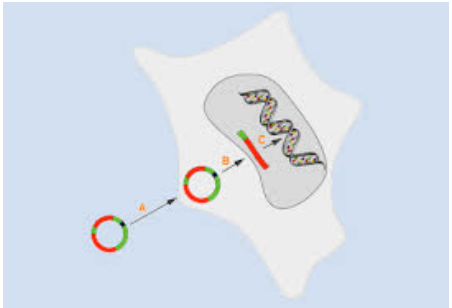
Multiple Delivery Systems Can Be Used to Deliver ddRNAi to Cells



Viral Vectors (AAV, lentivirus)



Nanoparticle delivery of DNA plasmids (jetPEI)



Stem cells transfected with DNA constructs for delivery of expressed shRNA

TT-034: Our Potential One Shot Cure for Hepatitis C

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Hepatitis C Overview

- Hepatitis C is a complex public health problem, characterized by
  - High prevalence of chronic infection by an RNA virus
  - Increasing burden of HCV-associated disease
  - Low rates of testing and treatment
  - Prospect of increasing incidence associated with injectable drug abuse
- According to the WHO, over 170 million individuals worldwide have chronic hepatitis C
- Chronic infection can result in cirrhosis and death in 20% of patients due to end-stage liver disease or hepatocellular carcinoma

Existing Therapies

SOVALDI® SOFOSBUVIR 400 mg TABLETS

Once Daily OLYSIO® SIMEPREVIR 150 mg capsule

HARVONI® ledipasvir/sofosbuvir 90 mg/400 mg tablets

viekira pak™ ombitasvir, paritaprevir and ritonavir tablets; dasabuvir tablets

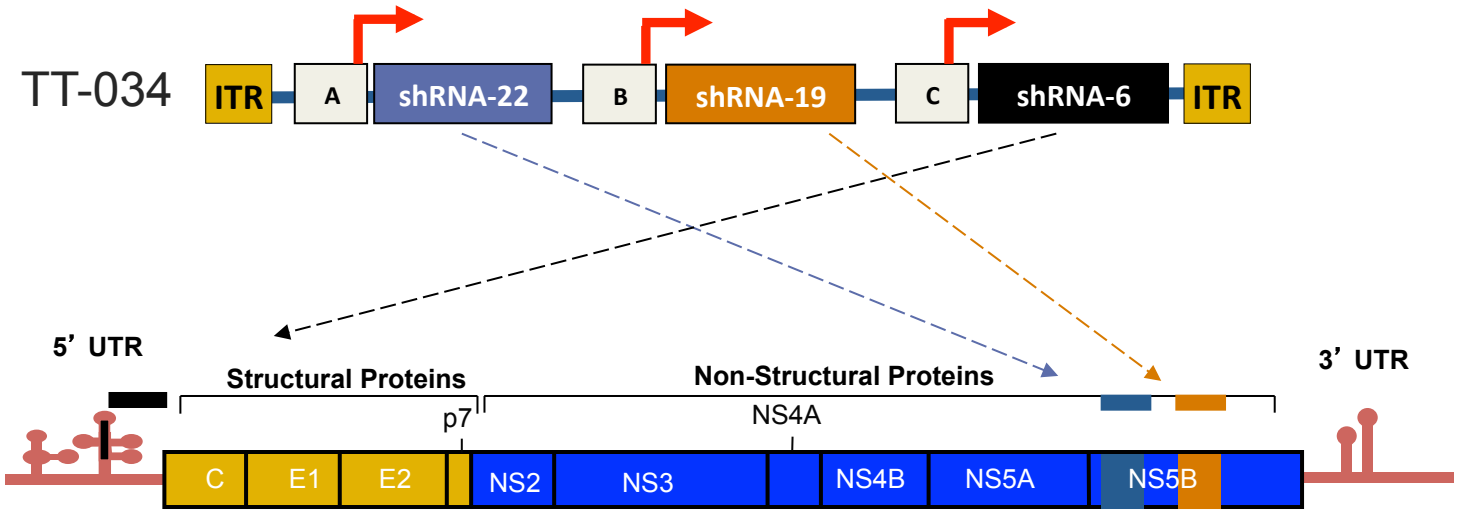
- Therapies require frequent dosing and long treatment durations of 12 to 24 weeks
- Treatments are expensive
- Result in adverse side effects in some cases[1]
  - Fatigue
  - Headache
  - Nausea
  - Insomnia
  - Itching
  - Rash
  - Decreased appetite
  - Diarrhea
  - Photosensitivity reaction
  - Muscle pain
  - Indigestion
  - Weakness
- Lack ability to prevent reinfection

Our Solution

- TT-034 is a ddRNAi-based therapeutic for HCV
  - Goal is to achieve **complete and sustained** elimination of virus with **a single infusion**
  - Produced anti-viral shRNAs for over 180 days from a single administration in preclinical studies
  - Targets three separate, well conserved regions of HCV RNA
  - Potentially **eliminates long treatment courses** and patient compliance issues
  - Potential to **protect against viral reinfection**
  - Potential for combination with oral antiviral drugs for enhanced efficacy
- Program was initially co-developed with Pfizer
- Currently conducting a Phase I/IIa first-in-human clinical trial

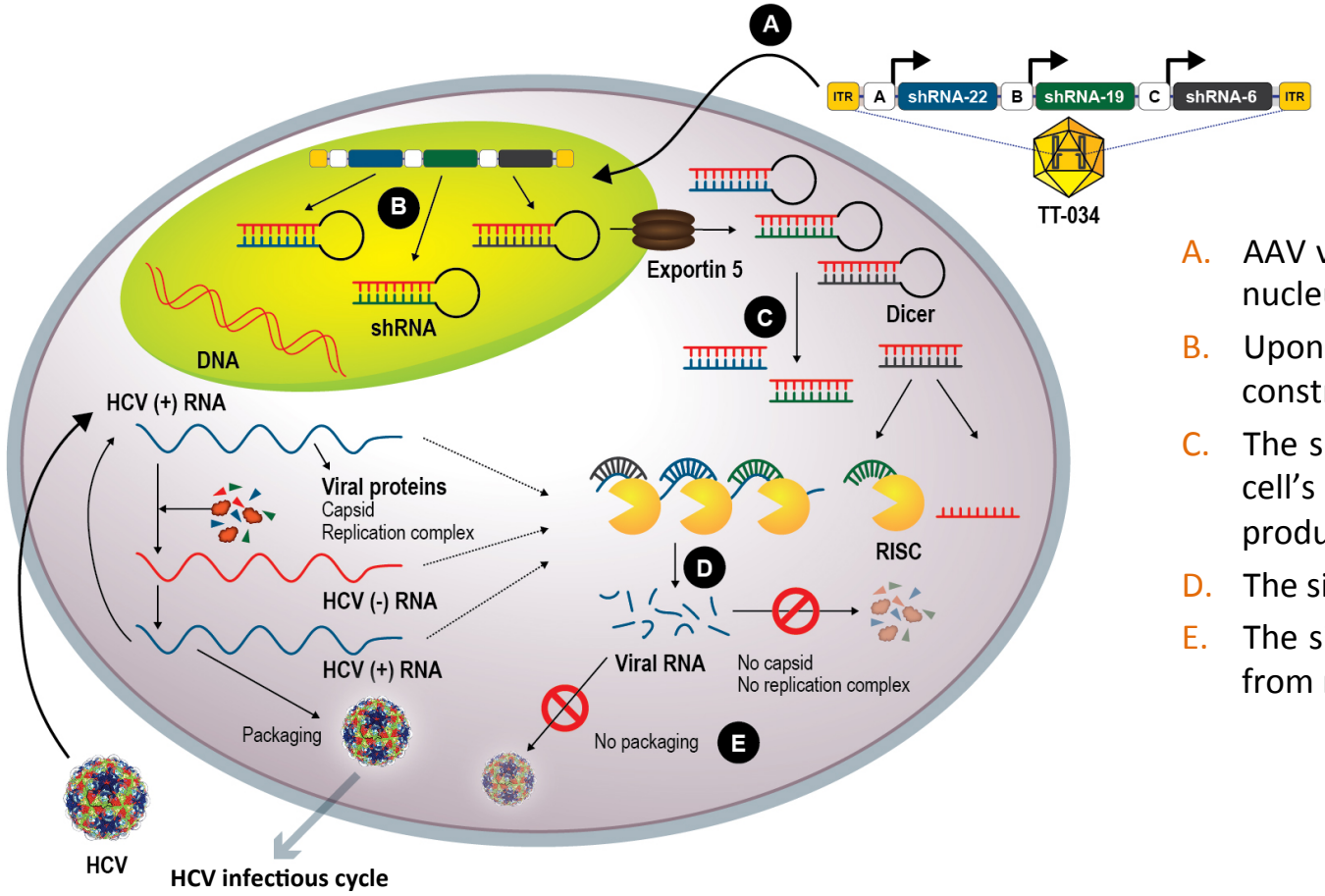
1. All side effects do not apply to each treatment

Design of TT-034



- Three independently transcribed RNAi elements target three separate, well-conserved regions of the HCV genome; **helps prevent the generation of viral escape mutants**
- Expected patent protection to 2025

Mechanism of Action of TT-034 against HCV

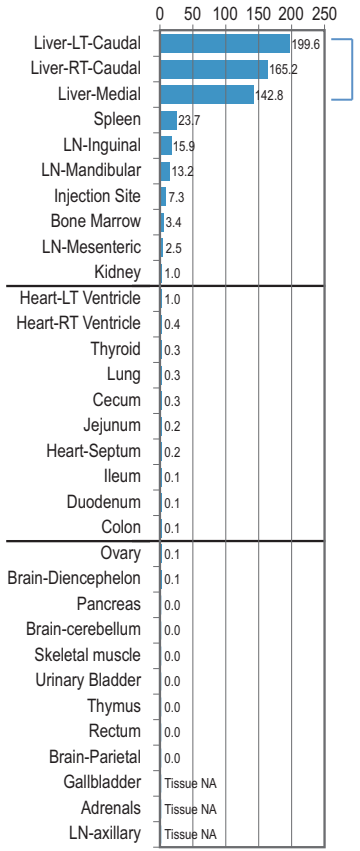


A. AAV vector delivers TT-034 to the nucleus of hepatocytes
B. Upon reaching the nucleus the construct expresses 3 shRNAs
C. The shRNAs are processed by the cell's endogenous machinery to produce siRNAs
D. The siRNAs cleave the HCV RNA
E. The siRNAs also prevent the virus from replicating

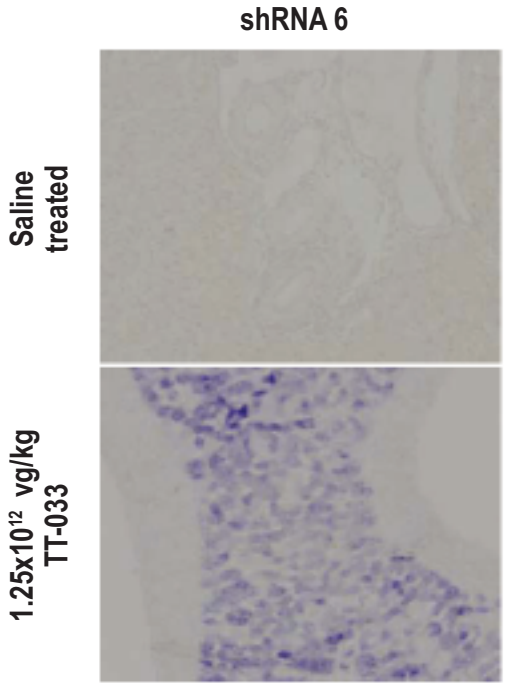
Utilization of the AAV8 Viral Vector Potentially Permits Complete Hepatocyte Transduction

Animal ID: SSAN3
Dose (vg/kg): 1.25E12

Biodistribution Analysis



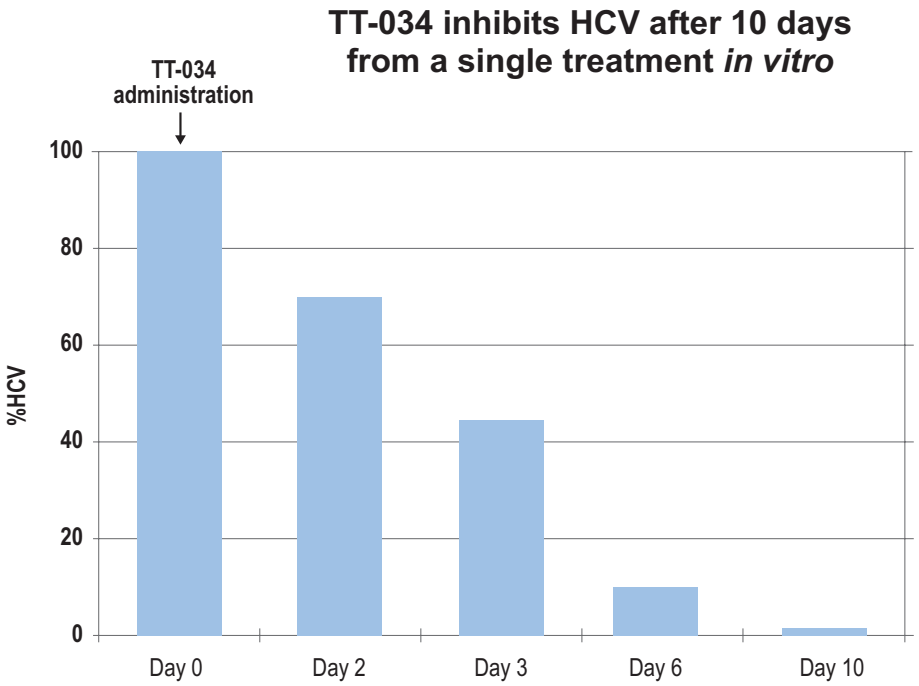
Qualitative In Situ Hybridization

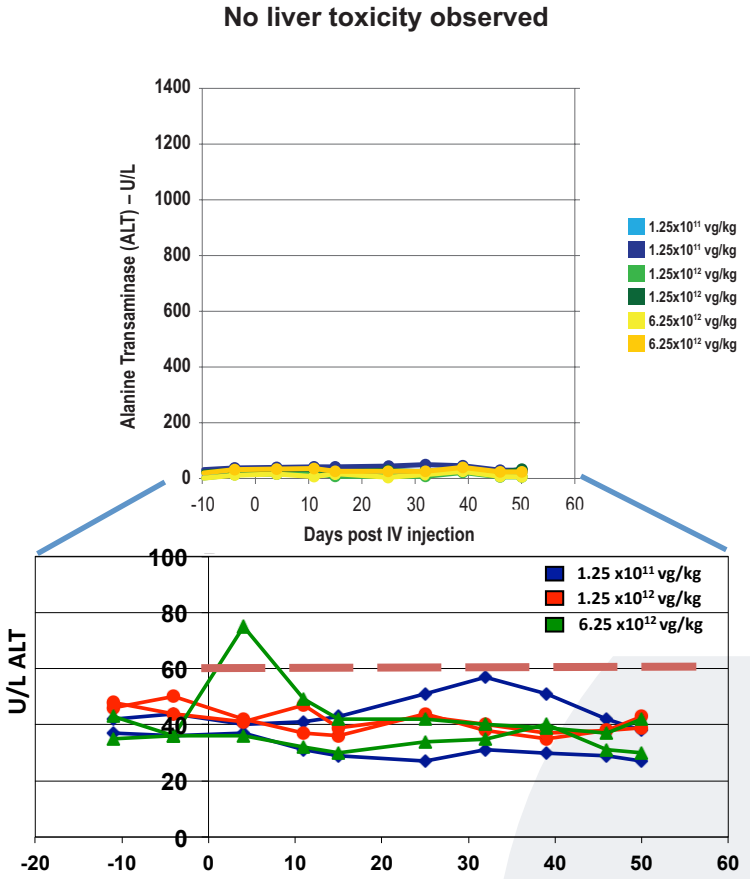


- Sections of 30 day liver tissue were treated with a probe that detects the expression of shRNA6
- Cytoplasmic staining and uniform distribution of the shRNA was seen across nearly 100% of the hepatocytes from an animal treated with TT-033
- No staining was noted in the surrounding vascular wall and perivascular connective tissue within the hepatic parenchyma
- No shRNA staining was noted in the saline-treated animal

TT-034: Pre-clinical Efficacy and Safety

- Clinically relevant doses of TT-034 produced HCV inhibition for 10 days in an *in vitro* model, without toxicity in non-human primates





TT-034 Clinical Trial: Phase I/IIa Dose Cohorts

Open-label dose-escalation Phase I/IIa trial underway:

Cohort	Dose (9vg/kg)	Total No subjects	Dosing scheme for subjects	Observation period per subject and between cohorts
1	4.00×10^{10}	2	Sequential (1+1)	6 weeks
2	1.25×10^{11}	3	Sequential and parallel (1+2)	6 weeks
3	4.00×10^{11}	3	Sequential and parallel (1+2)	6 weeks
4	1.25×10^{12}	3	Sequential and parallel (1+2)	10 weeks
5	4.00×10^{12}	3	Sequential and parallel (1+2)	10 weeks

Trial sites

- Duke Clinical Research Unit, Durham, North Carolina
- University of California, San Diego
- Texas Liver Institute, San Antonio

Reviews

- DSMB review after first patient in each cohort and between cohorts
- Extensive safety monitoring during 24 weeks observation

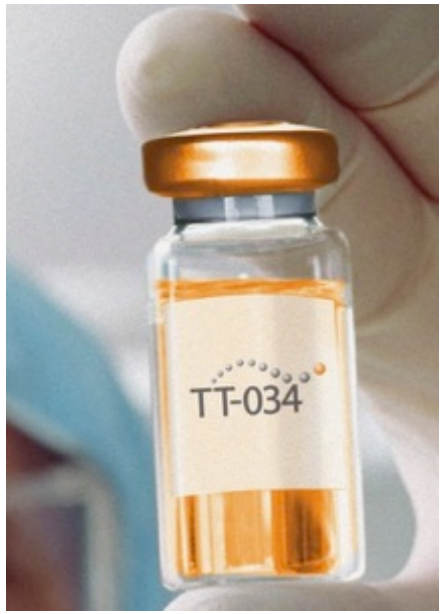
TT-034 Trial Endpoints

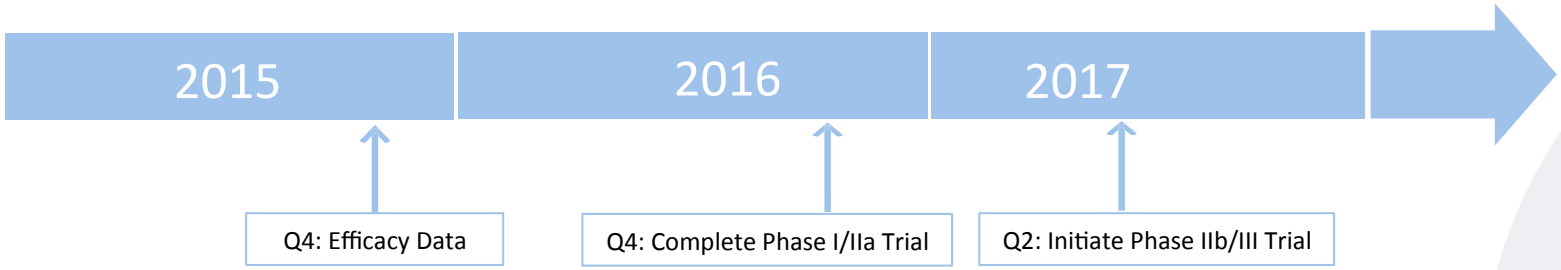
Primary Endpoints (Safety):

- Incidence of adverse events
- Changes in clinical parameters

Secondary Endpoints (Efficacy):

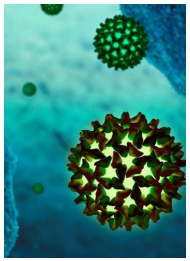
- Sustained reduction in HCV viral load in the blood
- Assessment of TT-034 levels in day 21 liver biopsy
- Assessment of shRNA expression in liver biopsy





Hepbarna®: Our Potential One Shot Cure for Hepatitis B

Hepatitis B Overview



- Hepatitis B is a small DNA virus with an unmet medical need
- 240 million infected worldwide, resulting in up to 780,000 deaths per year
- Patients can suffer in phases ranging from a silent, acute phase that can be resolved by the immune system to a persistent chronic infection requiring life-long therapy
- In chronic HBV, the presence of viral proteins, especially the s-antigen, causes hepatic inflammation leading to liver dysfunction, acute hepatic failure, cirrhosis, or hepatocellular carcinoma
- Hepatitis B virus causes 60-80% of the world's primary liver cancers

Existing Therapies

Zeffix (Lamivudine)

Hepsera (adefovir dipivoxil)

Baraclude (entecavir) 0.5 mg tablets

Viread (tenofovir disoproxil fumarate) 300 mg

PEGASYS (Peginterferon alfa-2a) FOR INJECTION

Interferon

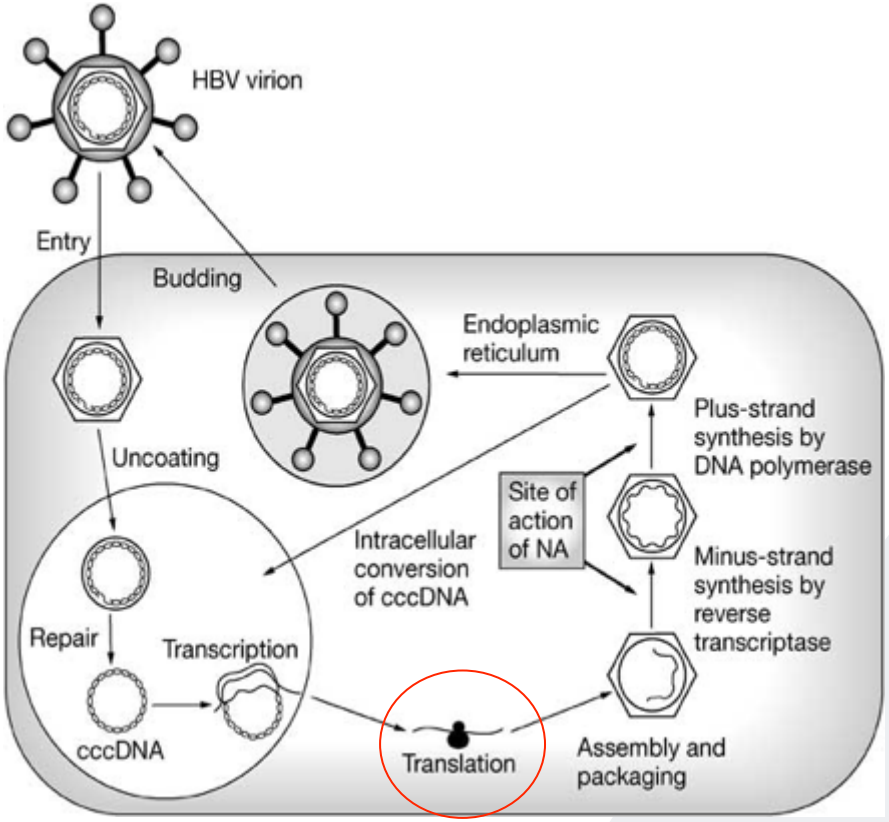
- Therapies require frequent dosing and treatment durations of a year or longer
- Low cure rates and persistence of virus
- Possible development of HBV virus mutations and antiviral drug resistance
- In some cases, Pegasys or Interferon may result in adverse side effects
  - ⚠ Flu symptoms
  - ⚠ Depression
  - ⚠ Nausea
  - ⚠ Vomiting
  - ⚠ Shortness of breath
  - ⚠ Dizziness and fatigue
- Direct anti-virals, cytotoxics and siRNA under development but have treatment limitations

Our Solution

- Hepbarna is a **ddRNAi-based therapeutic** for HBV
  - Goal is to achieve complete and sustained elimination of virus with a single infusion, thereby **eliminating long treatment courses** and patient compliance issues
  - shRNA targets three separate, highly conserved regions of HBV genome
  - **Inhibits both viral replication and viral protein** production, including s-antigen, on long-term basis
- Expect to complete *in vivo* proof-of-concept studies in Q2 2016
  - Hepbarna **leverages HCV approach**
    - Uses same AAV vector as TT-034
    - Similarities between both therapeutics may **enable a faster regulatory pathway** for Hepbarna depending on TT-034 results

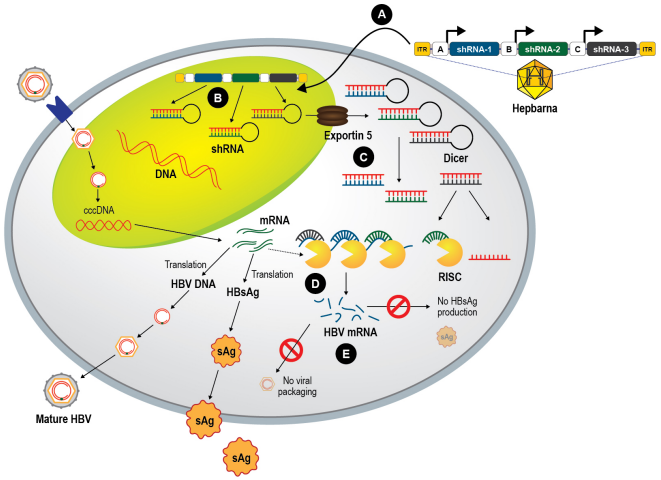
Rationale for RNAi in HBV Therapy

- HBV is susceptible to RNAi because **its RNA produces viral proteins and HBV replicates via an RNA intermediate**
- siRNA needs repeated doses potentially indefinitely
- ddRNAi can potentially provide a single dose treatment to silence HBV mRNA long term



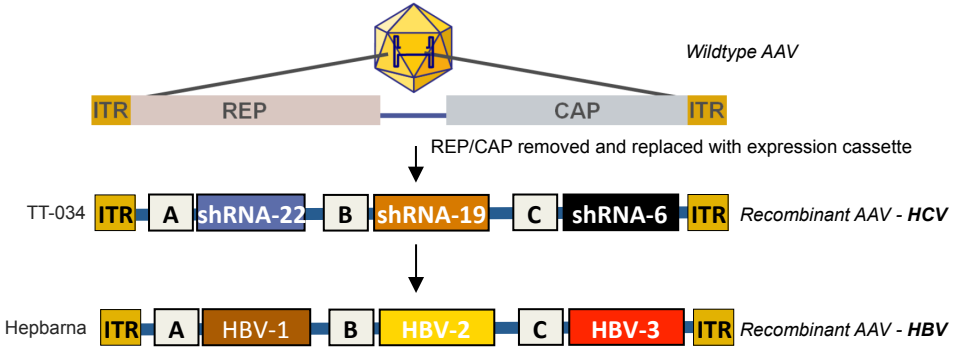
Hepbarna's Mechanism of Action

Mechanism of Action



A. AAV vector delivers DNA construct to the nucleus of hepatocytes
B. Upon reaching the nucleus the construct expresses 3 shRNAs
C. The shRNAs are processed by the cell's endogenous machinery to produce siRNAs
D. The siRNAs cleave the HBV mRNA
E. The siRNAs also prevent the virus from replicating and producing viral proteins including HBsAg

Vector design mimics TT-034



- Keeps the same AAV capsid – designed for identical biodistribution as TT-034
- Keeps the same expression cassette – designed for identical expression properties
- May be able to fast track REG/TOX studies using TT-034 data as part of IND package
- TT-034 clinical data guides HBV protocol development and may provide simpler regulatory path

2016

Q2: Complete *in vivo* Proof of Concept

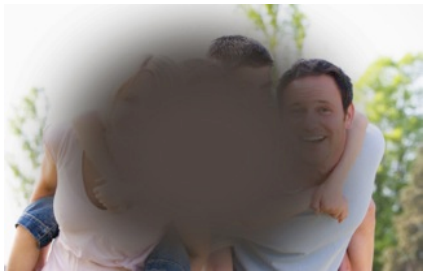
2017

Q1: File IND

Q2: Initiate Phase I/IIa Trial

TT-211 and TT-231: Our Treatment for Age Related Macular Degeneration

Age Related Macular Degeneration Overview



- AMD is the deterioration of the eye's macula, the small area in the retina responsible for central vision
  - Dry AMD: Breakdown of retinal pigment epithelial cells in the macula which can cause profound vision loss
    - Currently no approved treatments
  - Wet AMD: Blood vessel overgrowth stimulated by vascular endothelial growth factor ("VEGF-A") damages the macula and results in vision loss
- AMD is the leading cause of irreversible vision loss in the US – estimated 1.75M people
- Estimated 196 million people will be affected worldwide by 2020
- Age related – 10% of people between 60 and 75 and 25% of people >75 years old

Existing Therapies

LUCENTIS® RANIBIZUMAB INJECTION

AVASTIN® Roche bevacizumab

EYLEA® (aflibercept solution for injection)

MACUGEN® PEGAPTANIB SODIUM INJECTION

- Therapies require frequent intravitreal injection, the risks associated with which include
  - ⚠ Increase in intra-ocular pressure
  - ⚠ Retinal detachment and endophthalmitis
  - ⚠ Inflammation of the eye's internal chambers
  - ⚠ Blood clots

Other Technologies Under Development

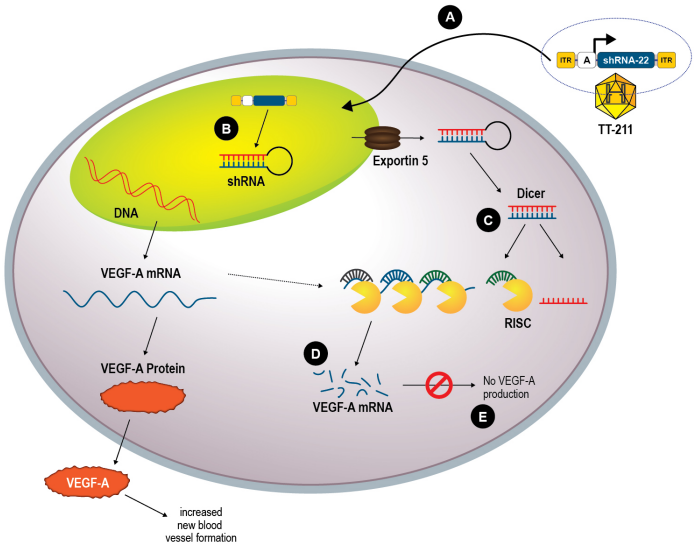
- Gene therapy-based treatments for AMD
  - Approaches deliver genes expressing VEGF inhibitors and other factors that activate new blood vessel formation – do not "cure" the disease
  - Many require subretinal injection with long treatment durations

Our Solution

- Designed to provide sustained **inhibition of VEGF-A from a single intravitreal injection**
  - Less invasive route of administration
- Developing two ddRNAi-based therapies
  - TT-211 is being developed for **wet AMD**
  - TT-231 is being developed for **wet and dry AMD**
- TT-211 is a ddRNAi construct expressing a single shRNA targeting the VEGF-A gene
- TT-231 is a second generation product candidate designed to target three different genes all of which play a role in progression of AMD
- Developing intravitreal delivery vector (AAV) in collaboration with 4DMT

TT-211 and TT-231 Mechanism of Action

Mechanism of Action of TT-211

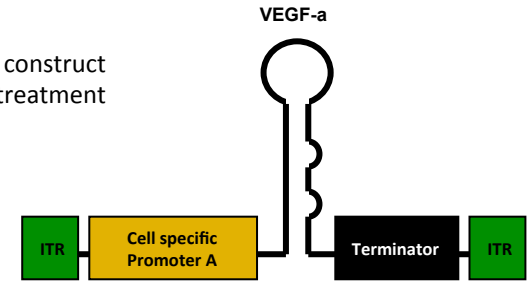


A. AAV vector delivers DNA construct to the nucleus of retinal cells after an intravitreal injection
B. Upon reaching the nucleus the TT-211 construct expresses one shRNA
C. The shRNA is processed by the cell's endogenous machinery to produce siRNAs
D. The siRNAs cleave the VEGF-A mRNA
E. As a result, the VEGF-A protein is not expressed and the stimulation of new blood vessel growth is inhibited

Two Shots on Goal

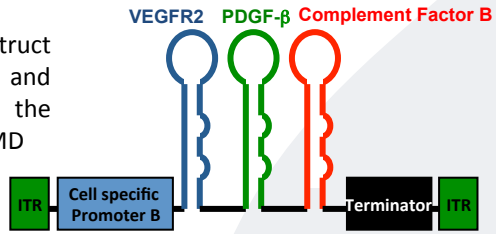
TT-211

A single hairpin ddRNAi construct targeting VEGF-A for the treatment of wet AMD



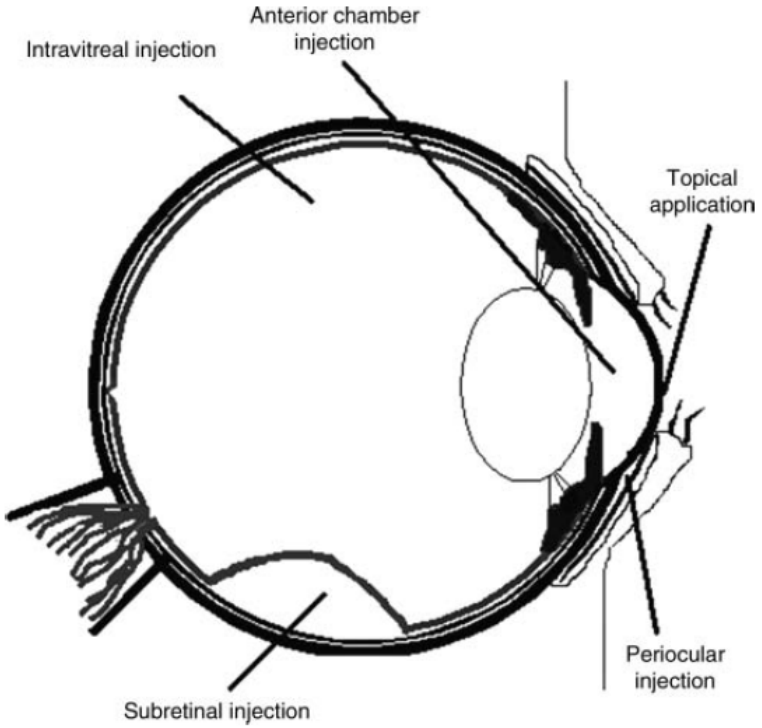
TT-231

A triple hairpin ddRNAi construct targeting VEGFR2, PDGF-β and Complement Factor B for the treatment of wet and dry AMD



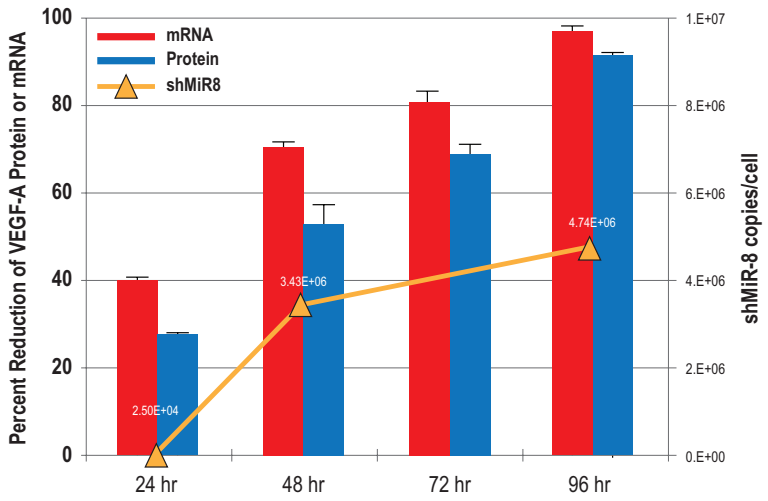
Development of new AAV vector for AMD intravitreal injection

- Benitec has contracted 4D Molecular Therapeutics (4DMT) to identify and develop new AAV vectors with pan-retinal specific targeting from an intravitreal injection
- Benitec has exclusive access to these new vectors from 4DMT for use in ocular ddRNAi applications
- Intravitreal delivery is less invasive and more commercially viable than subretinal injection



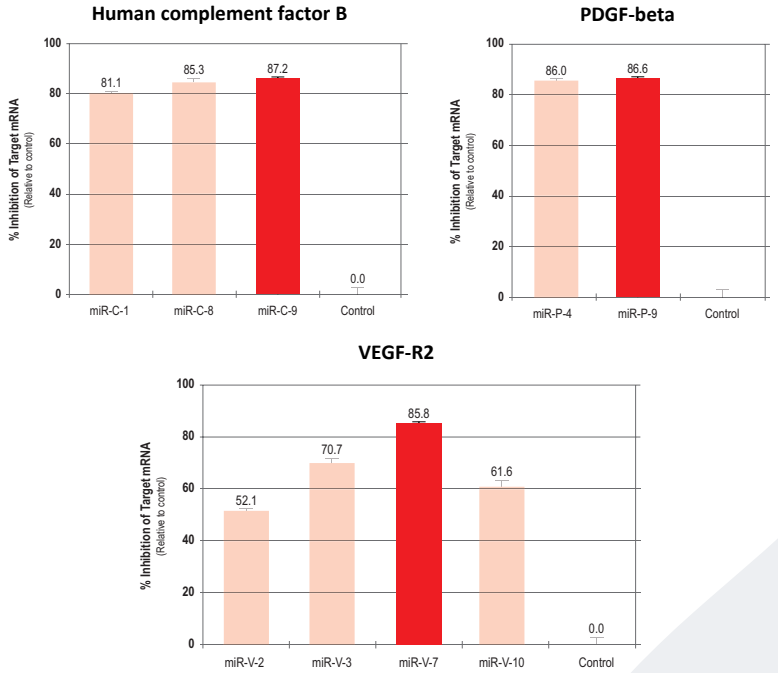
TT-211 and TT-231 Preclinical Highlights and Next Steps

Efficacy of TT-211 on silencing VEGF-A



TT-211 (shMiR-8) inhibited VEGF-A (both mRNA and protein) in retinal pigment epithelial cells *in vitro*

Efficacy of TT-231 on silencing three gene targets



In *in vitro* studies in retinal pigment epithelial cells, TT-231 inhibited target mRNA levels of VEGF-receptor, PDGF-beta and human complement factor B

2015	2016	2017	
Q4: Complete AAV Vector Development	Q2: Complete *in vivo* Proof of Concept	Q2: File IND	Q3: Initiate Phase I/IIa Trial

Tribetarna®: Our Treatment for Drug-Resistant Non-Small Cell Lung Cancer

Non-Small Cell Lung Cancer Overview

- Lung cancer is the most common cancer worldwide
- Lung cancer is the leading cause of cancer-related deaths worldwide (1.3 million deaths per annum)
- The rapid emergence of drug resistant cancer cells provides a major challenge in the treatment of NSCLC
- The efficiency of existing chemotherapeutic agents is restricted by dose limiting systemic toxicity
- A significant opportunity therefore exists for treatments that enhance the effect of therapeutic drugs and are capable of reducing side effect

Existing Therapies

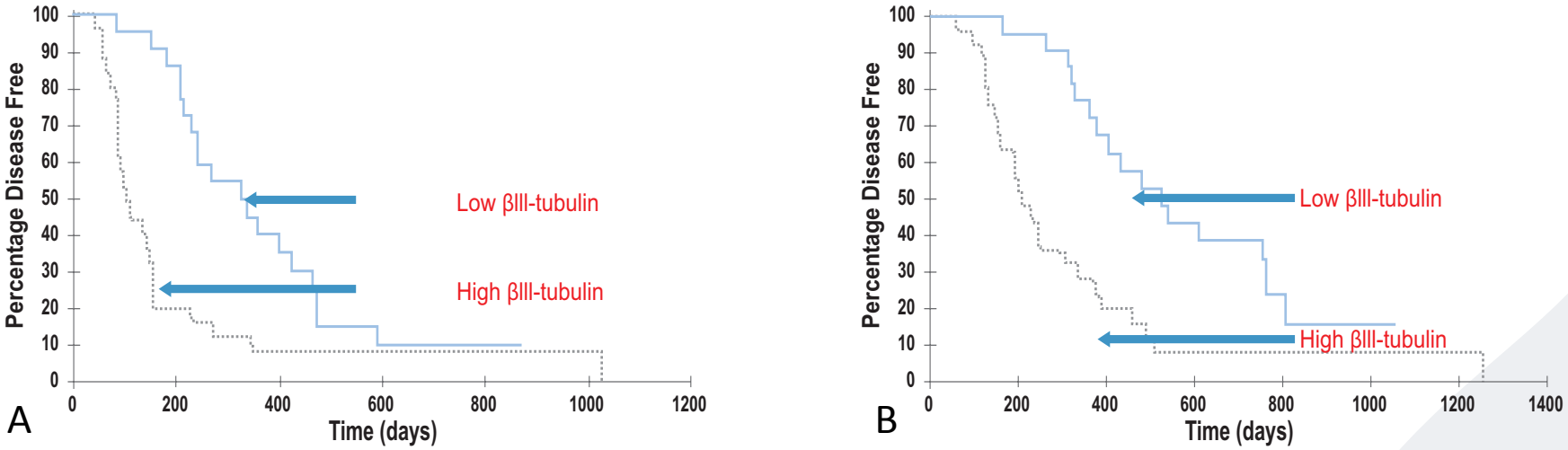
- Chemotherapy combinations are the main treatment approach
- Cisplatin is the mainstay. Combined with taxanes or other DNA damaging agents
  - Nausea
  - Vomiting
  - Diarrhea
  - Loss of appetite
  - Loss of taste
- Rapid development of resistance to chemotherapy limits current treatment options

Our Solution

- Resistance to chemotherapy drugs is strongly associated with over-expression of βIII-tubulin which appears to act as a tumor survival factor
- **Inhibition of βIII-tubulin by RNAi can restore chemosensitivity**
- Tribetarna is a ddRNAi-based therapeutic **targeting three separate regions on the βIII-tubulin gene**
- Administered prior to chemotherapy treatment, it has the potential to **reduce resistance to chemotherapy**, thus making the drug regimen more effective
- Potential to develop Tribetarna for use in **other drug-resistant cancers** with high levels of βIII-tubulin, including pancreatic, renal, breast, ovarian and gastric cancers

βIII-Tubulin Gene is Strongly Associated with Resistance to Chemotherapy in NSCLC

- Up to 70% of NSCLC patients express βIII tubulin in their tumors
- These patients show significantly decreased survival

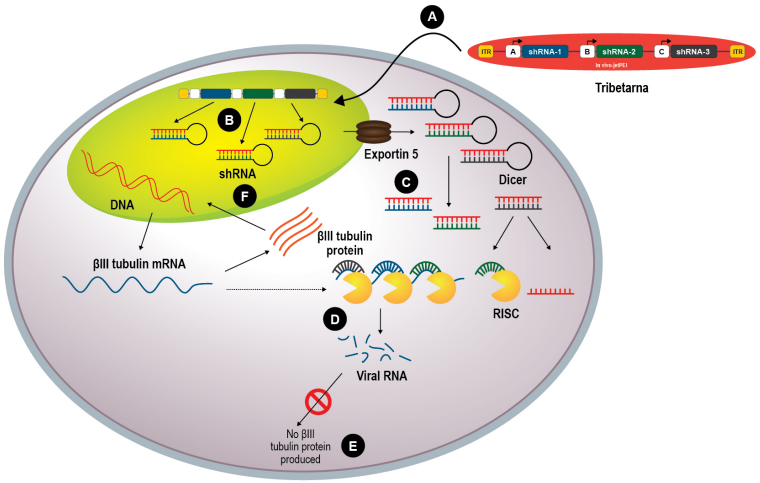


Progression-free survival (A) and overall survival (B) for 47 paclitaxel-treated patients with advanced NSCLC according to class III β-tubulin expression in tumors.
(Seve et al., *Mol Cancer Ther* 2005;4:2001-2007)

Tribetarna® Targets the βIII-Tubulin Gene to Re-Sensitize Tumors to Chemotherapy

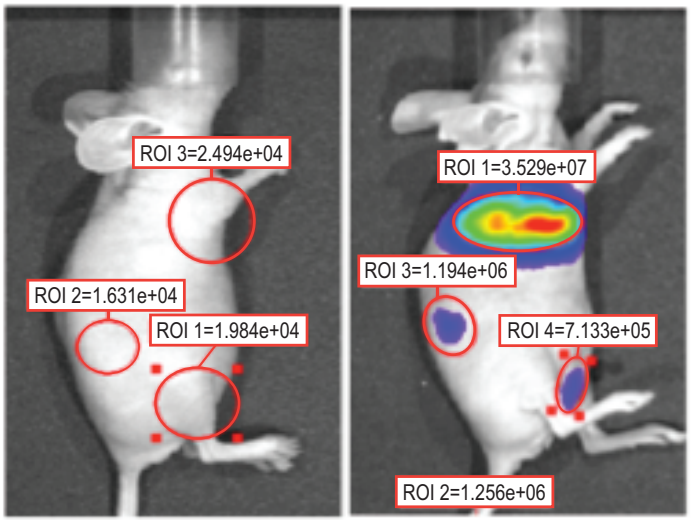
BENITEC BIOPHARMA
silencing genes for life™

Tribetarna Mechanism of Action



A. DNA construct is delivered to the NSCLC cells via in vivo-jetPEI
B. Upon reaching the nucleus the construct expresses 3 shRNAs
C. The shRNAs are processed by the cell's endogenous machinery
D. The siRNAs cleave TUBB3 mRNA
E. The siRNAs also prevent the expression of beta III tubulin protein
F. Thus the protein's effects on modulating the cell's DNA to regulate cancer-associated processes are eliminated

Preclinical Highlights



- jetPEI delivers DNA constructs to lung and lung tumors in vivo from an i.v. injection
- Strong localisation (1000 fold higher) is apparent in NSCLC tumors (right) but not control animals (left)

Tribetarna® Targets the βIII-Tubulin Gene to Re-Sensitize Tumors to Chemotherapy

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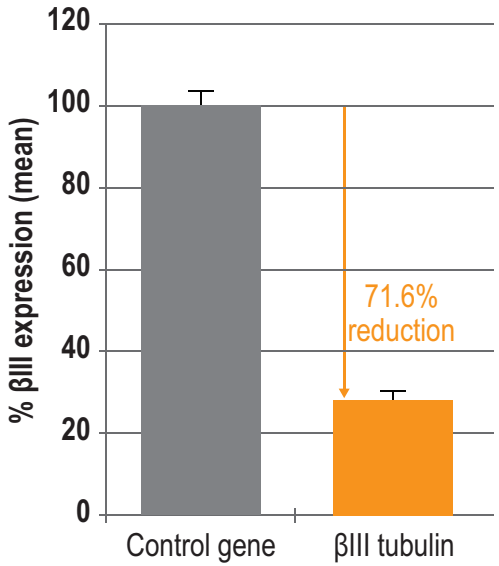
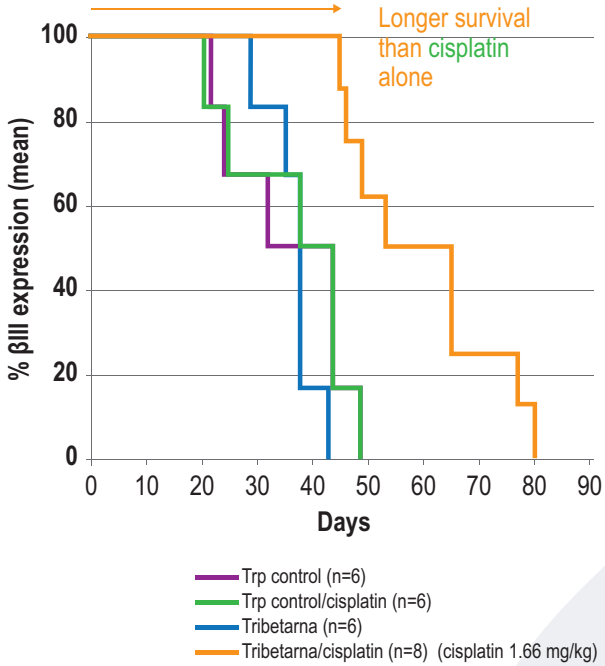
Administration of Tribetarna i.v. provided >70% knockdown of the βIII tubulin gene *in vivo*



Tribetarna significantly enhanced survival in a preclinical model of lung cancer in combination with chemotherapy



2015 | 2016

Q4: Dose Optimization

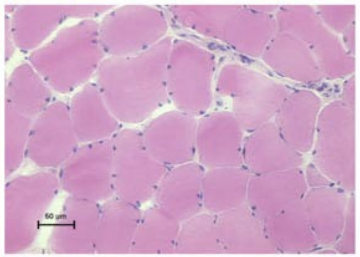
Q3: Complete IND-enabling studies

Q3: File IND

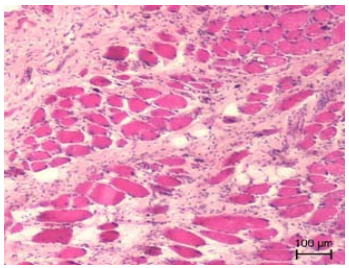
Pabparna™: Our Treatment for Oculopharyngeal Muscular Dystrophy

Oculopharyngeal Muscular Dystrophy Overview

- OPMD is an autosomal-dominant inherited, slow-progressing, late-onset degenerative muscle disorder
- Caused by a specific mutation in the PABPN1 gene
- Characterized by:
  - Progressive eyelid drooping
  - Swallowing difficulties
  - Proximal limb weakness
- Adult onset disease: occurs in patients in 40s or 50s
- Rare disease: 1:100,000 in Europe & 1:1,000 in French/Canadian population



Non-affected



Affected

Existing Therapies

- Cricopharyngeal myotomy is a surgical intervention to improve swallowing but does not correct the progression of the disease

Other Technologies Under Development

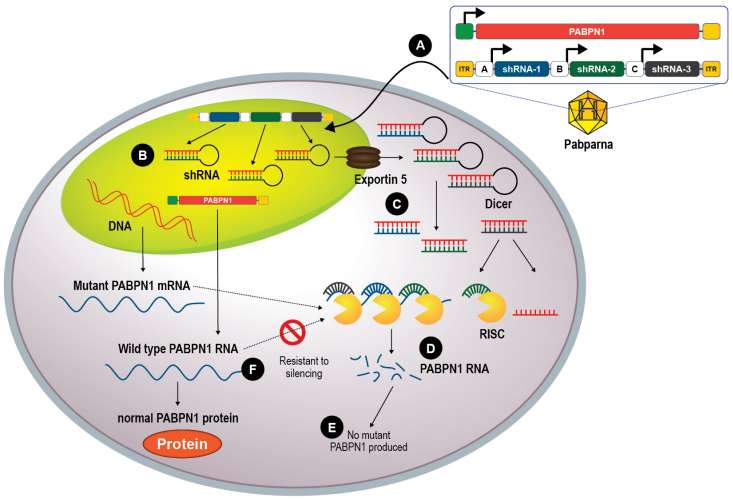
- Cabaletta, a Phase II/III product candidate, is a solution of trehalose administered i.v., and may require ongoing re-administration to remain effective
- Cell transplantation therapy is in Phase I/II and is a highly invasive procedure requiring surgery in two different sites of a patient's body

Our Solution

- Pabparna is a ddRNAi-based therapeutic for OPMD
  - **Monotherapy** delivered using an AAV vector
  - Utilizes a **"silence and replace"** approach designed to silence the expression of mutant PABPN1 gene and replace the mutant gene with the normal PABPN1
- Collaborating with Royal Holloway University of London
  - Conducting studies investigating the optimal delivery of Pabparna
- A successful Pabparna approach may be a **model for other inherited disorders**

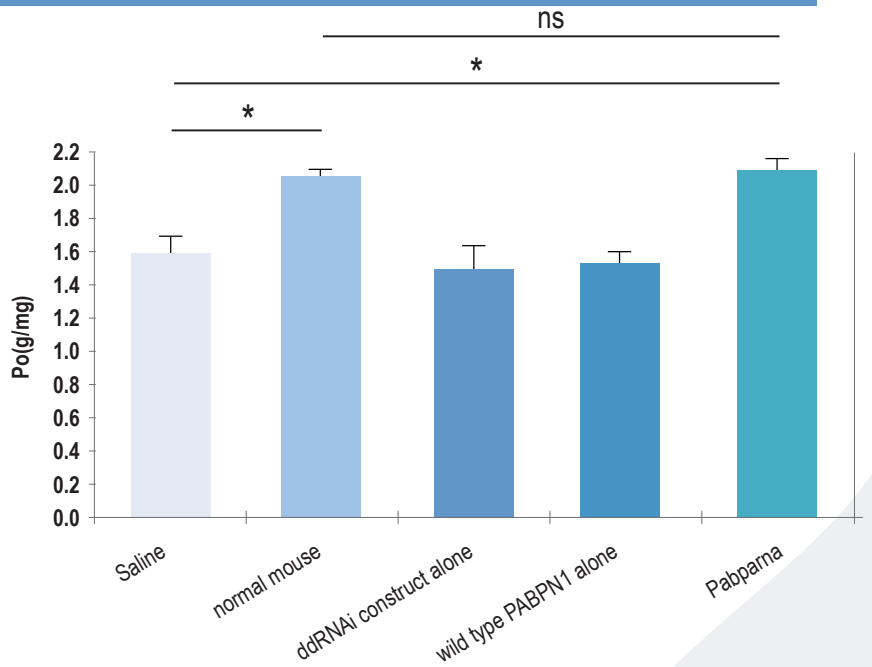
Papbarna™ Mechanism of Action and Preclinical Highlights

Papbarna Mechanism of Action



A. AAV vector delivers the DNA construct to the affected muscle cells
B. Upon reaching the nucleus the construct expresses 3 shRNAs
C. The shRNAs are processed by the cell's endogenous machinery to produce siRNA
D. The siRNAs cleave the mutant PABPN1 mRNA
E. The siRNAs also silences the expression of the mutant gene
F. PABPN1 gene expression construct also expresses a silencing-resistant version of the normal PABPN1 gene, which may promote restoration of muscle function to the cell

Preclinical Highlights



- Restoration of muscle function in vivo following concomitant suppression of the mutant PABPN1 and replacement with the normal PABPN1 gene

CAR T: Immunotherapy

Overview

- Immunotherapy allows using patient's own immune cells engineered to recognize and attack tumors by a process known as adoptive cell transfer
  - T-cells can be genetically engineered to express chimeric antigen receptors (CAR) which enable these cells to recognize antigens on tumor cells
  - Engineered CAR T cells can be reintroduced into the patient, relying on the cells multiplying in the body to recognize and kill cancer cells that present the antigen on their surfaces
- Potential side effect of CAR T cell therapy is cytokine-release syndrome which can lead to high fevers and precipitous drops in blood pressure caused by rapid and massive release of cytokines into the bloodstream
- Improvement to CAR T immunotherapy could involve silencing of multiple genes known to be associated with cytokine release syndrome

Our approach

- Aim to use ddRNAi to silence genes known to be associated with adverse side effects of CAR T therapy
- DNA construct could be designed to allow the expression of several shRNAs targeting the genes of interest to be silenced, along with the CAR antigen gene, in a single construct
  - Cells that are transduced to express the CAR protein would also receive the gene silencing constructs to potentially eliminate related side effects
- Potential to use ddRNAi in this model to also enhance the properties of the engineered CAR T cells

Cell therapies

Overview

- Stem cell therapy market is expected to grow at a compound annual growth rate of 39.5% (reaching $330million by 2020)
- Cell therapy offers a clinical tool to improve, repair or renew the function of damaged or diseased tissue
  - Uses unique properties of stem cells for self-renewal and the ability to differentiate into a range of mature cell types

Our approach

- Aim to use ddRNAi to produce modified stem cells that express shRNA for enhanced therapeutic benefit
- Currently in development in two Benitec programs and one licensee program
- Benitec's OPMD program
  - Collaborating with Royal Holloway University London to develop autologous stem cells that express Pabparna to regenerate new esophageal muscle cells that have the mutant PABPN1 gene suppressed and replaced by the normal PABPN1 gene
- Benitec's NSCLC program
  - Leverages innate ability of stem cells to home to sites of wounding, including cancer
  - Testing the ability of stem cells and exosomes to deliver Tribetarna to lung tumors *in vivo*
  - Potential to be an alternative delivery modality for Tribetarna to target metastatic disease
- Calimmune's HIV/AIDS program
  - Uses patient's own human CD34+ hematopoietic progenitor cells for *ex vivo* transduction with a ddRNAi construct that silences the target gene, CCR5 (HIV co-receptor) gene

Robust Global IP Portfolio

ddRNAi Technology

- International coverage for ddRNAi platform technology
- 30 Granted Patents (in-licensed)
- 9 patent applications (in-licensed)
- Expected expiration: 2019

Additional IP Portfolio

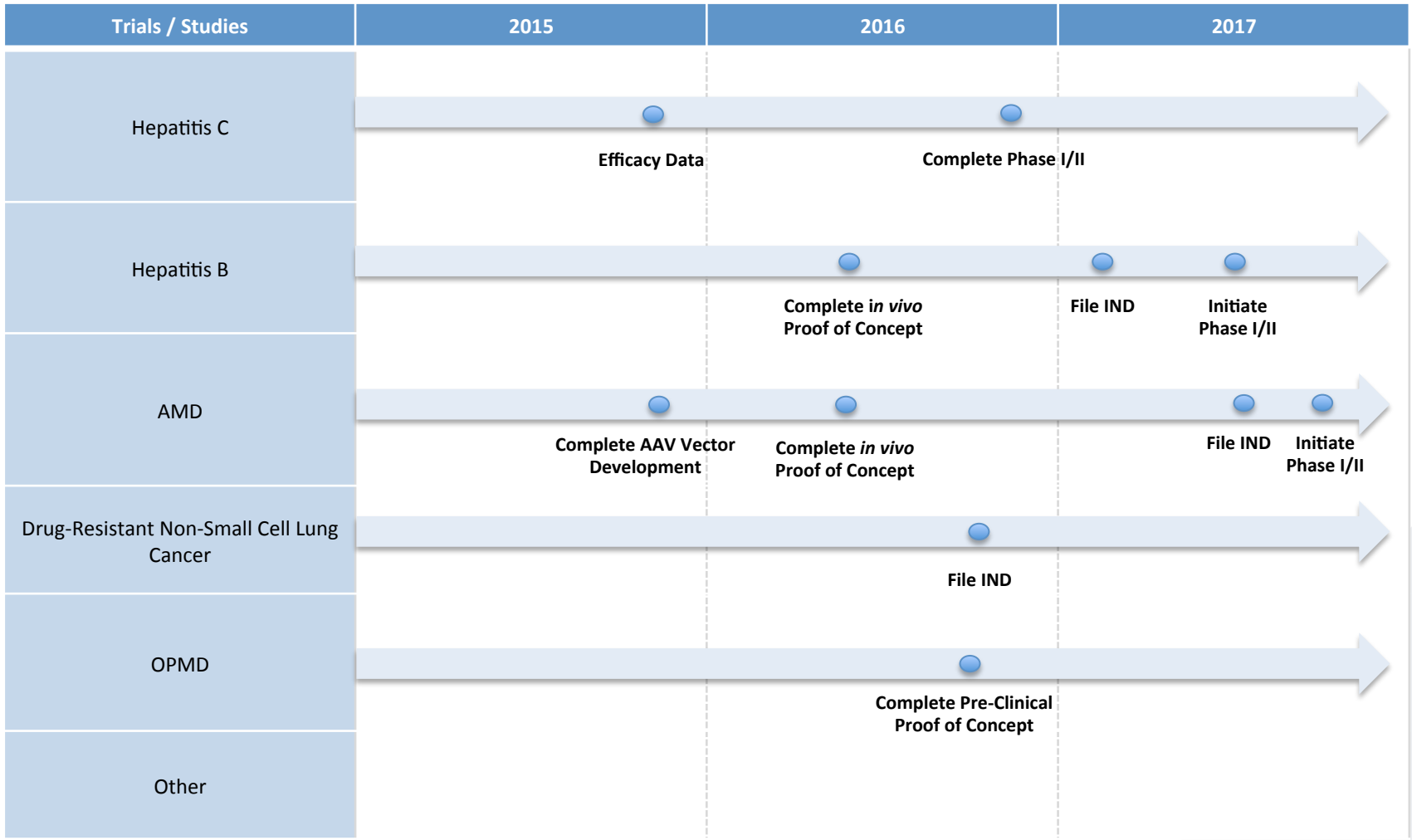
- Target indications, product candidates, technology improvements
- 32 Granted Patents (owned, co-owned or in-licensed)
- 29 Patent Applications (owned, co-owned or in-licensed)
- Expected Expiration for target indications and product candidates at least 2025 and for technology improvements at least 2021

Out-licensed Programs

Clinical Trials

Focus	Indication	Product Candidate	Company	Discovery	Preclinical Proof of Concept	Phase I/IIa	Phase IIb/III
Infectious Disease	HIV/AIDs	Cal-1	Calimmune				
Cancer	Cancer Immunotherapy	dCellVax	Regen Biopharma				
Ocular Disease	Retinitis Pigmentosa	RhoNova	Genable				
Genetic Disease	Huntington's Disease		uniQure				
Central Nervous System	Intractable Neuropathic Pain		Circuit Therapeutics				

Key Milestones



Company Financial Snapshot

Key Financial Details	ASX:BLT OTC: BTEBY
Share Price as of May 15, 2015:	AUD $0.82
Market Capitalization as of May 15, 2015:	AUD $94M
Issued Securities as at August 21, 2014: Ordinary shares Options	 115,218,993 22,695,098
Cash Balance as of December 31, 2014:	AUD $26.8 M

Experienced Board and Expert Partners

Board of Directors	
Dr Peter French *Managing Director and Chief Executive Officer*	Former Chief Scientific Officer, Benitec Biopharma Prior roles at CSIRO, and St Vincent's Hospital Sydney
Peter Francis *Chairman*	Partner at Francis Abourizk Lightowlers Lawyers Former Director, Xceed Capital
Dr John Chiplin *Director*	Director at Cynata Pty Former CEO at Polynoma, Arana Therapeutics, and ITI Life Science Fund; Former Director at Medistem, Inc.
Iain Ross *Director*	Chairman at Biomer Technology Ltd., and Premier Veterinary Group, plc Director at Amarantus Bioscience, Inc., Anatara Lifesciences, and Tissue Therapies Ltd
Kevin Buchi *Director*	President and CEO, TetraLogic Pharmaceuticals Director at Stemline Therapeutics, Inc., Forward Pharma A/S, Alexza Pharmaceuticals, Inc., and Epirus Biopharmaceuticals

Partners and Collaborators

PhoenixBio

ROYAL HOLLOWAY UNIVERSITY OF LONDON

UNSW THE UNIVERSITY OF NEW SOUTH WALES

4D molecular therapeutics

THE PRINCE OF WALES HOSPITAL

Biomics Biotech

cnBio innovations
A member of cn innovations

DukeMedicine

TOUCHLIGHT GENETICS

Investment Highlights

NOVEL GENE SILENCING PLATFORM	ddRNAi combines RNAi with gene therapy delivery to potentially provide "one shot" treatments and cures for a variety of diseases
BROAD PIPELINE	Programs in indications with high unmet clinical need or large patient populations such as hepatitis B and C, lung cancer, and macular degeneration. Key inflection points in 2015 and 2016
CLEAR STRATEGY	Maintain leadership in development of ddRNAi-based therapeutics through clinical proof-of-concept for a range of human diseases
MANAGEMENT EXPERTISE	Significant experience in designing and developing ddRNAi therapeutics; founding scientists in the ddRNAi field
STRONG INTELLECTUAL PROPERTY POSITION	Portfolio of patents, patent applications, and rights to intellectual property directed to our ddRNAi platform and each product candidate

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