COWEN AND COMPANY

ChemoCentryx

Initiating With Outperform (1)

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Better Science Begets Better Drugs

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Conclusion: We are initiating coverage of ChemoCentryx with an Outperform rating. The company utilizes a proprietary understanding of chemokine biology to develop potentially more selective drug candidates for autoimmune and inflammatory disorders. ChemoCentryx's approach has been productive thus far: Traficet-EN has produced positive Phase IIb data in Crohn's, a potential $1B+ market opportunity, and partner GSK is conducting a series of Phase III trials. In addition, CXC140 (wholly-owned) and CXC354 (also partnered with GSK) have yielded intriguing signs of activity in diabetic nephropathy and rheumatoid arthritis, respectively. Based exclusively on Traficet-EN's potential, we believe CCXI shares are 50% undervalued relative to the market.

- **Traficet-EN Holds Much Promise In Crohn's.** Data from a 436-patient Phase IIb trial indicate Traficet-EN has activity in inducing and maintaining remissions in patients with moderate-to-severe Crohn's disease. Phase III trials should read out in 2013 (induction) and 2014 (maintenance), respectively. Consultants are highly complimentary of Traficet-EN's pristine safety profile and are optimistic for success in Phase III. We believe a novel oral option for maintaining remissions in Crohn's disease will have much commercial appeal, and model peak sales of $1.4B in 2020.

- **CCX140 Seeks Validation In Nephropathy.** Inflammation is increasingly recognized as a contributor to diabetes, and clinical manifestations such as diabetic nephropathy in particular. CCX140 has been shown to improve kidney function in animals and glycemic control in humans. Data from an ongoing Phase II trial in diabetic nephropathy (YE:12) could establish efficacy in this large unmet medical need.

- **CCX354 Active In RA.** Data from a 160-patient Phase II trial provide proof-of-concept for this oral therapy. Partner GSK is responsible for all future clinical development, and will initiate additional studies in H2.

CCXI (03/21)	**$9.98**
Mkt cap	**$374.3MM**
Dil shares out	37.5MM
Avg daily vol	7.3K
52-wk range	$9.9-12.8
Dividend	Nil
Dividend yield	Nil
BV/sh	$2.50
Net cash/sh	$3.70
Debt/cap	2.0%
ROE (LTM)	NA
5-yr fwd EPS growth (Norm)	NA
S&P 500	**1402.9**

Revenue $MM

FY Dec	2010 Actual	2011E Prior	2011E Current	2012E Prior	2012E Current	2013E Prior	2013E Current
Q1	—	—	—	—	0.5	—	—
Q2	—	—	—	—	0.5	—	—
Q3	—	—	—	—	0.5	—	—
Q4	—	—	—	—	0.5	—	—
Year	**34.9**	—	**31.6**	—	**2.0**	—	**2.0**
EV/S	—	—	7.9x	—	125.0x	—	125.0x

EPS $

FY Dec	2010 Actual	2011E Prior	2011E Current	2012E Prior	2012E Current	2013E Prior	2013E Current
Q1	—	—	—	—	(0.27)	—	—
Q2	—	—	—	—	(0.28)	—	—
Q3	—	—	—	—	(0.29)	—	—
Q4	—	—	—	—	(0.30)	—	—
Year	**(0.10)**	—	**(0.21)**	—	**(1.15)**	—	**(1.25)**
P/E	—	—	—	—	—	—	—

Investment Summary

ChemoCentryx looks to take advantage of the selectivity of chemokine biology to create small molecule drug candidates for autoimmune and inflammatory conditions. The company has five clinical-stage programs led by Traficet-EN for Crohn's disease (in Phase III trials), CCX140 for diabetic nephropathy (in Phase II), and CCX354 for rheumatoid arthritis (Phase IIa complete). While the clinical profile of these candidates is still being assessed, each addresses a large market and has blockbuster ($1B+) potential. A major collaboration with GlaxoSmithKline (covering Traficet-EN, CCX354, and potentially one other candidate) has provided over $200MM in funding, and following a recent IPO in which ChemoCentryx raised $52MM in gross proceeds, we believe the company is sufficiently well capitalized to reach key milestones including pivotal data on Traficet-EN, Phase II data on CCX140, and potential proof-of-concept data on multiple earlier stage compounds. We expect CCXI shares to appreciate as the company achieves these milestones and investors gain greater familiarity with the company's assets.

ChemoCentryx R&D Pipeline

Therapeutic Class/Product	Indication	P-C	I	II	III	FILING	MKT	Comments
Inflammation								
Traficet-EN (CCR9)	Crohn's Disease				•			With partner GSK
CCX140 (CCR2)	Diabetic Nephropathy			•				Two trials, data around YE
CCX354 (CCR1)	Rheumatoid Arthritis			•				GSK responsible for development
CCx168 (C5aR)	Vasculitis			•				Phase II data H2:12
CCR4	Atopic Dermatitis	•						
CCR9	Ulcerative Colitis	•						Next generation Traficet-EN
CXCR6	Chronic Hepatitis	•						
CCR6	Autoimmune Disease	•						
CCX832 (ChemR23)	Skin Inflammation		•					Development on hold
Cancer								
CCX662 (CXCR7)	Glioblastoma Multiforme	•	⇒					Phase I to start in H2:12
Total Drugs In Development		**5**	**1**	**3**	**1**			

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Source: Cowen and Company

Good Drugs Don't Appear Out Of Thin Air

ChemoCentryx was founded in 1997 by CEO Tom Schall and others. From the outset the company's goal was to leverage the inherent selectivity of chemokine biology to develop more specific (less immunosuppressive) small molecules to treat inflammatory disorders. The chemokine system comprises a network of approximately 50 ligands and 25 receptors that guide inflammatory cells to tissues where an inflammatory response is initiated. The system is highly complex, and partially redundant, and ChemoCentryx has spent much of the past 15 years deciphering optimal points of attack for treating multiple disease conditions. The company has assembled a platform of proprietary tools (including novel receptor clones, functional genomic maps, and high-throughput cell migration-based assays) that have enabled the broadest pipeline of chemokine-targeted compounds in the industry (five in the clinic, five pre-clinical, all orally available). The efficiency and productivity of this platform is evidenced by the fact that ChemoCentryx has

generated its pipeline from capital raises totaling $175MM, yet ended 2011 with nearly $100MM in cash.

Pipeline Led By Traficet-EN

Traficet-EN, a CCR9 antagonist, is in a series of pivotal trials for the treatment of Crohn's disease. CCR9 is one of several chemokine receptors implicated in the recruitment of leukocytes to the gut, and work conducted by ChemoCentryx suggested it might be an ideal target for intervention in inflammatory bowel disease. Data from Traficet-EN's 436-patient Phase IIb PROTECT-1 trial provide support for this notion. In that trial, Traficet-EN was effective at inducing clinical responses by 12 weeks and maintained clinical remissions over a 36-week period. Traficet's safety profile in PROTECT-1 and earlier trials (785 subjects treated) is unblemished. Based upon these data, partner GSK opted into Traficet-EN's development and is responsible for conducting the Phase III program. Assuming positive Phase III results, Traficet-EN could be approved by 2015. We are particularly optimistic for the role of Traficet-EN in maintenance of Crohn's remissions, an indication that is devoid of good oral options. A favorable profile in Phase III could allow Traficet-EN to achieve sales in excess of $1B. We estimate ChemoCentryx will receive royalties of 15-22% on U.S. sales and 13-18% on Ex-U.S. sales.

CCX140 and CCX354 Follow

CCX140, a CCR2 antagonist, is ChemoCentryx's lead independent drug candidate. CCR2 is expressed on a subset of monocytes and macrophages. Activation of CCR2 is increasingly recognized as a component of the underlying inflammation associated with diabetes and diabetic nephropathy in particular. In preclinical studies, CCX-140 reduced inflammation, improved diabetic parameters, and positively impacted renal function. A Phase II trial in patients confirmed CCX-140's benefits on glycemic markers, but the drug's impact on renal parameters in humans still needs to be established. ChemoCentryx is conducting an adaptive design Phase II trial in 135-270 patients that will assess CCX140's ability to reduce albuminuria. Diabetic nephropathy is a huge unmet medical need, and any safe and effective therapy is likely to be rapidly adopted.

CCX354 targets the CCR1 receptor, which is activated by chemokine ligands in the synovial fluid of patients with rheumatoid arthritis (RA). Last year ChemoCentryx completed a Phase II trial on CCX354 in 160 patients with moderate to severe RA. CCX354 dosed 200mg QD improved ACR20 scores relative to placebo with the best separation seen in patients who were biologics naïve. In November 2011, partner GSK opted into development of CCX354, triggering a $25MM milestone payment to ChemoCentryx. GSK will be responsible for all further development and we estimate that ChemoCentryx will receive a 10-15% royalty on worldwide sales.

Other candidates include ***CCX168***, a C5a receptor antagonist in Phase II studies for vasculitis and ***CCX662***, a CXCR7 antagonist entering Phase I development for a form of brain cancer (GBM).

GSK Collaboration Provided Much Early Support

In 2006, ChemoCentryx signed a development collaboration with GlaxoSmithKline (GSK) worth up to $1.3B, of which $246MM has been received. The deal covers specific drug candidates directed against four receptor targets. GSK has opted into development of Traficet-EN and CCX354, and will owe ChemoCentryx milestones

and double-digit royalties as each of these candidates progress. The pharma company also retains an option on CCX168 pending proof-of-concept Phase II data. However, everything else in ChemoCentryx's pipeline is excluded from the collaboration, including a next generation CCR9 antagonist (preclinical) that could have utility in inflammatory bowel disease.

Financials And Valuation

Following the company's February IPO, ChemoCentryx has roughly 38MM shares outstanding and estimated cash on hand of $140MM. Given a fairly modest burn rate of approximately $40MM, ChemoCentryx appears well financed for the foreseeable future and has plenty of cash to see it through to the milestones noted below.

ChemoCentryx (CCXI) Upcoming Milestones

Milestone	Timing
Partner GSK initiates Phase IIb trial on CCX354 in RA	H2:12
Initiate Phase I trial on CCX662 in GBM	H2:12
Report Phase II data on CCX168 in vasculitis	YE:12
CCX140 Phase II results in diabetic nephropathy	YE:12
Pivotal data on Traficet-EN in Crohn's disease (induction)	2013
Pivotal data on Traficet-EN in Crohn's disease (maintenance)	2014

Source: Cowen and Company

We based our valuation of ChemoCentryx on Traficet-EN for Crohn's disease, the opportunity we know most about and have the most confidence in. Assuming moderate success in the induction setting and more significant utilization as a maintenance therapy, we believe Traficet-EN can achieve revenues of $1.4B in 2020, supporting fully taxed EPS of $2.40 to ChemoCentryx. Applying a 30X multiple and 25% discount rate on such earnings implies that CCXI shares are 50% undervalued relative to the market.

ChemoCentryx Quarterly P&L Model ($MM)

	Q1-Q3:11A	Q4:11E	2011E	Q1:12E	Q2:12E	Q3:12E	Q4:12E	2012E
Product sales								
Traficet-EN (GSK'786) royalties								
Other Products								
Collaborative and other revenue	5.6	26.0	31.6	0.5	0.5	0.5	0.5	2.0
Total Revenue	5.6	26.0	31.6	0.5	0.5	0.5	0.5	2.0
COGS								
gross margin								
R&D	22.9	8.0	30.9	8.0	8.5	8.5	9.0	34.0
SG&A	5.7	2.0	7.7	2.0	2.5	3.0	3.0	10.5
Total Expenses	28.6	10.0	38.6	10.0	11.0	11.5	12.0	44.5
Operating Income/Loss	(23.0)	16.0	(7.0)	(9.5)	(10.5)	(11.0)	(11.5)	(42.5)
Interest and Other Income	0.3	0.1	0.4	0.1	0.2	0.2	0.2	0.6
Interest and Other Expense	(0.2)	(0.1)	(0.3)	(0.1)	(0.1)	(0.1)	(0.1)	(0.4)
Provision for income taxes	-	-	-	-	-	-	-	-
Tax Rate								
Net Income (Loss)	(22.8)	16.0	(6.8)	(9.5)	(10.5)	(11.0)	(11.5)	(42.4)
GAAP EPS	**($0.71)**	**$0.50**	**($0.21)**	**($0.27)**	**($0.28)**	**($0.29)**	**($0.30)**	**($1.15)**
Basic and Diluted Shares Outstanding	32.1	32.1	32.1	35.0	37.5	37.6	37.8	37.0

Source: Cowen and Company

ChemoCentryx Annual P&L Model ($MM)

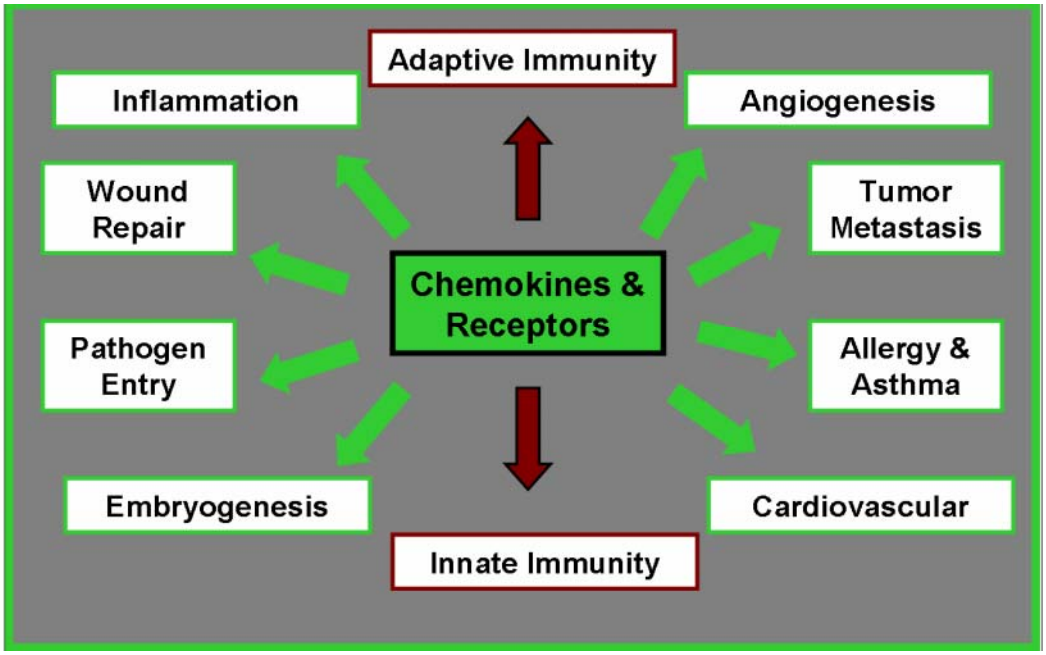
	2011E	2012E	2013E	2014E	2015E	2016E	2017E	2018E	2019E	2020E
Product sales										
Traficet-EN (GSK'786) royalties	-	-	-	-	-	-	-	-	-	-
Other Products	-	-	-	-	15.0	65.0	125.0	175.0	235.0	280.0
Collaborative and other revenue	31.6	2.0	2.0	26.0	51.0	26.0	0.0	0.0	0.0	0.0
Total Revenue	31.6	2.0	2.0	26.0	66.0	91.0	125.0	175.0	235.0	280.0
COGS		-	-	-	-	-	-	-	-	-
gross margin										
R&D	30.9	34.0	38.0	42.0	46.0	50.0	54.0	58.0	62.0	65.0
SG&A	7.7	10.5	12.5	16.0	23.5	29.0	32.0	34.0	36.0	38.0
Total Expenses	38.6	44.5	50.5	58.0	69.5	79.0	86.0	92.0	98.0	103.0
Operating Income/Loss	(7.0)	(42.5)	(48.5)	(32.0)	(3.5)	12.0	39.0	83.0	137.0	177.0
Interest and Other Income	0.4	0.6	0.5	0.5	0.5	1.0	1.0	2.0	2.0	2.0
Interest and Other Expense	(0.3)	(0.4)	(0.5)	(0.5)	(1.0)	(1.0)	(1.0)	(1.0)	(1.0)	(1.0)
Provision for income taxes	-	-	-	-	-	-	-	4.2	41.1	53.4
Tax Rate								5%	30%	30%
Net Income (Loss)	(6.8)	(42.4)	(48.5)	(32.0)	(4.0)	12.0	39.0	79.8	97.0	124.6
GAAP EPS	**($0.21)**	**($1.15)**	**($1.25)**	**($0.80)**	**($0.10)**	**$0.25**	**$0.80**	**$1.60**	**$1.90**	**$2.40**
Basic and Diluted Shares Outstanding	32.1	37.0	38.8	40.0	42.0	48.0	49.0	50.0	51.0	52.0

Source: Cowen and Company

Chemokines Are Key Players In Human Biology

Chemokines, or **chemo**tactic cyto**kines**, are a superfamily of small proteins that play a fundamental role in biology by directing the migration of leukocytes, endothelial cells, and epithelial cells within the body. Around 50 different chemokines have been identified, and their classification is based on the arrangement of their N-terminal cysteine residues (for example C, CC, CXC, and CX3C). The role of the chemokine system in human biology is very broad and includes guiding immune cells to sites of inflammation and injury, directing embryogenesis, and mediating angiogenesis/angiostasis. Chemokines are expressed by a wide variety of cell types, either constitutively or in response to inflammatory stimuli. Their expression and subsequent secretion leads to a chemokine concentration gradient, which helps guide chemokine-responsive cells to their appropriate destinations and forms the basis of specificity, both in terms of immune response and the potential for therapeutic interactions.

Diverse Roles For Chemokines

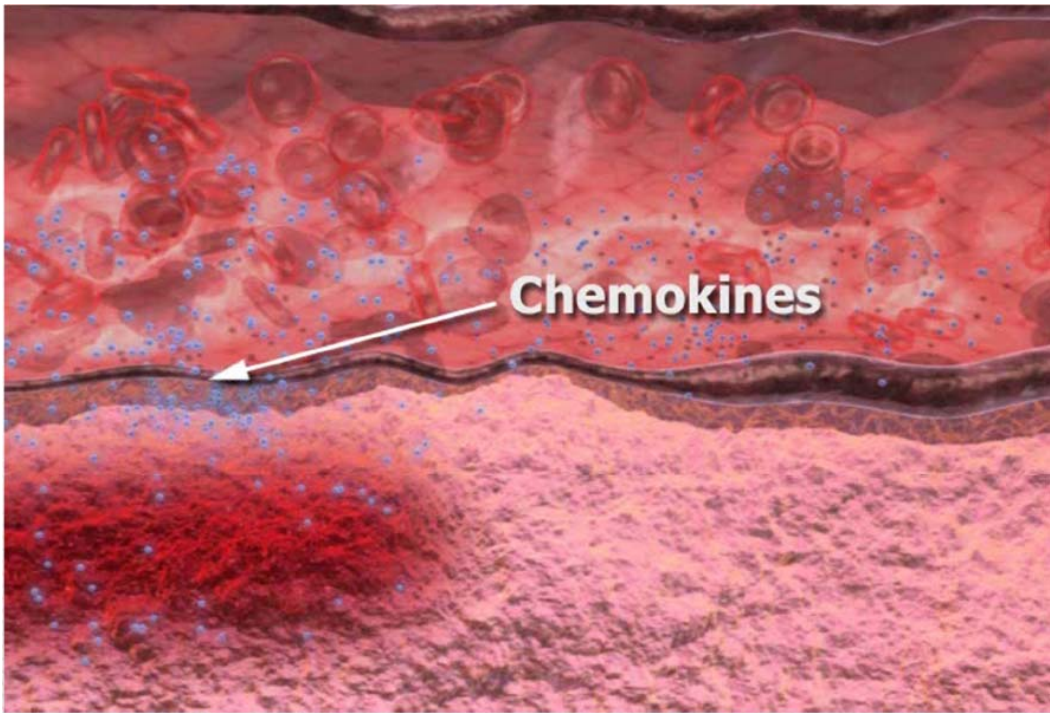


Source: Cowen and Company, Adapted from Le *et. al.*, Cellular and Molecular Immunology, 2004

Chemokines interact with their receptors (approximately 25 identified so far) in widely varying ways: some chemokines bind multiple receptors and vice versa while others bind only one receptor and vice versa. Given the varied and overlapping selectivity between chemokines and their receptors, some have suggested that the chemokine system is redundant. The implication is that multiple components of the chemokine system can carry out the same function, and thus therapeutic targeting of one particular chemokine or chemokine receptor may have limited efficacy. However, there is emerging evidence to support a different view, namely that divergent spatial and temporal expression patterns *in vivo* lead to each member of the chemokine system having a distinct function, even in instances where chemokine and chemokine receptors have overlapping binding activity. For example, depending on the particular tissue type and environment, one chemokine

binding a receptor may induce a different biological response than multiple chemokines binding the same receptor.

Chemokines Guide Inflammatory Cells To Their Destination



Source: ChemoCentryx

Chemokines Behaving Badly

Consistent with their fundamental role in biology, chemokine mis-regulation can have far-reaching consequences. For example, tumor-induced over-expression of chemokines and chemokine receptors such as CXCR7 can promote angiogenesis, which confers tumors with the ability to grow and metastasize. Another example is the role of CCR1 in rheumatoid arthritis (RA). Studies have shown that high expression of this chemokine receptor on monocytes and macrophages may guide their recruitment to and subsequent attack of the synovium lining the joints. Another example is the chemokine CCL25, whose over-expression is linked to Crohn's Disease (discussed in more detail below).

EnabaLink: A Key To Chemokines

ChemoCentryx's proprietary drug discovery platform is based on a set of tools known collectively as the EnabaLink technology suite. The company believes that EnabaLink provides it with key insights into the complexity of chemokine biology that can be used to propel drug discovery. EnabaLink includes a comprehensive functional genomic map that details the roles of chemokine receptors and gives insights into the most likely basis for inflammation in any particular condition. The system allows scientists at ChemoCentryx to accurately predict which chemokine receptors are involved in a given pathology and to design small molecules that target the underlying genes. EnabaLink also includes an assay referred to as the RAM Assay that allows the company to conduct high throughput screens for selective,

non-cytotoxic inhibitors of chemokine receptors based on cell migration assays. Using this system, ChemoCentryx has developed a broad pipeline that includes five clinical and five preclinical drug candidates that selectively and potently inhibit chemokine receptors. The drugs are orally available, and in comparison to biologics, are straightforward and inexpensive to manufacture.

Traficet-EN Aims To Quiet Crohn's

ChemoCentryx's lead candidate is Traficet-EN (CCX282 or GSK1605786), an orally available small molecule for the treatment of inflammatory bowel disease (IBD). The drug is a first-in-class anti-inflammatory agent that inhibits a chemokine receptor known as CCR9. By selectively inhibiting CCR9, Traficet-EN curbs inflammation in the gastrointestinal (GI) tract and diverts autoimmune attacks. It is partnered with GlaxoSmithKline. Inflammatory bowel disease refers to two autoimmune conditions: Crohn's Disease and Ulcerative Colitis. Although ChemoCentryx is developing Traficet-EN for both conditions, the company is focusing initial efforts on Crohn's Disease. A back up CCR9 antagonist in preclinical development may be advanced into development in Ulcerative Colitis.

Crohn's Disease, A Gut Reaction

First described in 1932 by Burrill Bernard Crohn, Crohn's Disease is a chronic and recurring inflammatory condition of the GI tract. It differs from Ulcerative Colitis in that it can affect any part of the GI tract from the anus to the mouth, although it usually only affects the colon and the last part of the small intestine. Ulcerative Colitis, on the other hand, is limited to the colon. Both conditions are thought to occur when cells of the immune system become overactive and mount a harmful inflammatory response in the GI tract.

Crohn's Disease affects all three layers of the intestine which include (1) the inside mucosal layer composed of epithelial tissue that is responsible for digesting food and absorbing nutrients into the body, (2) the middle mucosa layer composed of smooth muscle that pushes food through the intestine, and (3) the outer serosa layer composed of connective tissue coated with mucus that reduces friction in the abdomen. Inflammation in these different layers can cause intestinal scarring, swelling, and obstruction. In many cases patients develop fistulae, which are sores that tunnel through the affected areas and connect to other organs such as the vagina, bladder, or skin. If connected to the urethra, gas or stools may leak out during urination. Fistulae can also become infected, resulting in severe pain, fever, and even death.

Most Have Mild Disease, But Active Disease Is A Real Pain In The Gut

At diagnosis around 75% of Crohn's Disease patients have inflammatory disease while around 25% have more severe stricturing (narrowing of the intestines due to scarring) and/or penetrating disease. Following the first year of diagnosis, around 55% are in remission and 15% have mild disease. This leaves around 30% of patients who suffer from more active, moderate-to-severe disease. On average, it is thought that patients with Crohn's Disease spend around 65% of their lifetimes in remission, 27% of their lifetimes with mild disease, 7% of their lifetimes with severe disease, and a substantial amount of time in surgery. Over time, Crohn's Disease tends to develop a more aggressive behavior leading to stricture, abscess, and fistula formation.

Not surprisingly, Crohn's Disease can seriously diminish the quality of life for patients. Patients most often present with diarrhea and severe pain in the lower right abdomen. They can also suffer from weight loss, rectal bleeding, anemia, and fever. Furthermore, patients with Crohn's Disease usually have nutritional problems due to diarrhea, insufficient absorption of nutrients by the intestines, or altered eating habits. Other problems can include inflammation in the eyes or mouth, gall stones, kidney stones, liver disease, arthritis, rashes, and increased risk of colon cancer.

Crohn's Disease Terrorizes The West

Although there are no known causes for Crohn's Disease, there is some thought that it may be caused by the immune system responding to a virus or bacteria in the GI tract. Additionally, IBDs are linked to Westernized lifestyles and are associated with high socioeconomic status, smoking, high fat and sugar diets, stress, and use of medications. As would be expected, the highest incidence and prevalence rates are found in Northern Europe and North America, while the lowest rates are in Asia. The WW incidence of Crohn's Disease ranges from 0.03 to 15.6 cases per 100K per year while the prevalence is 3.6 to 214.0 cases per 100K. The Crohn's and Colitis Foundation of America (CCFA) estimates that around 700K Americans suffer from Crohn's Disease. Patients can be diagnosed at any age, though most are diagnosed in adolescence or early adulthood with onset between the ages of 15 and 35. Men and women are equally affected by Crohn's and around 20% of patients have a blood relative with an IBD. Although additional research is needed to characterize genetic links, studies have shown that variations in the *ATG16L1*, *IRGM*, and *NOD2* genes have been associated with increased risk of developing Crohn's Disease.

CDAI Score Used To Assess Disease Status

The Crohn's Disease Activity Index (CDAI) is most often used to determine the clinical activity of disease and includes the assessment of parameters such as body weight, diarrhea, abdominal pain, and anemia. Index values of less than 150 indicate remission, between 150 and 450 indicate active disease, and greater than 450 indicate extremely severe disease. According to the American College of Gastroenterology, patients classified as "mild to moderate" are ambulatory and can tolerate food without developing dehydration, toxicity, obstruction, tenderness in the abdomen, painful masses, or >10% weight loss. Patients who are classified as "moderate to severe" have failed "mild to moderate" treatments and have more severe symptoms such as high anemia, nausea or vomiting (without obstructions), fever, considerable weight loss, and pain or tenderness in the abdomen. Patients classified as "severe" have persisting symptoms despite outpatient steroid use and evidence of abscesses and/or obstructions, cachexia, high fever, continued vomiting, and rebound tenderness (Gionchettie, 2011).

In addition to CDAI, physicians use markers such as C-reactive protein (CRP) which is a nonspecific marker of inflammation, platelet count, and erythrocyte sedimentation rate to evaluate the activity of disease. Fecal lactoferrin and calprotectin may also be used to detect inflammation in the intestine. Unfortunately, despite these tests, there are no specific clinical or genetic predictors to help identify patients who have the highest likelihood of developing debilitating disease.

Current Treatments Are Focused On Controlling Symptoms

There are no cures for Crohn's Disease and current treatments are nonspecific and aimed at controlling active disease and/or acute flare-ups, rather than addressing the underlying mechanisms that drive disease. Therapy usually begins with an initial induction treatment followed by maintenance therapy to keep the disease symptoms under control. Current treatment options vary in efficacy and are often expensive and accompanied with notable side effects. Within the first 20 years of diagnosis an estimated 75% of patients with ileal (distal portion of the small intestine) or ileocolonic disease will require surgery, after which 80% of patients have an endoscopically-detectable recurrence within the first year, though most are asymptomatic. As would be expected, the choice of treatment depends on the activity of the disease, its location, and its behavior (fistulating, inflammatory, etc) (Gionchettie, 2011).

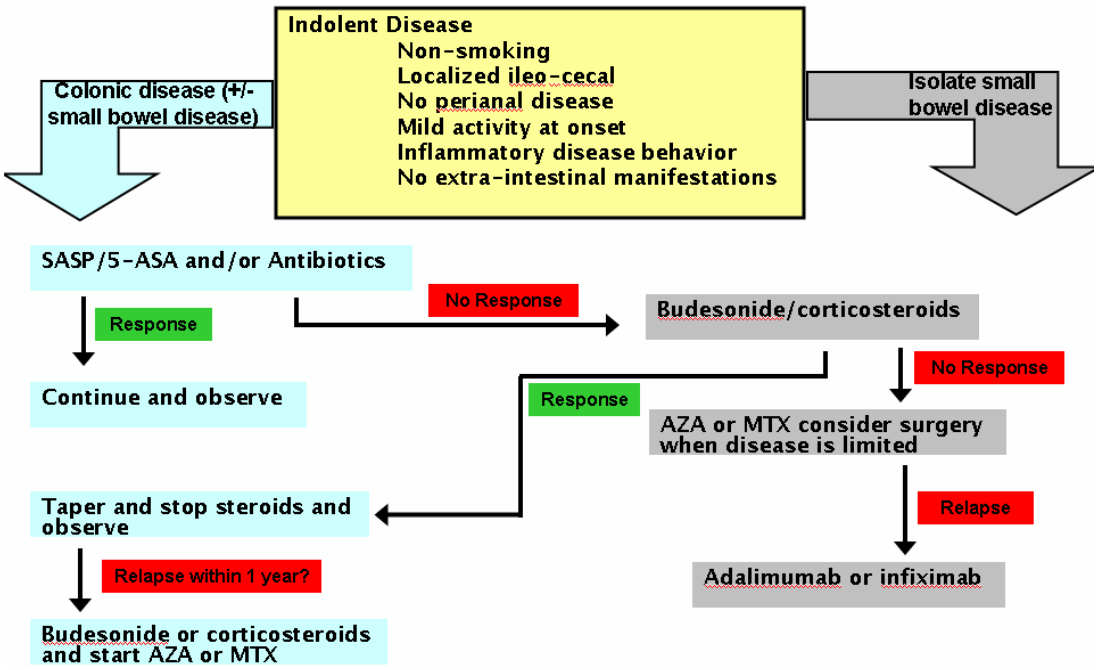
A Brief Overview Of The Current Armamentarium

One well-known and much debated option for Crohn's Disease is treatment with 5-aminosalicylic acid (5-ASA)-containing drugs. Although numerous studies have tested these drugs over the last 25 years, no study has convincingly proven their efficacy. Furthermore, these drugs can have a high incidence of side effects and are therefore not recommended by the European Crohn's and Colitis Organization (ECCO) for use as a front-line therapy. Budesonide is another treatment option that has more convincing evidence for inducing remission of active Crohn's Disease. Budesonide is not as effective at inducing remission as conventional steroids, but it has fewer side effects and is not as harsh on the kidneys.

Steroids are also used to treat active Crohn's Disease. A first course of steroid use results in around a 50-60% complete response rate, 30% partial response rate, and 10-20% no response rate. After one year, however, only around a third of patients have a durable response. The other two-thirds of patients either develop resistance to steroids or become dependent on them. Steroids are also not ideal due to the well-established toxicities associated with long-term use. Mercaptopurine and azathioprine (AZA) are the most common immunomodulators in use for Crohn's, but data showing their benefit on efficacy are conflicting and these drugs are most often relegated to the maintenance setting. Additionally, their benefit has to be weighed against their higher treatment-associated risk with lymphoproliferative diseases. Methotrexate is used to treat patients who have active or relapsing Crohn's Disease and who are refractory/intolerant of mercaptopurine or azathioprine.

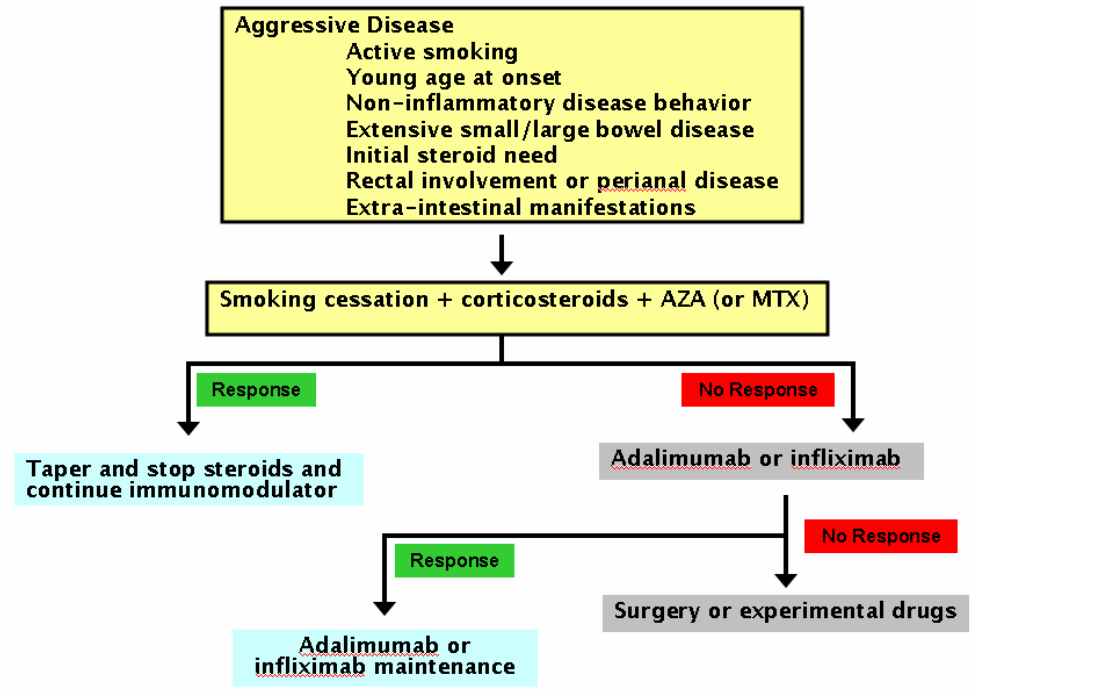
Since the late 1990s, physicians have employed TNF-alpha inhibitors such as infliximab (Remicade®) and adalimumab (Humira®) to treat patients who have disease that cannot be controlled with immunosuppressives or steroids. While these therapies represent a major advance in Crohn's treatment, they have several limitations, including their high cost and injectable administration. In addition, infliximab, a chimeric antibody, can be neutralized by antibodies that recognize its murine components. Moreover, both therapies are somewhat nonspecific and thus have been associated with serious side effects including an increased risk for developing treatment-induced lupus, lymphoma, sepsis, and reactivation of tuberculosis.

Treatment Algorithm For Indolent Crohn's Disease



Source: Cowen and Company, Adapted from Gionchetti *et. al.*, World Journal of Gastroenterology, 2011

Treatment Algorithm For Aggressive Crohn's Disease



Source: Cowen and Company, Adapted from Gionchetti *et. al.*, World Journal of Gastroenterology, 2011

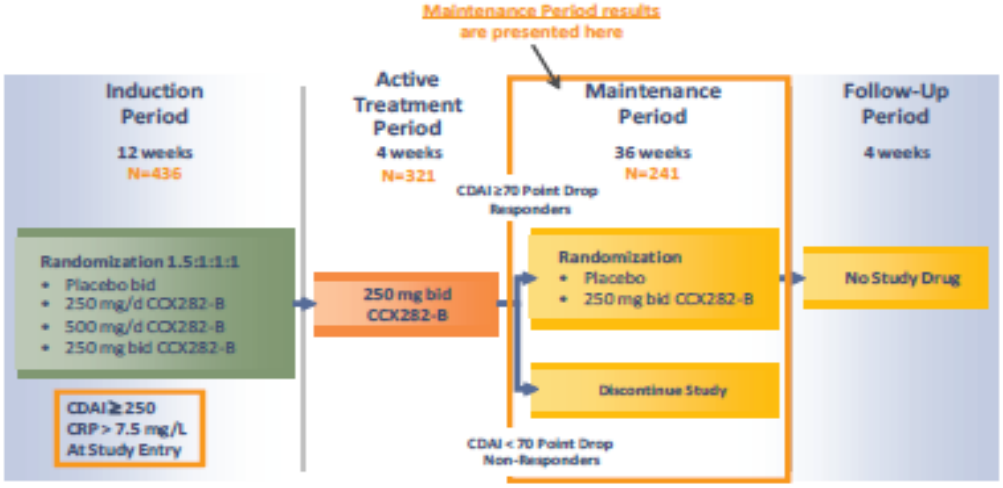
ChemoCentryx's Strategy: Selective Inhibition

To address the limitations of current therapies, ChemoCentryx is developing Traficet-EN, a selective inhibitor of the chemokine receptor CCR9. CCR9 is expressed on inflammatory T-cells and responds to a chemokine called CCL25 (also known as TECK). In Crohn's Disease, CCR9-expressing T-cells are guided by a concentration gradient of over-expressed CCL25 to the digestive tract where they carry out an autoimmune attack. Importantly, it is thought that in adults CCR9-expressing inflammatory cells only respond to CCL25 in the gut and not in any other tissues. Thus, inhibiting CCR9 should only affect the cells' ability to migrate to the digestive tract and not to other parts of the body where they may be needed to fight off infection. By inhibiting CCR9, Traficet-EN may curb inflammation in the digestive tract and provide a therapeutic benefit for patients with Crohn's Disease.

PROTECT1 Lays The Foundation

ChemoCentryx's Phase IIb PROTECT-1 (**P**rospective **R**andomize **O**ral **T**herapy **E**valuation in **C**rohn's Disease **T**rial) trial in Crohn's Disease laid the foundation for Phase III development. The trial was a randomized, double-blind, placebo-controlled trial conducted at over 100 locations in 17 different countries (ex-U.S.). The trial enrolled 436 patients with a CDAI score of 250 to 450 (average baseline CDAI = 330), an active disease flare-up, and CRP levels of at least 7.5mg/L. Four separate study periods were included in the trial: (1) A 12-week induction phase testing 250mg QD, 500mg QD, or 250mg BID Traficet-EN vs. placebo. This phase examined the ability of Traficet-EN to induce a clinical response or remission in patients who had active disease; (2) A 4-week phase in which all patients received twice-daily treatments of 250mg Traficet-EN; (3) A 36-week phase examining the ability of 250mg BID Traficet-EN to maintain a clinical response or remission in patients who had achieved a ≥70-point drop in CDAI scores relative to baseline following the previous 4-week phase; (4) A final 4-week phase evaluating patients for safety. As a whole, the results from this trial showed that 500mg QD Traficet-EN is effective at both inducing a clinical response and in maintaining remission in patients with moderate to severe disease. Traficet-EN was also found to be safe and well-tolerated by patients who had taken the drug during the one year timeframe of the trial.

PROTECT1 Phase IIb Trial Design

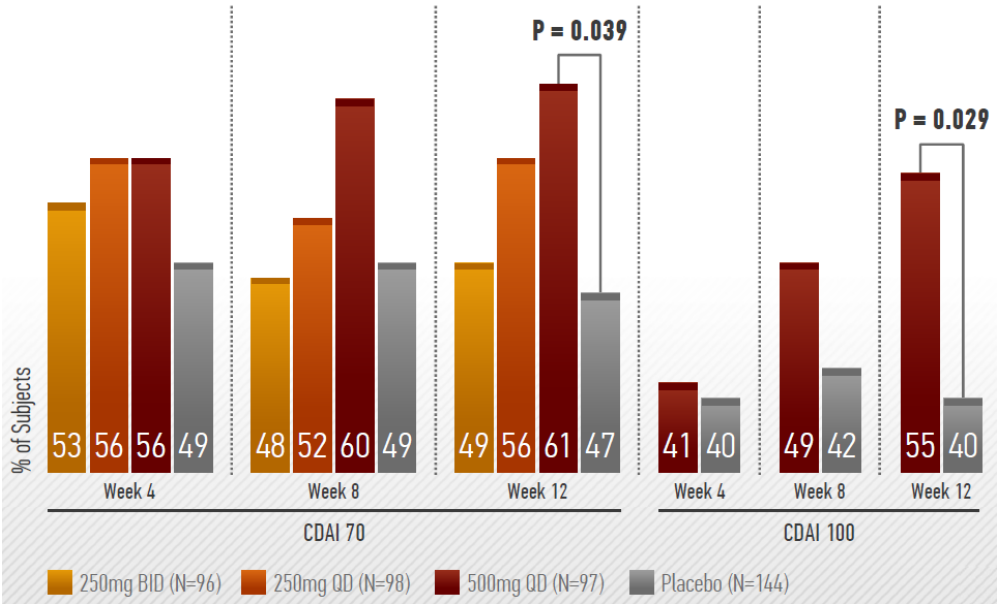


Source: ChemoCentryx

12-Weeks Of Traficet-EN Hits The Mark

Results for the 12-week induction phase were solid. At each time point evaluated, the difference between the 500mg QD vs. placebo groups increased, ramping from a 7% difference at 4 weeks to an 11% difference at 8 weeks to a 14% difference at 12 weeks. Though ChemoCentryx chose 8-weeks as the prospective primary endpoint (and statistical significance was not achieved at this juncture), at Week 12 the data did cross statistical hurdles. The ≥100-point CDAI score response was 55% in the 500mg QD group compared to 40% in the placebo group (p=0.039). However, the 500mg QD dose did not positively impact the percent of patients achieving remission (CDAI ≤ 150-points). Although the other doses produced less consistent activity, the 250mg QD group also showed a trend towards improvement vs. placebo at Week 12. The 250mg BID group, however, did not repeat or improve on the trend, and CCXI has alluded to the possibility that the lack of a consistent dose response may be related to the pharmacodynamic properties of the molecule, and the need to achieve a high Cmax plasma concentration.

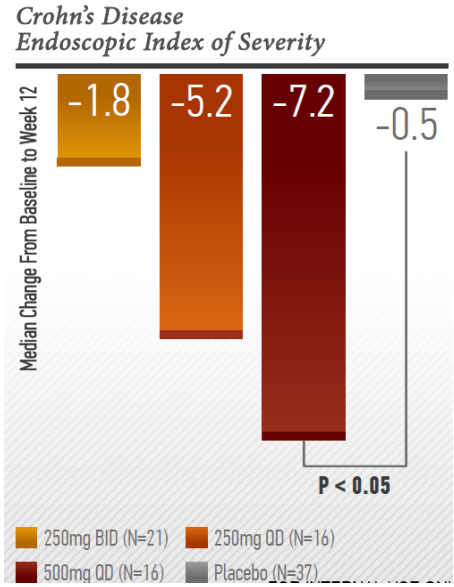
12-Week Induction Data



Source: ChemoCentryx

Interestingly, the data also showed a benefit for patients who had not previously responded to anti-TNF therapy. In those patients, 57% treated with 500mg QD Traficet-EN vs. 28% treated with placebo had ≥100-point drop in CDAI score, while 21% of Traficet-EN-treated patients vs. 6% of control-treated patients had a CDAI remission (CDAI ≤ 150). Additionally, patients on Traficet-EN experienced an improvement in CRP levels: the median change from baseline at 12 weeks was -6.7 mg/L for 500mg QD vs. -2.9 mg/L for placebo. Furthermore, in the subset of patients in the trial who consented to endoscopic examination, the study found that 500mg QD significantly reduced endoscopic lesions compared to control (p=0.049).

Reduced Endoscopic Lesions In Patients Treated With Traficet-EN

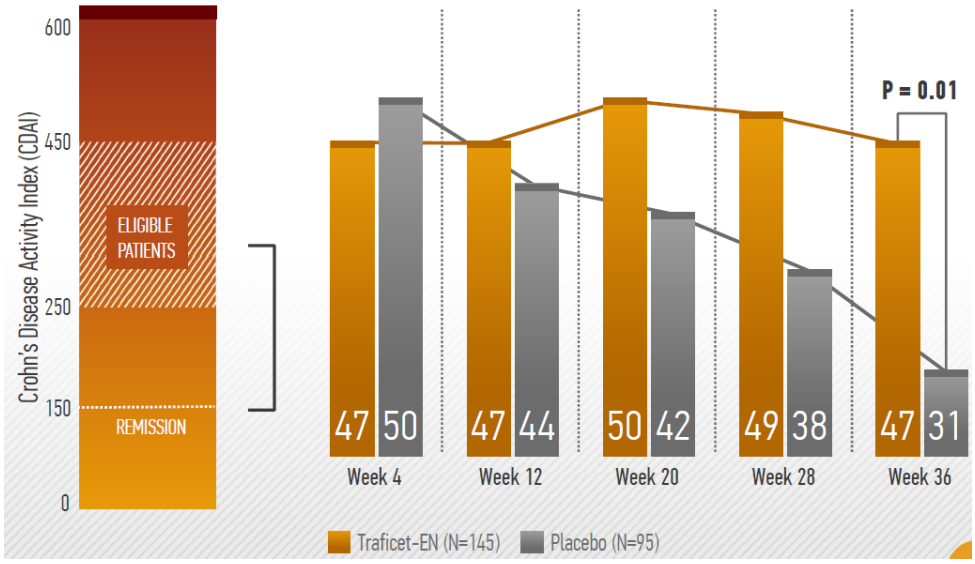


Source: ChemoCentryx

Traficet-EN Helps Maintain Remission

In our view, Traficet-EN is most likely to be adopted in the maintenance setting where the benefits of an oral therapy are most pronounced and the data on Traficet-EN is strongest. Results from the 36-week arm of the trial examining Traficet-EN at a dose of 250mg BID showed that the proportion of patients with clinical remission was 47% on Week 36 versus 31% for patients treated with placebo during the same time period (p=0.011).

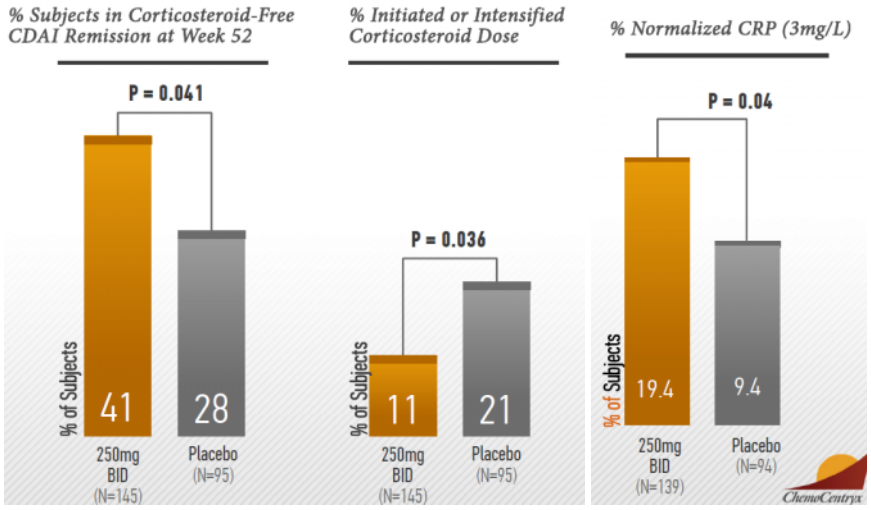
Traficet-EN Maintains Remission Compared To Placebo



Source: ChemoCentryx

Notably, patients treated with Traficet-EN in the maintenance study had a reduced need for corticosteroids: around 11% of patients in the Traficet-EN group vs. 21% of patients in the control group initiated or increased corticosteroid treatment (p=0.04). Furthermore, 57% of patients in the Traficet-EN group vs. 43% of patients in the control group were able to discontinue corticosteroid treatment during the study, and at Week 52, 41% of patients treated with Traficet-EN vs. 28% of patients treated with placebo had corticosteroid-free CDAI remission (p-0.041). Additionally, 19.4% of patients on Traficet-EN had normalized CRP levels (3mg/L) compared to only 9.4% of patients on the control arm.

Steroid-Free Remission And Normalized CRP In Patients Treated With Traficet-EN



Source: ChemoCentryx

Traficet-EN Is Well-Tolerated And Has No Safety Concerns

One of the biggest advantages of Traficet-EN over currently available therapies is its favorable safety and tolerability profile. During the 12-month duration of the trial, no patients had evidence of a compromised immune system, nor were there any opportunistic infections or concerns with laboratory data, vital signs, or ECGs. In the induction phase of the study, a similar proportion of patients treated with Traficet-EN and placebo had treatment-emergent adverse events (59.8% vs. 62.5%, respectively). Similarly, the numbers were about equal for patients reporting treatment-emergent SAEs during the induction phase (8.6% vs. 10.4%). There were no infectious SAEs on Traficet–EN. The same held true for the maintenance phase of the trial with roughly equal numbers of patients in each group reporting AEs, GI AEs, SAEs, serious infections, and AEs leading to withdrawal.

Consultants are impressed with Traficet-EN's safety profile, which they believe is enabled by its more targeted (gut specific) mechanism of action. They believe an oral option with efficacy on par or better than azathioprine at maintaining remissions would witness broad adoption, especially if Traficet-EN's steroid sparing properties are validated. Given Traficet-EN's clean safety profile, they believe it could be ideal for use in combination with other agents.

Favorable Safety For Traficet-EN In The Induction Phase

Safety Results – Induction Period

	PLACEBO (N=144)	All Traficet-EN (N=291)	250mg QD (N=98)	500mg QD (N=97)	250mg BID (N=96)
Subjects with AEs	63%	60%	63%	58%	58%
Subjects with SAEs	10%	9%	5%	9%	12%
W/Ds due to AEs	13%	7%	8%	7%	5%
W/Ds due to GI AEs	10%	5%	6%	5%	3%
W/D due to Crohn's	4%	1%	3%	1%	0%

Source: ChemoCentryx

Favorable Safety For Traficet-EN In The Maintenance Phase

Safety Results – Maintenance Period (9 months additional dosing)

	PLACEBO (N=95)	Traficet-EN (N=145)
Any Adverse Event	61%	62%
GI Adverse Events	43%	41%
Serious Adverse Events	10%	9%
Serious Infections	0	0
Adverse Events Leading to Withdrawal	6%	7%

Source: ChemoCentryx

Phase III Clinical Development In GSK's Hands

Traficet-EN is currently being developed in four Phase III clinical trials (SHIELD1-4) for Crohn's Disease, all of which are being conducted by partner GlaxoSmithKline (GSK). SHIELD1 (**S**tudy in Cro**H**n's Disease Patients **I**nvestigating the **E**fficacy and Safety of an Ora**L**ly **D**osed CCR9 Antagonist) is a multi-national, double-blind, randomized study evaluating the safety and efficacy of Traficet-EN vs. placebo over 12 weeks of treatment. The trial, which will enroll approximately 600 patients with moderate to severe disease, is testing the ability of two doses of treatment (500mg once daily and 500mg twice daily) to induce remission. The primary endpoint for the trial is proportion of patients achieving a clinical response as defined by a decrease in CDAI score of at least 100 points. Secondary endpoints will include remission (CDAI < 150) and change from baseline in Inflammatory Bowel Disease

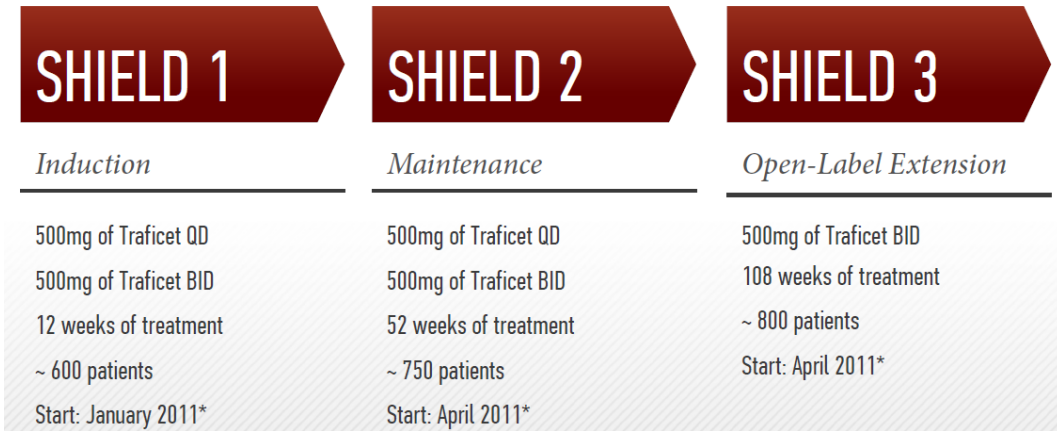
Questionnaire (IBDQ) total score. The estimated completion date according to clinicaltrials.gov is August 2012.

SHIELD2 is the second multi-national, double-blind, randomized, placebo-controlled trial. This trial began in April 2011 and is evaluating the safety and efficacy of Traficet-EN in maintaining remission over 52 weeks of treatment. To be eligible for the trial patients must have achieved a clinical response (decrease in CDAI of at least 100 points) and/or remission (CDAI < 150) in a previous GSK-sponsored study. The trial is testing 500mg once daily and 500mg twice daily and will enroll around 750 patients. The primary endpoint for the study is the number of patients in remission at Weeks 28 and 52. According to clinicaltrials.gov the trial is estimated to be complete in July 2014.

SHIELD3 is a multi-national, open-label extension study that is testing 500mg Traficet-ENtwice-daily in around 800 patients who previously received Traficet-EN. The trial began in April 2011 and is primarily focused on evaluating the safety of Traficet-EN over the course of 108 weeks of treatment. The trial is expected to be completed in July 2014.

Lastly, SHIELD4 is a multi-center, randomized, double-blind study with a very similar design to SHIELD1. The study's primary endpoint is proportion of patients achieving clinical response (decrease in CDAI score of at least 100 points) with the objective of qualifying patients for enrollment in SHIELD2. The trial began in November 2011 and is expected to enroll around 900 patients. The estimated study completion date is January 2014.

Phase III Pivotal Trials In Crohn's



Source: ChemoCentryx

Traficet-EN Licensed To GSK

In January 2010 GSK exercised its option for an exclusive license to develop and globally commercialize Traficet-EN. Subsequently, ChemoCentryx received a $35MM option exercise fee and the company remains eligible for additional regulatory and commercial milestone payments. ChemoCentryx is also entitled to receive double-digit royalties on product sales (we estimate 15-22%). GSK is responsible for all clinical development and commercialization costs, including the SHIELD1-4 Phase III trials. Additionally, ChemoCentryx maintains the option to co-promote Traficet-EN (up to 50% of all promotional efforts) in the U.S., and if it exercises this option, it will

be required to pay 35% of GSK's development costs and be eligible for a higher royalty rate.

Potential IP Dispute Not Cause For Concern

Millennium Pharmaceutical, a subsidiary of Takeda, holds a patent on small molecules that modulate CCR9, including a series of constructs that cover Traficet-EN's structure. ChemoCentryx also holds an issued U.S. patent on Traficet-EN. There is no dispute between the companies, though it is possible that one might arise in the future. There is reason to believe that Millenium's claims are invalid as the company did not appear to be in possession of Traficent-EN at the time of filing and the body of the patent may teach away from Traficet-EN's structure. Under a worst case, we believe any future dispute could be settled for a nominal payment.

Commercialization Of Traficet-EN

Due to the exercise of its option, GSK will be fully responsible for global commercialization of Traficet-EN. The drug will most likely be positioned as the therapy of choice for patients who have moderate-to-severe disease in both the frontline and maintenance settings. Furthermore, in light of the drug's very favorable tolerability and side effect profile, Traficet-EN could be combined with current treatments as needed. Given its many benefits over immunosuppressive drugs and steroids, Traficet-EN is likely to take considerable share.

The Crohn's and Colitis Foundation of America estimates that approximately 700K people in the U.S. have Crohn's Disease. Using a similar incidence for Western Europe, we estimate that around 1MM patients might be affected in that geography. Approximately 30% of patients have moderate-to-severe disease, and of these we estimate that 20% have active disease and 80% are in remission. Based on the Phase IIb data, we think it is likely that Traficet-EN will be used as both an induction therapy and as a maintenance therapy. However, we believe the drug will be more rapidly adopted for maintenance, and thus we have modeled a higher penetrance in this setting. Traficet-EN will likely be priced commensurate with its clinical benefits, but we estimate initial U.S. pricing of $14K/year and E.U. pricing of $10K (substantially below that for ant-TNF therapy). Given these assumptions, we estimate peak sales in the U.S. and Europe of $1.4B in 2020. For comparison, we estimate worldwide sales of JNJ's Remicade in Crohn's at $1.5-2.0B. With a royalty rate of 15-22% on U.S. sales and 13-18% on Ex-U.S. sales, this translates into $280MM to ChemoCentryx in 2020.

Traficet-EN Revenue Model

	2015	2016	2017	2018	2019	2020
U.S. Prevalence Of Crohn's (000s)	743	758	773	788	804	820
Percent Diagnosed With Moderate-To-Severe	*30%*	*30%*	*30%*	*30%*	*30%*	*30%*
Percent Moderate-To-Severe In Need of Induction	*20%*	*20%*	*20%*	*20%*	*20%*	*20%*
Penetration of Traficet-EN For Induction	*1%*	*3%*	*6%*	*8%*	*10%*	*11%*
Number of patients who receive Traficet-EN For Induction (000s)	0	1	3	4	5	5
Percent Moderate-To-Severe In Remission	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*
Penetration of Traficet-EN In Maintenance Setting	*4%*	*9%*	*14%*	*16%*	*19%*	*21%*
Number of patients who receive Traficet-EN For Maintenance (000s)	7	17	25	30	37	42
Average price per course of therapy	$14,000	$14,700	$15,435	$16,207	$17,017	$17,868
Total US Sales (MM)	**$100**	**$270**	**$430**	**$550**	**$705**	**$850**
% Royalties for CCXI from GSK	15%	17%	20%	21%	22%	22%
U.S. Royalties to CCXI (MM)	**$15**	**$47**	**$86**	**$115**	**$155**	**$187**
EU Prevalence of Crohn's (000s)	987	1,007	1,027	1,047	1,068	1,090
Percent Diagnosed With Moderate-To-Severe	*30%*	*30%*	*30%*	*30%*	*30%*	*30%*
Percent Moderate-To-Severe In Need of Induction	*20%*	*20%*	*20%*	*20%*	*20%*	*20%*
Penetration of Traficet-EN For Induction	*0%*	*2%*	*4%*	*6%*	*9%*	*10%*
Number of patients who receive Traficet-EN For Induction (000s)	0	1	3	4	6	7
Percent Moderate-To-Severe In Remission	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*
Penetration of Traficet-EN In Maintenance Setting	*0%*	*5%*	*9%*	*12%*	*15%*	*17%*
Number of patients who receive Traficet-EN For Maintenance (000s)	0	12	22	31	38	45
Average price per course of therapy	$10,000	$10,000	$10,000	$10,000	$10,000	$10,000
Total EU Sales (MM)	**$0**	**$130**	**$245**	**$350**	**$440**	**$515**
% Royalties for CCXI from GSK	13%	14%	16%	17%	18%	18%
EU Royalties To CCXI (MM)	**$0**	**$18**	**$39**	**$60**	**$80**	**$93**
Total Sales (MM)	**$100**	**$400**	**$675**	**$900**	**$1,146**	**$1,365**
Total Royalties To CCXI (MM)	**$15**	**$65**	**$125**	**$175**	**$235**	**$280**

Source: Cowen and Company

CCX140 Takes On Kidney Disease

ChemoCentryx's lead independent drug candidate, CCX140, is an oral antagonist of the chemokine receptor CCR2 in Phase II development for the treatment of diabetic nephropathy. CCX140 has generated intriguing preclinical data in animal models of kidney disease and completed a Phase II clinical trial in type 2 diabetics with normal renal function. However, the drug has yet to establish proof-of-concept in humans with impaired renal function. ChemoCentryx is optimistic that two Phase II clinical trials in patients with diabetic nephropathy will provide such evidence of activity. Phase II data are expected in late 2012/early 2013. Though CCX140's development is high risk, the market opportunity in diabetic nephropathy is large, and any safe and efficacious agent could have blockbuster potential. We await data from CCX140's Phase II trials before including revenue in our model.

Diabetic Nephropathy – Insidious And All Too Common

The Centers for Disease Control and Prevention (CDC) estimates nearly 26 million Americans have diabetes with 800,000 new cases diagnosed yearly. Another estimated 79 million Americans are in a state of prediabetes. At present, the total healthcare cost of this disease is $98B comprising roughly 15% of all US healthcare costs.

Diabetes is so costly and detrimental because of its secondary complications. One particularly common complication is diabetic nephropathy (DN). Between 20-30% of patients suffering from diabetes (type 1 or 2) will develop renal disease after 10

years. In fact, diabetic nephropathy is responsible for 30-40% of all end-stage renal disease (ESRD) cases in the United States. While medical advances have helped to prevent disease progression through earlier diagnosis and aggressive glycemic control strategies, the volume of patients presenting with new onset diabetic nephropathy provides increasing challenges to practitioners.

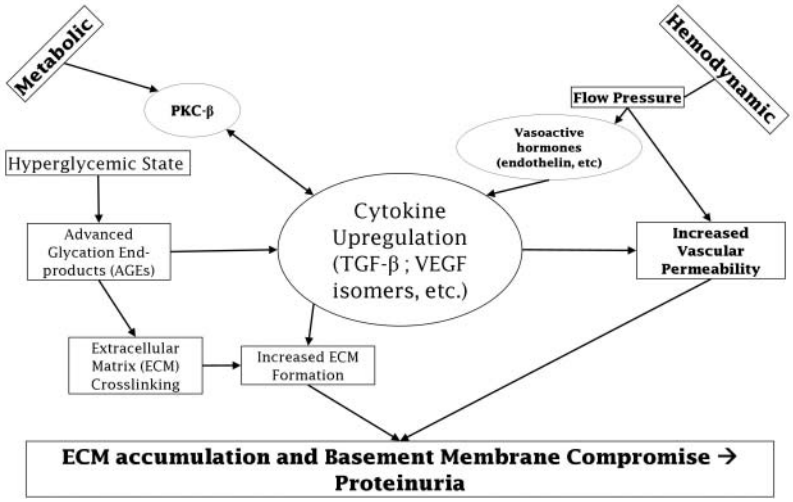
From a clinical perspective, diabetic nephropathy patients often present with variable symptoms. Some patients may complain of hematuria or flank pain, while other patients may present with subtle symptoms such as edema, hypertension, and/or uremia. However, often patients may be diagnosed by lab parameters alone (elevated serum creatinine, microscopic hematuria, or proteinuria secondary to glomerular hyperfiltration). From time of diagnosis, patients with declining Glomerular Filtration Rate (GFR) will witness progressive renal dysfunction and require higher level care.

The primary goal of medical treatment is aggressive glycemic control to prevent associated sequelae. Patients with signs of kidney disease are treated with angiotensin-converting enzyme (ACE) inhibitors, or to a lesser extent angiotensin-receptor blockers (ARBs), usually in combination with diuretics. Such drugs decrease the effects of hyperfiltration and slow the path to renal failure. There are no drugs for improving function in damaged kidneys.

Inflammation Plays A Role In Pathogenesis

Inflammation is increasingly recognized as playing a role in diabetic nephropathy. There are two competing hypotheses for how inflammation might be triggered. The first is via direct cellular damage: hyperglycemia causes proteins to react with glucose to form an advanced glucosylation end-product (AGEs), which can cause cellular dysfunction leading to cytokine production and inflammatory cell recruitment in the kidney. Alternately, the hemodynamic changes such as increased flow pressure could trigger increased cytokine production. Either way, studies have shown elevated levels of macrophages and in the kidney and a correlation between macrophages levels and kidney damage.

Inflammation Contributes to Pathology



Source: Cowen and Company, Adapted from WebMD

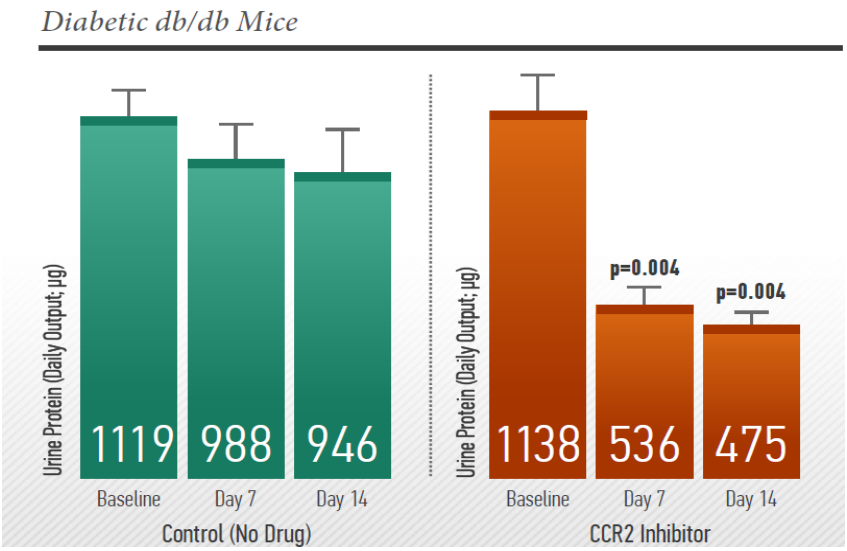
Studies have shown that glomerulosclerosis (damage to the filtering units of the kidney) can be ameliorated with inhibition of macrophage infiltration. The CCR2 receptor, and its ligand CCL2 (or MCP-1), are believed to be the main drivers of monocyte and macrophage recruitment into the kidney. CCL2 is produced in response to high glucose levels and the excretion of CCL2 in the urine correlates with kidney damage. As such the CCR2/CCL2 signaling pathway appears to be a good target for intervention.

CCX140 Goes To The Root Of The Problem

CCX140 is a potent antagonist of CCR2 and inhibits CCL2-induced monocyte chemotaxis with an IC50= 8nM. Furthermore, it selectively binds CCR2 over its related chemokine receptor CCR5.

In diabetic mice induced to develop glomerulosclerosis, CCX140 was found to have significant renal-protective effects. Blockade of the CCL2/CCR2 activation cascade in mice was noted to reduce monocyte/macrophage mediated chemotaxis, decrease circulating monocyte/macrophages, and reduce renal lymphocyte infiltration. Mice were noted to have less evidence of glomerulosclerosis, improved albuminuria, and have an improved GFR. In fact, CCX140 also exerted beneficial effects on fasting glucose levels. Thus, in animal studies ChemoCentryx has shown that CCR2 blockade has potentially significant beneficial effects in the setting of diabetic kidney disease.

CCR2 Inhibition Improves Renal Function



Source: ChemoCentryx

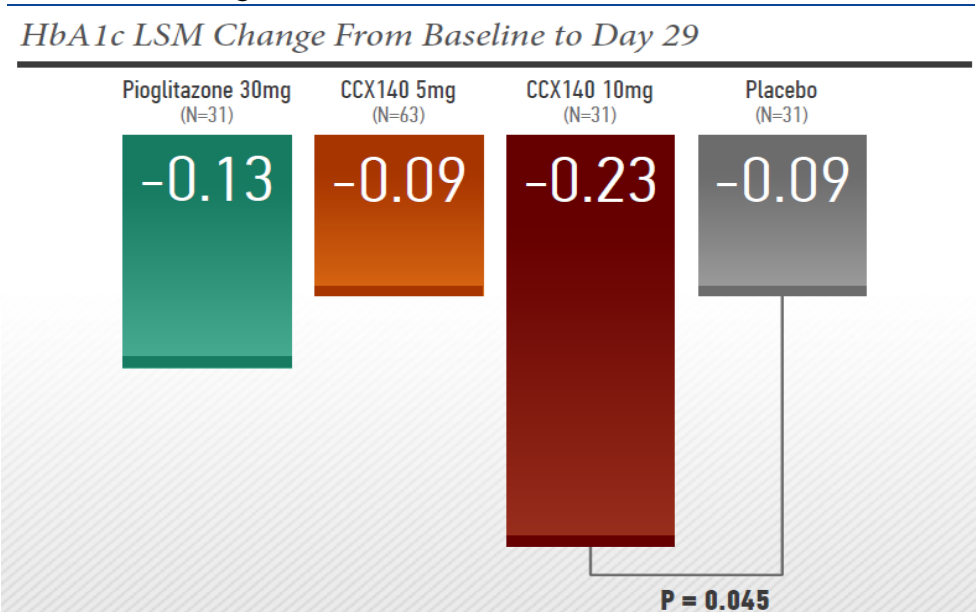
CCX140 Well Tolerated In Early Trials

ChemoCentryx conducted a Phase I single ascending dose trial in 56 volunteers. CCX140 was given at a single dose of 0.05 mg, 0.1 mg, 0.3 mg, 0.6 mg, 1 mg, 3 mg, and 10 mg and compared to placebo. CCX140 at these doses was well tolerated. No serious adverse events (SAE) were noted and mild to moderate AEs did not correlate with the dose level of CCX140. A second Phase I multiple ascending dose trial was conducted in 32 patients to evaluate doses of 0.6 mg or 2 mg (for 7 days) and 5 mg

or 10 mg (for 10 days). CCX140 appeared to be well tolerated with no SAEs and any AEs were similarly mild to moderate. A Phase 1 trial evaluating higher doses (5 mg, 10 mg, 12.5 mg and 15 mg) is currently ongoing. Results of this study will support the evaluation of doses higher than 10 mg if needed in Phase II.

A Phase II clinical trial in subjects with Type 2 diabetes was completed in 159 patients on stable metformin with no evidence of renal disease. The clinical trial was conducted mostly in Latin America and designed to demonstrate safety of CCX140 in diabetics with normal renal function and the effect of CCX140 on glycemic indices. The patients were randomized to receive placebo, 5mg CCX140, 10mg CCX140, or 30mg pioglitazone (Actos, active comparator) once daily for 4 weeks. Fasting plasma glucose decreased in a dose-dependent manner with CCX140 treatment. Hemoglobin A1c changes from baseline to Week 4 were: -0.09%, -0.09%, -0.23% (p= 0.045 vs. placebo), and -0.13% for the placebo, 5mg, 10mg CCX140, and pioglitazone groups, respectively. Patients in the 10 mg CCX140 group also experienced a statistically significant decrease from baseline in HbA1c indicating an improvement in glycemic control. Parameters pertaining to renal function were not assessed. Plasma CCL2 and circulating monocyte levels were unchanged by CCX140 treatment. Importantly, this study confirmed CCX140 was safe in diabetics, paving the way for advancement into Phase II trials in patients with nephropathy.

Patients Receiving CCX140 Had a Decrease in HbA1c from Baseline



Source: ChemoCentryx

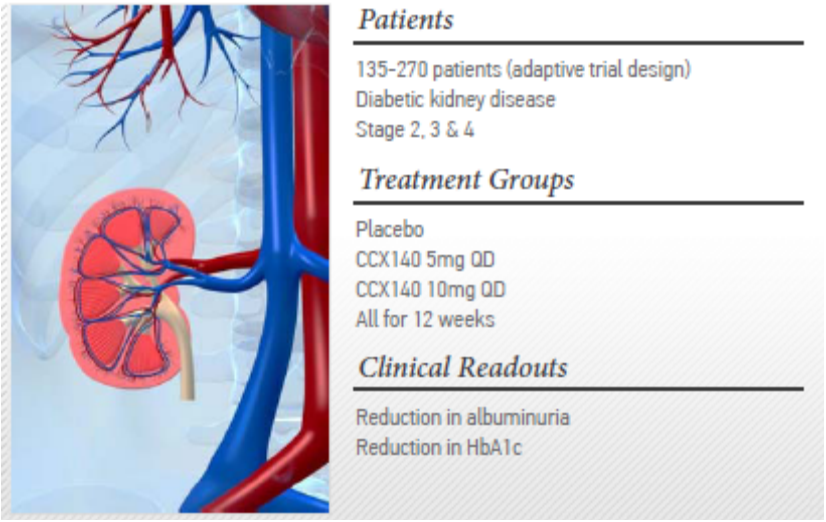
Success In Ongoing Phase II Trials Would Put CCX140 on the Map

CCX140 is currently in two phase II clinical trials in patients with diabetic nephropathy:

A randomized, double-blind, placebo-controlled trial is enrolling with a goal of 135 patients with DM and with evidence of diabetic nephropathy. The primary objective of this study is to evaluate the safety and tolerability of CCX140 in patients with active nephropathic disease. A key secondary objective is the evaluation of CCX140's effects on albuminuria and changes in serum HbA1c. The three treatment

groups will consist of placebo, 5mg, and 10mg of CCX140 treated for 12 weeks followed by a four-week follow-up period. This trial employs an adaptive design. An interim efficacy evaluation will be conducted, after which the sample size may be increased to up to 270 patients, and/or additional dose groups may be added, allowing for an increase in study power. Depending on whether or not the study is expanded, data could become available in late 2012 or early 2013.

Current Phase II Trial Design in Diabetic Patients with CKD



Source: ChemoCentryx

Another ongoing double-blind placebo-controlled trial conducted by ChemoCentryx is enrolling patients with Type II diabetes with evidence of renal disease despite stable doses of an ACE inhibitor or ARB. Patients will be randomized to placebo or 10mg of CCX140 and treated for 12 weeks followed by a four-week follow-up period. The trial's primary endpoint is to measure the effect of CCX140 treatment on urinary albumin excretion. Data are expected by late 2012.

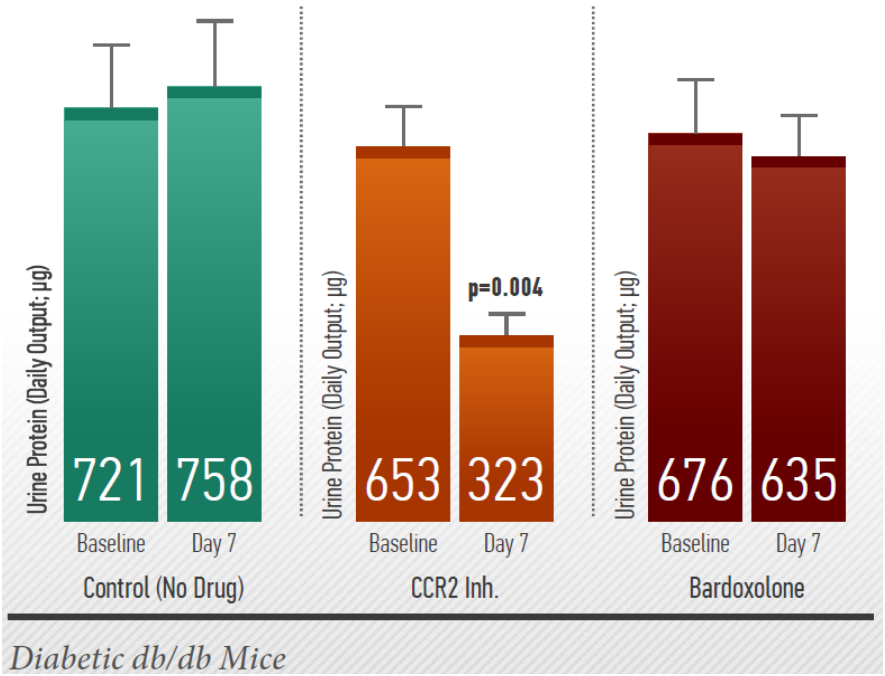
CCX140 Could Have Activity On Par With Bardoxolone

Bardoxolone methyl (Reata Pharmaceuticals and Abbott Labs), a modulator of inflammation, inhibits the proinflammatory nuclear factor κB pathway, possibly via Nrf2 activation. Bardoxolone currently is in Phase III studies in stage 4 CKD patients and top-line results are expected in mid 2013. In a Phase II study published in the *NEJM* (July 2011), CKD patients receiving bardoxolone had significant increases in mean GFR compared to placebo which were maintained throughout the 52 week study. The only adverse effects (AE) noted were muscle spasms, hypomagnesemia, mild elevations in liver enzymes, and GI effects.

These data and a lucrative collaboration with Abbott Labs for ex-U.S. rights have driven much optimism surrounding bardoxolone. Though much earlier in earlier in development, CCX140 appears to have activity on par with bardoxolone and might have a few differentiating properties. ChemoCentryx has compared the two compounds in a series of preclinical experiments. In a 7-day study in diabetic mice both appeared to improve many markers of renal output, but only CCX140 had a significant benefit in reducing proteinuria. This finding highlights the primary concern with bardoxolone that was also expressed by expert consultants at our 32nd

Annual Health Care conference in March. Some nephrologists are concerned that by increasing glomerular filtration rates without concomitant improvements in filtration capacity, bardoxelone may cause damage to renal filters, and worsened outcomes over time.

Comparison Of CCR2 Inhibition To Bardoxolone



Source: ChemoCentryx

Any Good CKD Drug Has $1B+ Revenue Potential

ChemoCentryx plans to retain commercial rights to CCX40 in North America and seek an ex-North America development partner following the release of Phase II data. The company believes that a modest-sized sales force can call on the approximately 8,300 U.S. nephrologists who treat diabetic nephropathy. Furthermore, CCXI expects a U.S. Phase III trial to be manageable in size, perhaps enrolling 1,500-1,800 patients with the standard composite endpoint of a doubling in creatinine, progression to ESRD, heart attack, or death.

Given ChemoCentryx has yet to establish produce proof-of-concept efficacy for CCX140 in its chosen indication, we do not include any revenue for the compound in our model. However, should CCX140 be shown to be effective, the revenue opportunity could be enormous. For illustrative purposes, we provide the following model of CCX140's potential in CKD. Moderate pricing ($2K/year) and modest penetration rates (up to 25% of the affected population) could support sales of $500M-$1B in each of the U.S. and Europe/Japan.

Potential CCX140 Revenue Build Up

	Year 1	Year 2	Year 3	Year 4	Year 5	Year 6
U.S. Market						
Number of Type-1 Diabetes Patients (MM)	2.60	2.68	2.76	2.84	2.93	3.01
Percent of Type-1 Diabetes with Renal Disease	30%	30%	30%	30%	30%	30%
Number of Type-1 Diabetes with Renal Disease (MM)	0.78	0.80	0.83	0.85	0.88	0.90
Number of Type-2 Diabetes Patients (MM)	23.40	24.10	24.83	25.57	26.34	27.13
Percent of Type-2 DM with Renal Disease	25%	25%	25%	25%	25%	25%
Number of Type-2 Diabetes with Renal Disease (MM)	5.85	6.03	6.21	6.39	6.58	6.78
Diabetics with Renal Disease (MM)	**6.63**	**6.83**	**7.03**	**7.24**	**7.46**	**7.69**
Diagnosed with Stage II,III,IV disease	**1.36**	**1.40**	**1.44**	**1.49**	**1.53**	**1.58**
CCX-140 penetration (%)	2%	9%	16%	20%	23%	24%
Patients treated /year (MM)	**0.03**	**0.12**	**0.21**	**0.27**	**0.31**	**0.32**
Price/yr	$2,000	$2,060	$2,122	$2,185	$2,251	$2,319
U.S. Sales Of CCX-140 ($MM)	$50	$250	$450	$600	$700	$750
y/y growth		*400%*	*80%*	*33%*	*17%*	*7%*
Europe and Japan						
Number of Type-1 Diabetes Patients (MM)	3.05	3.13	3.21	3.29	3.37	3.45
Percent of Type-1 Diabetes with Renal Disease	30%	30%	30%	30%	30%	30%
Number of Type-1 Diabetes with Renal Disease (MM)	0.92	0.94	0.96	0.99	1.01	1.04
Number of Type-2 Diabetes Patients (MM)	27.47	28.16	28.86	29.58	30.32	31.08
Percent of Type-2 DM with Renal Disease	25%	25%	25%	25%	25%	25%
Number of Type-2 Diabetes with Renal Disease (MM)	6.87	7.04	7.22	7.40	7.58	7.77
Diabetics with Renal Disease (MM)	**7.78**	**7.98**	**8.18**	**8.38**	**8.59**	**8.81**
Diagnosed with Stage II,III,IV disease	**1.60**	**1.64**	**1.68**	**1.72**	**1.76**	**1.81**
CCX-140 penetration (%)	1%	6%	13%	18%	22%	25%
Overall Patients treated /year (MM)	**0.01**	**0.08**	**0.18**	**0.25**	**0.30**	**0.34**
Price/yr (3%/yr inc)	$2,000	$2,000	$2,000	$2,000	$2,000	$2,000
Ex -US Sales ($MM)	$20	$150	$350	$500	$600	$675
y/y growth		*650%*	*133%*	*43%*	*20%*	*13%*
Total Sales ($MM)	**$70**	**$400**	**$800**	**$1,100**	**$1,300**	**$1,425**
y/y growth		*471%*	*100%*	*38%*	*18%*	*10%*

Source: Cowen and Company

CCX354 Carves A Path In Rheumatoid Arthritis

CCX354 is a novel orally-available inhibitor of the CCR1 chemokine receptor. CCR1 is highly expressed on monocytes and macrophages and responds to chemokine ligands such as the C6 superagonists. Synovial fluid aspirates have high levels of these chemokines, and studies have shown that they can attract CCR1-expressing leukocytes to the joints. By inhibiting CCR1, CCX354 reduces joint inflammation other signs and symptoms of RA. ChemoCentryx has successfully completed a Phase II study (CARAT-2 trial) in a population of 160 moderate-to-severe RA patients who were partially responsive to methotrexate (MTX). This study demonstrated that CCX354 is safe, well-tolerated, and active. Following completion of this trial GlaxoSmithKline (GSK) exercised its option to in-license CCX354. Under the terms of this agreement, ChemoCentryx received an exercise fee of $25MM, will be eligible for further sales and regulatory milestones, and will be entitled to double-digit royalties on net sales.

Rheumatoid Arthritis – A Scourge of Many

Rheumatoid Arthritis is a chronic systemic inflammatory disease of unknown cause. The most common presentation of a patient with RA is red, hot, swollen, and painful hands/wrists/feet/ankles. Patients usually experience morning joint stiffness that lasts over an hour, but which can usually be at least partially relieved with activity throughout the day. Patients may experience continual, yet vague joint pain, and in

severe cases may become incapacitated. Furthermore, tendon and ligament stretching caused by structural damage or direct bone and cartilage erosion can lead to joint destruction. Other organ systems can be affected, including hematologic, dermatologic, pulmonary, cardiac, ocular, vascular, and renal. Patients with RA have a decreased life expectancy from infection, cancer (especially lymphoma), and vascular disease. Cardiovascular morbidity and mortality are increased in patients with RA leading to sequelae such as Myocardial Infarction (MI), myocardial dysfunction, pericardial effusions. Mortality in patients with RA is roughly 2.5 times that of the general population, largely owing to infection, vasculitis, and poor nutrition.

A Normal vs. RA Affected Joint

Source: ChemoCentryx

The disease course is highly variable from patient to patient with some experiencing relatively limited disease and others suffering from chronically progressive illness. In order to make a prognosis, practitioners often follow titers of auto-antibodies (i.e. anti-rheumatoid factor [anti-RF] and anti-cyclic citrullinated peptide [CCP]) where high titers correlate with a worse prognosis. Other laboratory markers of poor prognosis include early radiologic evidence of bony injury, persistent anemia, elevated levels of the C1q component of complement, and the presence of anti-CCP antibodies.

In the United States, approximately 2MM people suffer from some form of rheumatoid arthritis, and the worldwide incidence is approximately 3 per 10,000. The overall prevalence rate is approximately 1%, which increases with age and peaks in 35 to 50 year-olds. RA affects all demographic groups, though not equally. In particular, women are affected more often than men by 2:1.

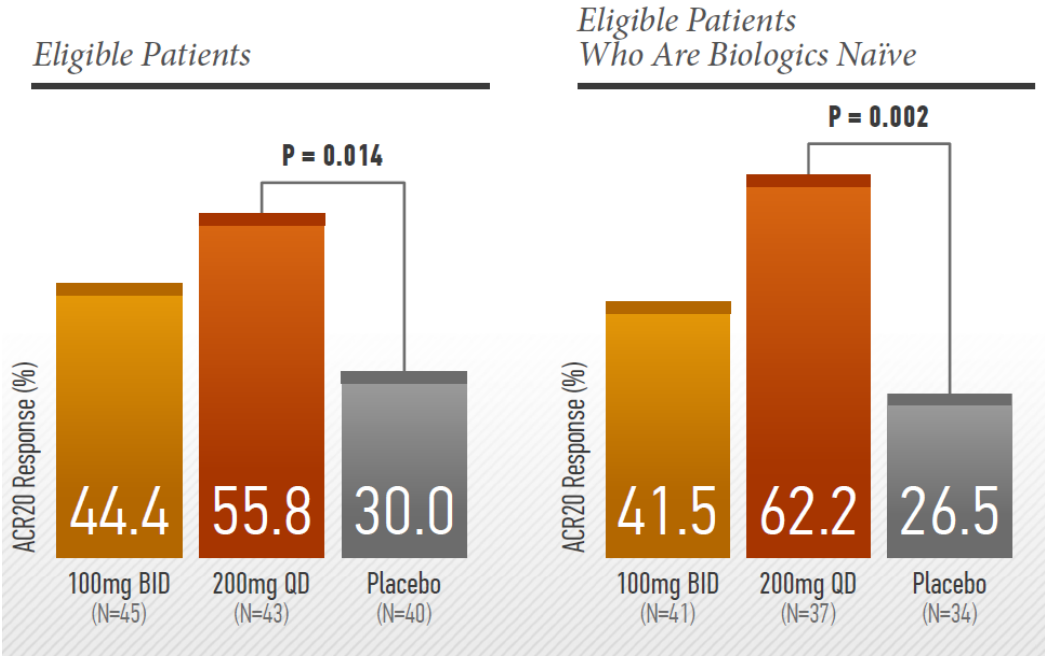
CCX354 Appears Safe, Efficacious In Early Trials

ChemoCentryx completed two Phase I studies testing CCX354 in 84 healthy volunteers. The first study was a single-ascending dose trial testing 1mg, 3mg, 10mg, 30mg, 100mg, and 300mg of CCX354 versus placebo, while the second study was a multiple-dose ascending trial that tested 3mg, 30mg, and 300 mg once per day and 10mg, 30mg, and 100mg twice per day. Both studies demonstrated that CCX354 has excellent safety and tolerability profiles. AEs were similar across treatment groups and did not appear to be dose-dependent. No SAEs were observed and no subjects had to be withdrawn due to AEs from either study.

ChemoCentryx also completed a Phase I/II trial evaluating the safety and tolerability of 100mg (given once per day or twice per day) and 200mg (given once per day) CCX354 vs. placebo in patients with stable RA on background methotrexate. Similar to the earlier Phase I studies, no patients treated with CCX354 had to be discontinued due to treatment emergent adverse events (TEAEs), nor did any patients have any signs of SAEs. Importantly, the study also demonstrated that the pharmacokinetic (PK) profiles of methotrexate and CCX354 were not significantly altered by co-administration of the drug.

At ACR 2011, ChemoCentryx reported positive results from its Phase II CARAT-2 (**C**CRT **A**ntagonist in **R**heumatoid **A**rthritis **T**rial-**2**) study. CARAT-2 was a double-blind, randomized trial that enrolled 160 patients with moderate-to-severe RA on a stable dose of methotrexate. Patients were randomized to placebo (n = 54), 100mg drug BID (n = 53), or 200mg drug QD (n=53) for 12 weeks. The primary endpoint was safety, and secondary endpoints included measures of disease activity such as the American College of Rheumatology (ACR) responses, Disease Activity Score 28, CRP, bone resorption markers, and ESR. ACR20, ACR50, and ACR70 responses refer to patients who receive a 20%, 50%, and 70% improvement score set by the American College of Rheumatology. ACR20 responses were 55.8%, 44.4%, and 30.0% of patients on 200mg QD, 100mg BID, and placebo, respectively, and the difference reached statistical significance for 200mg QD (p = 0.014). For patients who were biologics-naïve, the ACR20 responses were 62.2%, 41.5%, and 26.5% of patients on 200mg QD, 100mg BID, and placebo, respectively, and once again the difference reached statistical significance for 200mg QD (p = 0.002).

ACR20 Responses In Patients Treated With CCX354

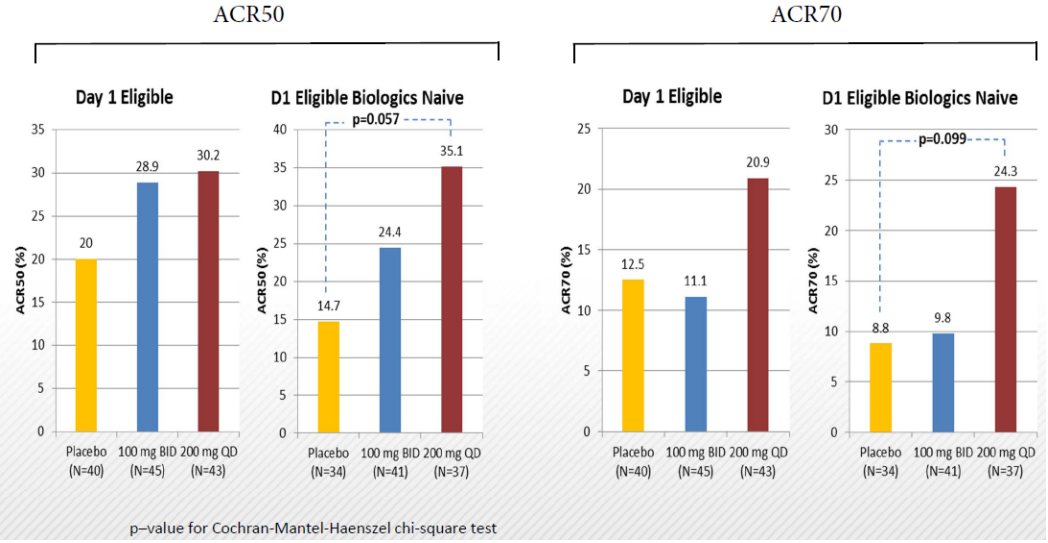


Source: ChemoCentryx

Although the high doses of CCX354 performed better than the low dose on ACR50 and ACR70 measurements, statistical significance was not reached. The greatest differences in response between control groups and CCX354 groups were in biologics-naïve patients. In these patients, the ACR50 response rates were 35.1%,

24.4%, and 14.7% for the 200mg QD, 100mg BID, and placebo groups, respectively (p=0.057). The ACR70 response rates for biologics-naïve patients were 24.3%, 9.8%, and 8.8% for the same groups, respectively (p=0.099).

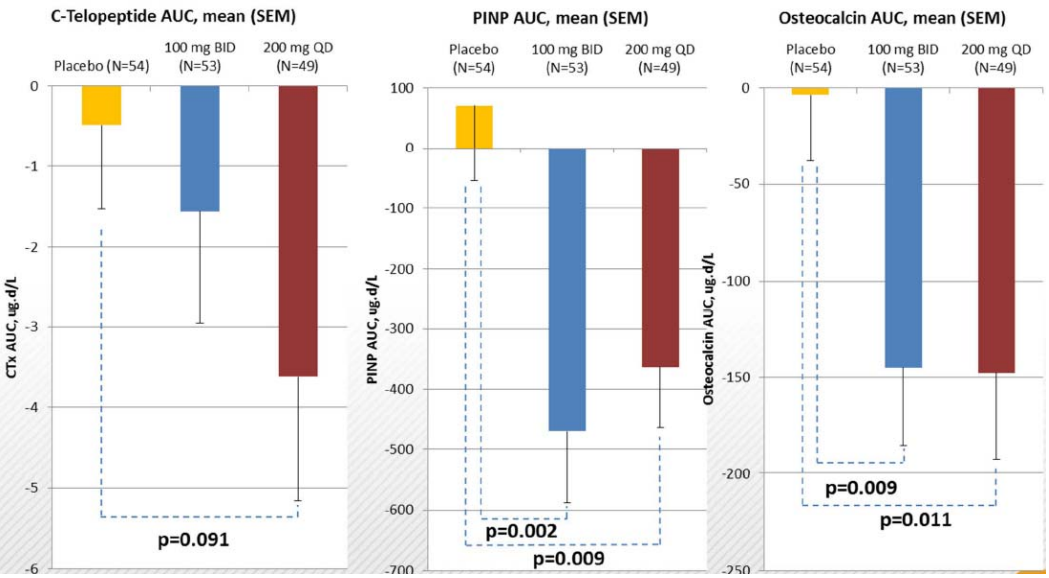
ACR50 and ACR70 Responses In Patients Treated With CCX354



Source: ChemoCentryx

The study also found that bone turnover markers were reduced compared to control, although not all measurements reached statistical significance. The markers examined were osteocalcin, C-telopeptide (CTx), and procollagen type I N-terminal peptide (PINP).

CCX354 Decreases Bone Turnover Markers



Source: ChemoCentryx

Notably, CARAT-2 showed that CCX354 was well-tolerated. No SAEs were reported in the 200 mg QD group, and although four SAEs were reported in the 100 mg BID group, none were considered related to CCX354 by the study investigators. Furthermore, no significant safety issues were reported with respect to laboratory parameters.

In January 2012, GSK exercised its option to CCX354 and will be solely responsible for further development and commercialization. RA trials are lengthy and costly, and we are pleased that GSK will be funding future development in a field that can be risky (biologic therapies have established a high bar for safety and efficacy). We expect GSK to begin a Phase IIb trial on CCX354 in late 2012.

The C5aR Antagonism Program – CCX168 for Vasculitis

ChemoCentryx is also developing a C5a receptor antagonist called CCX168. The small molecule decreases complement activation as well as leukocyte recruitment to sites of activation. CCX168 has completed a Phase I clinical trial showing good tolerability up to 100 mg. ChemoCentryx launched a Phase II proof-of-concept trial in patients with Anti-Neutrophil Cytoplasmic Antibody (ANCA) Associated Vasculitis (also known as AAV) in Q4:11 and expects to complete it in H2:12 with data readouts shortly thereafter. If the trial is successful, GSK may exercise its option to license and commercialize the drug.

ANCA Associated Vasculitis

AAV encompasses several different disease conditions including renal limited vasculitis, microscopic polyangiitis, and Wegener's granulomatosis, all of which are caused by the immune system attacking small vessels in the body. While the exact mechanism of pathogenesis is incompletely understood, recent developments have shed some light on underlying processes which may provide potential therapeutic targets. For example, a study published in the *NEJM* in 2010 suggested that monoclonal antibodies directed against CD-20 may be able to induce remission in patients with AAV. In the clinic, these agents are used by some clinicians for patients with refractory cases of AAV.

If left untreated, AAV may lead to renal and/or pulmonary failure and is often fatal. AAVs are currently treated with high-dose corticosteroids and cyclophosphamide, azathioprine, mycophenolate mofetil, rituximab, and plasma exchange in severe cases. Since the 1970s, few advances in treatment have been made, and available treatment regimens are associated with substantial morbidity and mortality secondary to infection and other severe side effects. Thus, there remains an unmet medical need for AAV therapies which are safe, effective, and corticosteroid-sparing.

CCX662 for Glioblastoma Multiforme

CCX662 is a selective small molecule inhibitor of CXCR7. The drug is currently in preclinical development to treat glioblastoma multiforme (GBM). CXCR7 is highly expressed in GBM and is thought to be involved in promoting tumor growth. Interestingly, studies have shown that CXCR7 expression is stimulated by VEGF, suggesting that it may play a role in tumor angiogenesis. Furthermore, *in vivo* preclinical studies have shown that tumor growth and angiogenesis are suppressed by CXCR7 inhibition. A Phase I clinical trial is expected to begin in H2:12.

Addendum

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Ticker	Company Name
CCXI	ChemoCentryx

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