Catalyst Biosciences

Exceptional Science. Essential Medicines.

Jefferies Complement Summit

3 May 2016

New York, NY

Forward Looking Statements

This presentation includes forward-looking statements relating to the Catalyst Biosciences, Inc. (the "Company"). Forward-looking statements include statements about the potential markets for the Company's product candidates, the potential advantages of the Company's product candidates, product development plans and timelines, potential safety and efficacy of the Company's product candidates, potential sales of product candidates, if approved, the Company's intellectual property and any statement of belief or assumptions underlying any of the foregoing. These statements reflect the current views of the Company's senior management with respect to future events. Forward-looking statements address matters that involve risks and uncertainties, such as the timing of, costs associated with and outcomes of development, clinical and regulatory activities, risks associated with third-party arrangements, including the risk that Catalyst must negotiate with Pfizer about obtaining manufacturing technology and know-how related to CB 813d, potential adverse effects arising from the testing or use of the Company's drug candidates, risks related to the Company's ability to develop, manufacture and commercialize product candidates, to obtain regulatory approval of product candidates and to obtain marketplace acceptance of product candidates, to avoid infringing patents held by other parties and to secure and defend patents of the Company, and to manage and obtain capital, including through any future financing or the conversion of outstanding convertible promissory notes. Further information regarding these and other risks is included in the Company's Form 10-K for the year ended December 2015 and Form 10-Q for the quarter ending March 31, 2016 filed with the Securities and Exchange Commission on March 9, 2016 and May 5, 2016 respectively, under the heading "Risk Factors".

Catalyst Biosciences : CBIO

Exceptional Science

Essential Medicines

Next generation protease therapeutics

Billion dollar market opportunities

Hemostasis

FVIIa, FIX & FXa

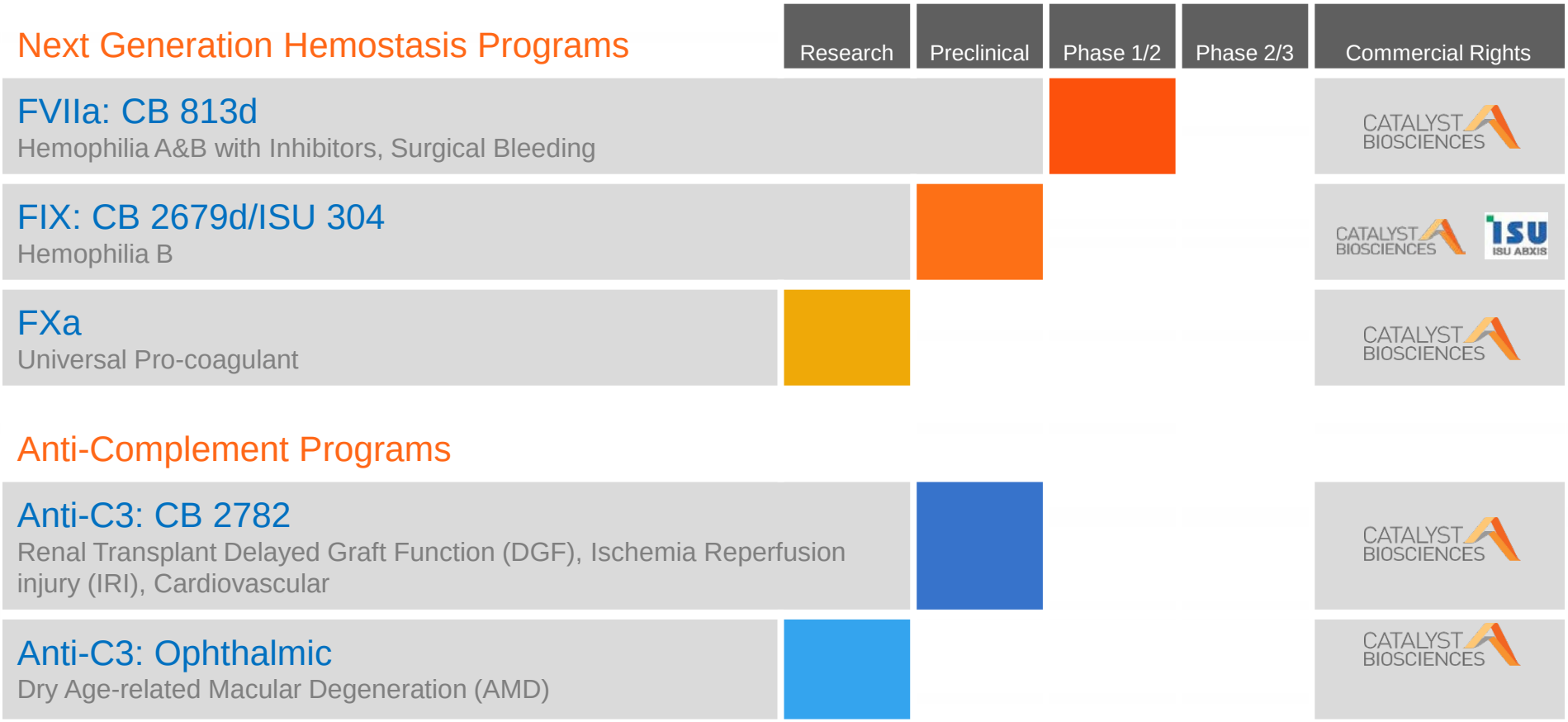
- Current products generate ~$3.3 billion/year in sales
- Catalyst Next Generation products have potential for multibillion/year in sales from growth in prophylaxis, new markets & new indications

Complement

Anti-C3 IRI (DGF) and Eye (AMD)

- Current anti-complement drug (Soliris® - Alexion) generates ~$2 billion/year in sales
- Catalyst Anti-Complement products have critical advantages in multiple new indications

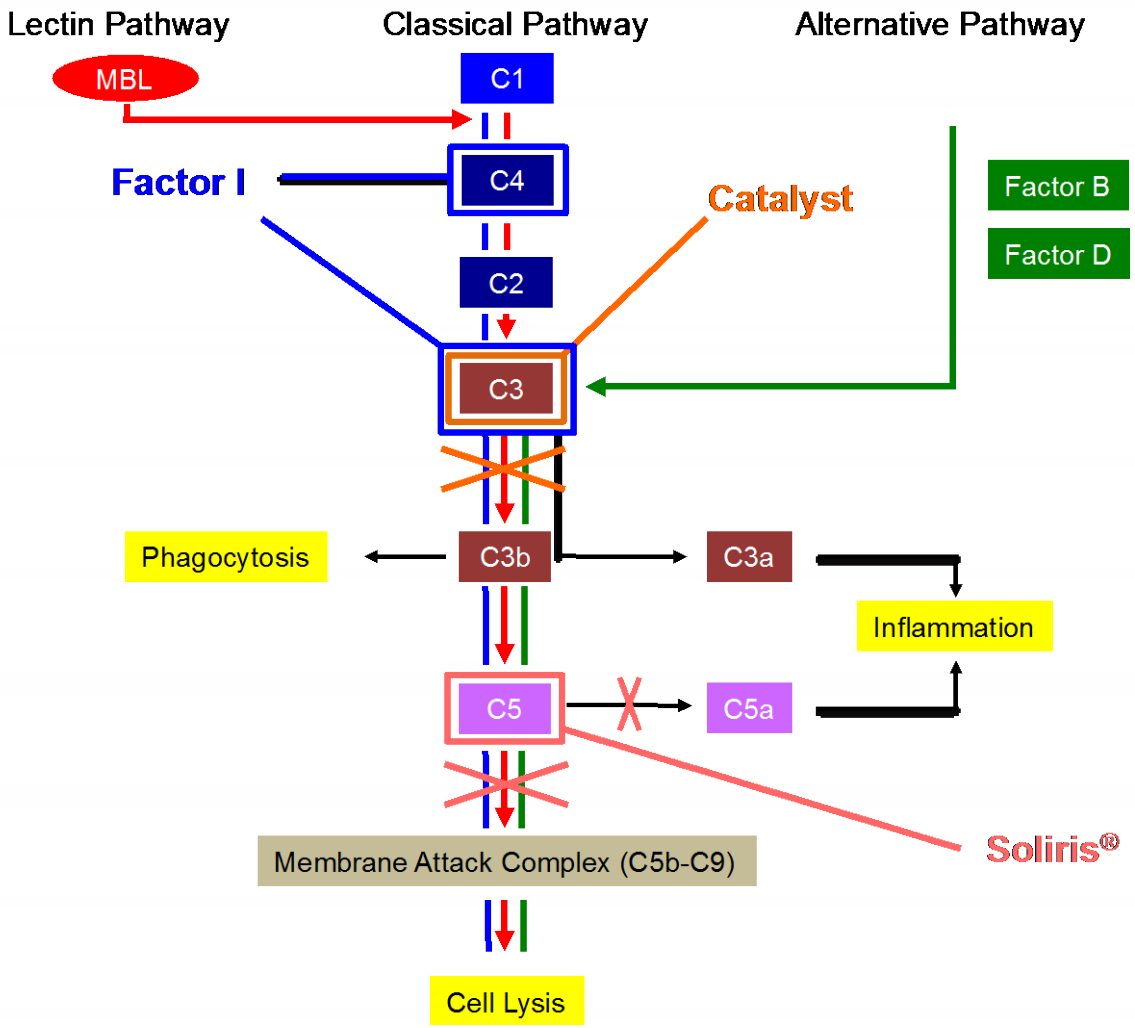
Catalyst Biosciences Pipeline



Anti-Complement Opportunity

- Complement targets are biologically & clinically validated
  - Knock Out (KO) mice studies
  - Human genetics
  - Approved drug (Soliris®) for Paroxysmal nocturnal hemoglobinuria (PNH) & Atypical hemolytic uremic syndrome (aHUS)
  - Positive P2 data for Dry Age-Related Macular Degeneration (AMD) – Geographic Atrophy (GA)
- Complement has been implicated in a very large number of acute and chronic therapeutic indications
- Multiple acute indications mediated by complement driven ischemia reperfusion injury
  - Initial indication – anti-C3 to prevent Delayed Graft Function (DGF) following kidney transplant
  - Cardiovascular: CABG, MI & Stroke (label expansion potential for an IRI drug)
- Chronic indications
  - Initial indication – anti-C3 (ocular) to slow the progression of GA in Dry AMD

Complement Cascade: Ideal Application for Catalyst's Protease Platform



Competitive advantages

- Proteases are a fundamentally better approach than antibodies or small molecules
- Mimics nature's solution
- Efficiently inactivates cascade amplification
- 1 protease drug molecule can efficiently inactivate several hundred target molecules/hour
- Anti-C3 prevents release of pro-inflammatory mediators (C3b) and anaphylotoxins (C3a) that anti-C5 cannot
- Potential to inactivate *any* target (especially high concentration ones)

Proteases – Best Approach in Anti-Complement

- Acute Systemic (DGF, MI, Stroke, CABG)
  - Catalytic activity allows for the rapid and complete depletion of 8 μM C3, a key target that is likely intractable with either small molecules or MAbs
  - Development Candidate selected
- AMD
  - All large molecules are cleared at a similar rate in the eye
    - Catalytic biologics will maintain effective antagonism at concentrations substantially below the target concentration
    - Greatly enhanced duration of action of Novel Proteases should allow for significantly decreased dosing frequency
- Both
  - C3 inhibition blocks all arms of the complement cascade and prevents formation of anaphylotoxins and other pro-inflammatory mediators as well as the membrane attack complex (MAC)

Anti-C3 for Dry AMD

- Advanced dry AMD, or geographic atrophy (GA), leads to loss of RPE photoreceptors, blindness
- No approved drugs
- Global wet AMD market is >$4 billion annually
- GA prevalence is equivalent to wet AMD
- Strong genetic evidence for complement in pathogenesis of dry AMD*
- Complement is the only validated anti-GA target
  - Roche anti-Factor D antibody @ 10 mg/eye intravitreal injection showed 20-44% inhibition of GA progression with monthly dosing; dosing every 2 months failed

- C3 is the “best target” in the complement cascade (recent AAO* plenary lectures)
  - Targeting either individual pathways, *e.g.*, alternative (Factor D) or C5 appears to be “leaky”, consequently limiting efficacy
- Efficient inactivation of C3 expected to provide greater efficacy compared with competing anti-Factor D or anti-C5 strategies (AAO)
- Less than 40% inhibition of GA progression over 18 months would be acceptable if dosing was less frequent than monthly (AAO)
- Catalytic turn-over of target expected to support efficacious dosing every 2 months or less frequently

*_Science_, April 2005; _JAMA_, July 2006; _NEJM_, July, 2007

*AAO – American Academy of Ophthalmology 2015

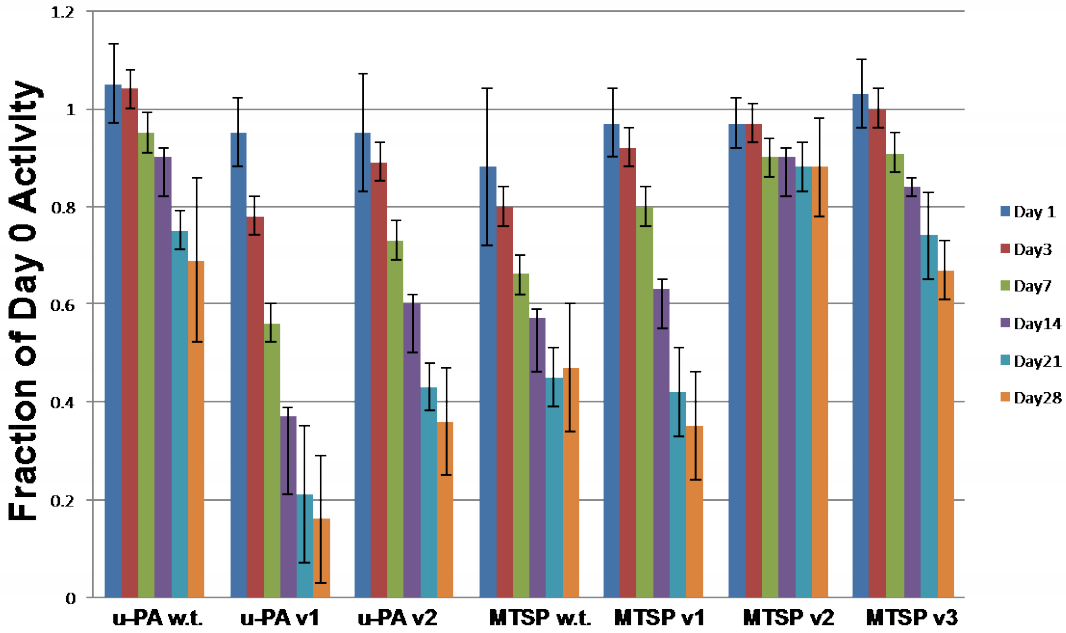
Catalyst Dry AMD (GA) Program

- Use proprietary selection/counter-selection technologies to discover and optimize orthogonal anti-C3 leads based on two distinct human serine proteases
  - u-PA (approved cardiovascular drug)
  - MTSP-1

C3 Inactivation & Stability in Vitreous Humor

- Catalyst anti-C3 AMD protease candidates have been optimized for the ocular environment
- 1 anti-C3 AMD protease molecule can efficiently inactivate several hundred target molecules/hour in non-human primate vitreous humor
- Current leads are stable in human and primate vitreous for >4 weeks
- Strong potential for less frequent than 1x/month administration

Anti-C3 Protease Stability in Cynomolgus Vitreous Humor



Anti-C3 Dry AMD Program: Toxicity Study

- Comprehensive, non-human primate single dose escalation ocular safety/toxicity study completed for advanced, MTSP-1 & uPA based leads
  - Three intravitreal doses (12.5, 37.5, or 125 µg/eye) with three animals (cynomolgus monkeys) per dose (no re-dosing)
  - Right eye received test article; left eye injected with vehicle control
  - "Clinical observations", food consumption, *etc*.
  - Ophthalmic examinations: slit-lamp biomicroscopy and indirect ophthalmoscope observations, followed by color fundus photography or optical coherence tomography (OCT) prior to dosing and on days 2, 8, and 15 post-dosing
  - Any observations followed for up to 4 weeks until resolution

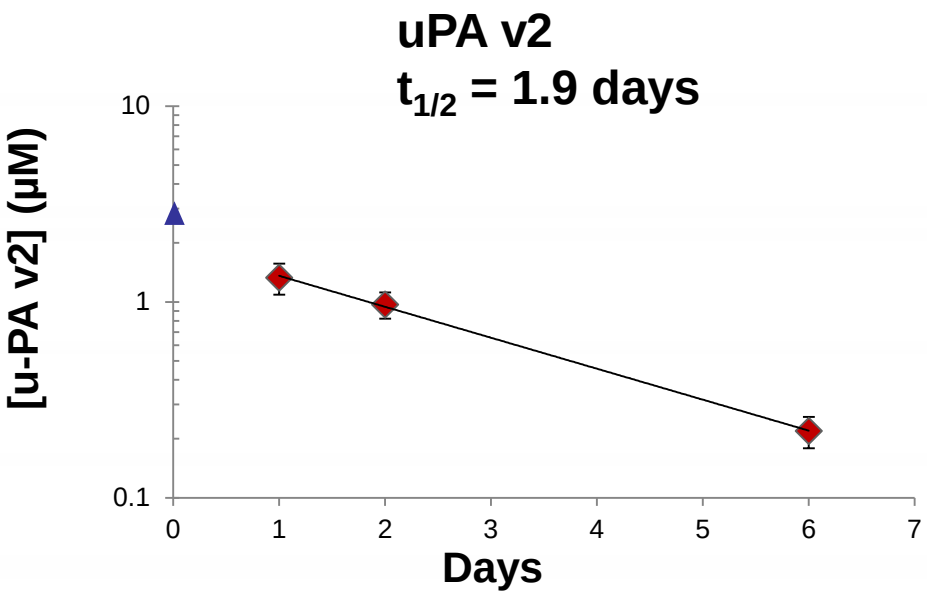
NHP Ocular Toxicity/Tolerability Study

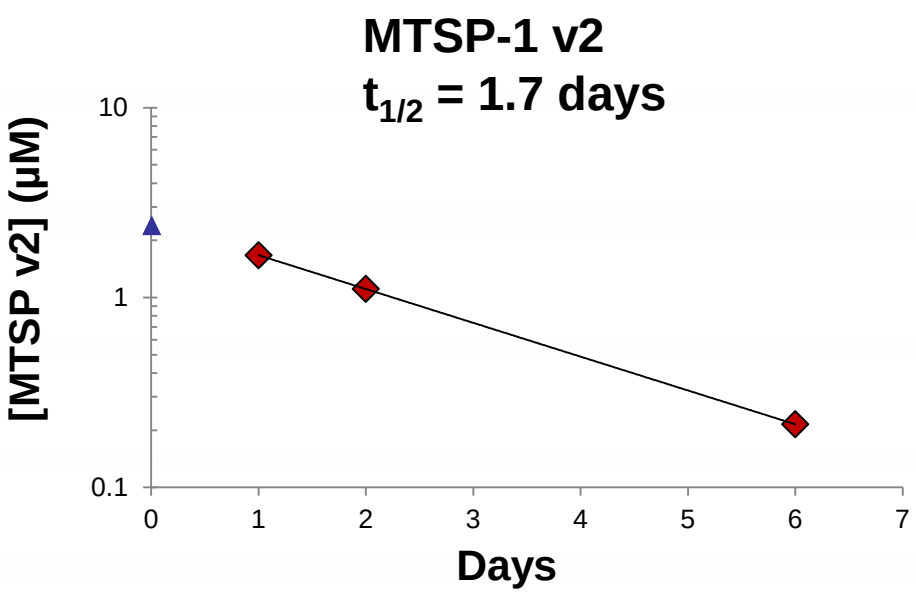
- MTSP Variants:
  - No observations in animals injected with MTSP Variant 2
  - NOAEL of MTSP Variant 1 ≥37.5 µg (higher than Jetrea® in same model)
  - NOAEL of MTSP Variant 2 determined to be ≥125 µg (no observations)
- uPA Variants:
  - No observations for animals injected with u-PA Variant 2
  - NOAEL of u-PA variant 1 found to be ≥37.5 µg (higher than Jetrea® in same model)
  - NOAEL of u-PA variant 2 found to be ≥125 µg

AMD Program – Ocular PK/PD study

- One week Cynomolgus monkey PK/PD study of MTSP Variant 2 and u-PA Variant 2 by intravitreal injection has been completed
- Study design:
  - One dose: 125 µg of test article in right eye, vehicle injection in left eye
  - 12 animals injected per variant
  - Four animals per variant sacrificed at each of the following time points: Day 1 (24 hours post-dose), Day 2, Day 6
  - Vitreous humor collected and analyzed for variant concentration, variant activity, and C3 concentration
- Results:
  - Undetectable levels of C3 (<10 pM) in variant-treated eyes at all time points
  - Variable levels of C3 (0.4 – 50 nM) in vehicle-injected eyes (2 outliers - probable blood contamination of vitreous)
  - Half-life of MTSPv2 ~2 days by both ELISA and activity, estimated *in vivo* recovery ~100%
  - Half-life of u-PAv2 ~2 days by ELISA, 1.5 days by activity, estimated *in vivo* recovery ~80%

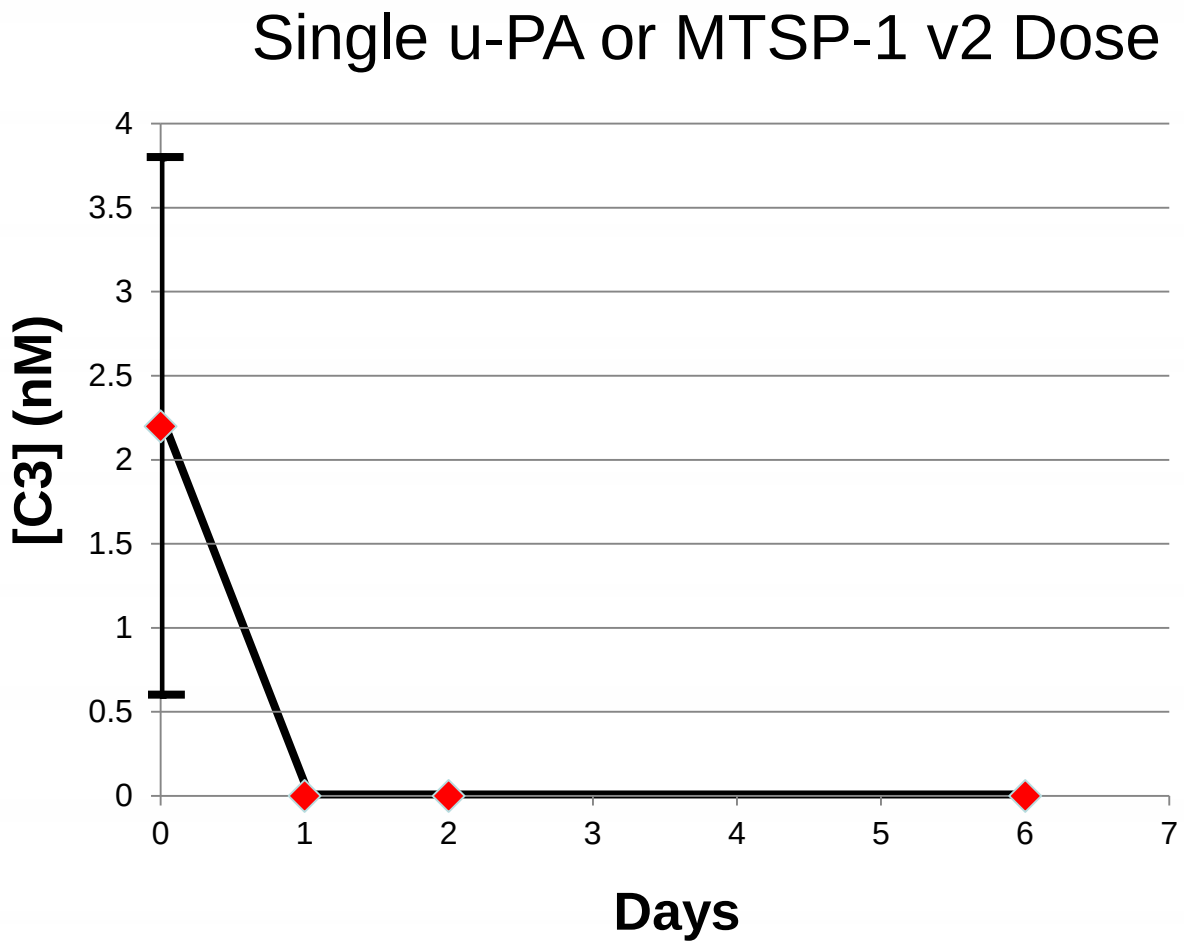
Cynomolgus Intravitreal PK by ELISA





▲ = predicted value at 100% in vivo recovery (2.5 µM)

Cynomolgus Vitreous Pharmacodynamics



Day 0 = Pooled, vehicle treated eyes

C3 levels were below the limit of detection (20 pM) in all dosed eyes through day 6

AMD Program – Duration of Action Study

- Calculated $t_{1/2}$ similar to that reported by Genentech for Lucentis (Fab) – 3 day $t_{1/2}$ calculated but 50% recovered in serum in 2 days
- Calculated $t_{1/2}$ of Lucentis in humans (systemic sampling after intravitreal injection) was 9 days
- Expected protease dosing frequency (at current dose) to maintain complete inhibition of C3 based on following human $t_{1/2}$:

[$t_{1/2}$]	[Expected Dosing]
2 days	every month
3 days	every 2 months
4 days	every 2-3 months
6 days	every 3-4 months
9 days	every 5-6 months

} Current range based on Lucentis extrapolation of Cynomolgus to human $t_{1/2}$

AMD Program Summary

- Convincing clinical and pre-clinical evidence that complement is an important dry AMD target
- C3 inhibition comprehensively down-regulates all three pathways of complement activation, inhibiting C3a/C5a and membrane attack complex (MAC) production
- Catalyst development leads expected to be differentiated from antibody and small molecule competitors
  - Catalytic turnover of target expected to allow significantly less frequent dosing in dry AMD
- Catalyst leads are potent, stable, and appear to be well tolerated in a NHP model
  - Anti-complement leads inactivate ~5 to >1100 human C3 molecules per hour
  - Anti-complement leads for dry AMD maintain ~30 to 90% activity following incubation at 37º for one month in vitreous humor
  - 4/4 compounds tested display higher NOAEL than Jetrea
  - Early PK/PD studies suggest that bimonthly dosing or less frequent than bimonthly dosing is feasible in humans

Anti-C3 for Renal Delayed Graft Function (DGF)

- High unmet medical need: DGF occurs in 20-30% of all cadaveric allograft recipients
- Multiple animal studies have established key role for complement in ischemia reperfusion injury
- Fast, straightforward development:
  - P1 in normal volunteers as a bridge to P2
  - P2 Endpoint: dialysis within 1 week of transplant
- Patient and payer benefits:
  - Reduced dialysis, shorter hospital stays
  - Reduced acute rejection, extended graft life
- DGF annual market opportunity of >$500M/year*
- Gateway to billion $ MI, CABG & stroke markets

*Health Advances Market Evaluation 2015

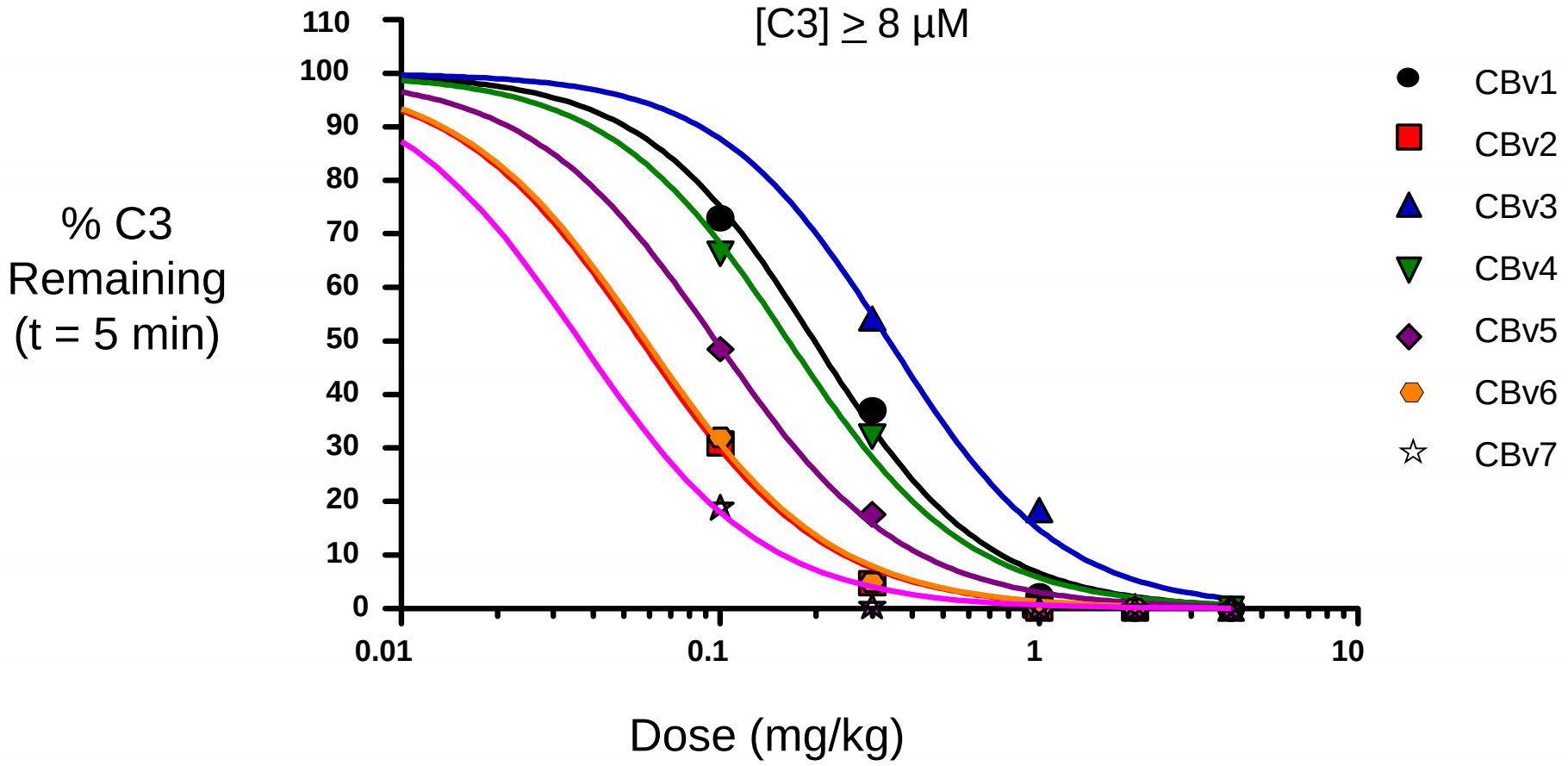
Anti-C3 Proteases in Non-human Primate Model

Variant	Scaffold	C3 Destruction ED_{50} (mg/kg)	Top Non-Toxic Dose (mg/kg)	Single Bolus T.I.
CB 2470	MTSP	0.2	≥2	~10
CB 2561	MTSP	0.1	≥2	~20
CB 2558	MTSP	0.2	≥4	~20
CB 2750	MTSP	0.06	≥1	~17
CB 2782	MTSP	0.07	≥4	>57
CB 3064	MTSP	0.04	≥2	>50
CB 2963	u-PA	0.3	≥4	>13

Anti-C3 Proteases in Non-human Primate Model

CATALYST BIOSCIENCES

Single dose pharmacodynamics



Acute Anti-C3 DGF Summary

- Catalytic activity of Catalyst's anti-C3 proteases allow complete antagonism of 8 μM systemic C3, a target likely intractable with either small molecules or MAbs
- Inhibition of C3 prevents both the release of anaphylotoxins and C3b, and formation of the membrane attack complex (MAC)
- Catalyst's anti-C3 proteases preserved heart function in an *ex vivo* Langendorff model at 700-fold lower drug/target ratio than an anti-C5 MAb
- Single injection of Catalyst's anti-C3 proteases completely inhibited complement contribution (~50%) to injury in a murine hind limb ischemia/reperfusion model
- Lead anti-C3 Development Candidate (CB 2782) is potent, well tolerated in non-human primate model with a therapeutic index >50

Catalyst Biosciences Investor Highlights - CBIO

- Clinical Stage Public Protease-Based Hemostasis and Anti-Complement Company
  - Improved, second generation proteases: FVIIa & FIX in clinical trials
  - Proprietary platform that creates novel proteases: Anti-complement
- Highly differentiated, clinically-validated anti-complement approach to multi-billion dollar Dry AMD market
- Novel, anti-complement orphan program (renal transplant delayed graft function) ready for IND-enabling studies
- Leading next generation, long-acting Factor VIIa for hemophilia A/B inhibitor patients in ~$1.5B market with significant growth potential
  - Proof of mechanism, safety & tolerability demonstrated in hemophilia patients
  - Phase 1 Clinical Data presented at ISTH in June 2015
  - Phase 2/3 pivotal trial to initiate in Q1 2017
- Best-in-class Factor IX in hemophilia B; fully-funded to clinical Proof of Mechanism in late 2016
- Best-in-class Factor Xa for hemophilia and surgical bleeding with strong pre-clinical efficacy

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