

ChemoCentryx

First Quarter 2017

Financial Results Conference Call

May 10, 2017



Forward-Looking Statements

This presentation contains forward-looking statements that involve substantial risks and uncertainties. In some cases, you can identify forward-looking statements by the following words: “may,” “will,” “could,” “would,” “should,” “expect,” “intend,” “plan,” “anticipate,” “contemplate,” “believe,” “estimate,” “predict,” “project,” “seek,” “potential,” “continue,” “ongoing” or the negative of these terms or other comparable terminology, although not all forward-looking statements contain these words. These statements relate to future events or our future financial performance or condition and involve known and unknown risks, uncertainties and other factors that could cause our actual results, levels of activity, performance or achievement to differ materially from those expressed or implied by these forward-looking statements. These risks, uncertainties and other factors are described more fully in our periodic reports filed with the SEC, including our Annual Report on Form 10-K for the year ended December 31, 2016 filed with the SEC on March 14, 2017, particularly in the sections titled “Risk Factors” and “Management’s Discussion and Analysis of Financial Condition and Results of Operations”. In light of the significant uncertainties in our forward-looking statements, you should not place undue reliance on these statements or regard these statements as a representation or warranty by us or any other person that we will achieve our objectives and plans in any specified timeframe, or at all. The forward-looking statements contained in this presentation represent our estimates and assumptions only as of the date of this presentation and, except as required by law, we undertake no obligation to update or revise publicly any forward-looking statements, whether as a result of new information, future events or otherwise after the date of this presentation.

This presentation also contains estimates, projections and other information concerning our industry, our business, and the markets for our drug candidates, as well as data regarding market research, estimates and forecasts prepared by our management. Information that is based on estimates, forecasts, projections, market research or similar methodologies is inherently subject to uncertainties and actual events or circumstances may differ materially from events and circumstances reflected in this information.



CCXI Discovery Platform

Late Stage

THERAPEUTIC AREA	DRUG	INDICATION (TARGET)	PRECLINICAL	PHASE I	PHASE II	PHASE III
Complement Inhibition in Orphan and Rare Diseases	Avacopan (formerly CCX168)	ANCA ASSOCIATED VASCULITIS (C5aR)				
		ATYPICAL HEMOLYTIC UREMIC SYNDROME (C5aR)				
		C3 GLOMERULOPATHY (C5aR)				
Chronic and Other Rare Kidney Diseases	CCX140	FOCAL SEGMENTAL GLOMERULOSCLEROSIS (CCR2)				
		DIABETIC NEPHROPATHY (CCR2)				

Early Stage

Immuno-Oncology	CCX872	ADVANCED PANCREATIC CANCER (CCR2)				
		OTHER ONCOLOGY TARGETS (CCR1, CXCR2)				
Other Inflammatory and Autoimmune Diseases	Vercirnon	INFLAMMATORY BOWEL DISEASE (IBD): CROHN'S DISEASE (CCR9)				
	CCX507	IBD: ULCERATIVE COLITIS (CCR9)				
	CCX991	PSORIASIS AND TH17 DISEASES (CCR6)				

C3 Glomerulopathy (C3G)

Ultra-Rare Disease with Very High Unmet Clinical Need

Problem: Uncontrolled activation of the complement system

- Complement-mediated disease linked to complement pathway dysregulation
- Deficiency of complement pathway regulatory factor H leads to uncontrolled activation of alternative C3 convertase and C3G development
- Characterized by C3 and C5a deposition in glomeruli, but scant or no immunoglobulin deposition
- Complement deposition in glomeruli disrupts kidney function

Prevalence

2-3 per million people
(similar to aHUS)

Devastating Disease:

- No approved effective treatment
- Can be life-threatening
- Half of all persons with C3G have kidney failure
- Immunosuppressive drugs minimally effective
- Kidney transplant does not cure the disease; relapsing disease is common

Solution: Clear glomerular endocapillary proliferation, decrease inflammatory macrophages with C5aR inhibitor, avacopan



ANCA Associated Vasculitis (AAV)

A Destructive Autoimmune Orphan Disease

Problem: Autoimmune attack of blood vessels by neutrophils

- Activated by anti-neutrophil cytoplasmic auto-antibodies
- Leads to complement system activation and C5a generation
- C5a main activator of neutrophils
- Neutrophils inflame and destroy blood vessels = vasculitis
- Kidney is a major target organ

Incidence / Prevalence

US: ~7,000 / 90,000

Europe: ~10,000 / 120,000

Devastating Disease:

- Results in organ damage and failure, especially kidney
- Fatal if left untreated

Solution: Avacopan Blocks C5a-C5aR Activation of Neutrophils that Destroy Blood Vessels

Atypical Hemolytic Uremic Syndrome (aHUS)

Rare, Life-Threatening, Progressive Disease

Problem: chronic, uncontrolled activation of the complement system

- Formation of tiny blood clots in small blood vessels
- Blood clots reduce/prevent proper blood flow to organs (esp. kidneys)
- Low levels of circulating red cells due to their destruction
- Low platelet count due to their consumption
- Inability of kidneys to process waste products from blood

Current Treatment: eculizumab

- Very expensive, not accessible to all
- Compliance concerns with life-long IV infusions
- Safety concerns with *Neisseria* infections
- Deposition of eculizumab in glomeruli may affect efficacy/safety profiles
- Eculizumab not effective in all patients (C5 gene polymorphisms >lack of C5 binding)

Incidence:

2 per million people

Solution: Reduce thrombogenic activity driven by complement activation through C5aR inhibitor avacopan



Focal Segmental Glomerulosclerosis (FSGS)

A Rare Disease Where the Kidney Becomes Dysfunctional

Problem: increased levels of proteinuria leads to deterioration of renal function

- Histologic lesion from glomerular injury affecting specialized kidney filtering cells known as podocytes
- 'Nephrotic syndrome' - presents with very high levels of proteinuria
- *Reduction in proteinuria is expected registration endpoint*

Incidence

5,400 new cases in U.S.
1,000 kidney transplants/year
In 30-40% of transplant patients, FSGS returns

Devastating Disease:

- No FDA-approved treatments
- Leads to ESRD if untreated
- Average time from onset of proteinuria to ESRD: 6 - 8 years

Solution: Decreasing CCR2-driven inflammatory macrophages and relieving CCR2-damaged podocytes with CCX140 reduces proteinuria



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