Five Prime Therapeutics

(FPRX-NASDAQ)

Stock Rating: **Outperform**
Industry Rating: **Outperform**

October 14, 2013

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Initiating With Outperform Rating

Investment Thesis

We are initiating coverage of Five Prime Therapeutics with an Outperform rating and $24 price target. Our favorable rating is driven by positive expectations for lead FGFR ligand trap FP-1039 in solid tumors and potential option value from a broad biologic drug pipeline and discovery platform. We believe that rational development of FP-1039 in targeted cancer patients with FGFR amplification could ultimately support $1.7B in peak sales, with significant data flow over the next 12-18 months to de-risk the program. While earlier in development we believe that FGFR2b antibody FPA144 could drive significant value in gastric cancer with phase 1 planned for 2H14 and that CSF1R antibody FPA008 could attract significant large pharma interest as it moves into phase 1 by year-end 2013 for rheumatoid arthritis (RA). Finally, a broad protein library covering all extracellular proteins and high-throughput biologics screening should support long-term productivity in generating cancer and immunology drug candidates.

Forecast & Valuation

We forecast losses per share of $2.17 in 2013 and $0.71 in 2014. We expect decreasing losses annually until 2017, when we estimate initial profitability with EPS of $0.38. For the period 2018-2020, our valuation year, we forecast EPS of $2.28, $3.85, and $5.65, respectively. We arrive at our $24 price target by applying a 20x multiple to 2020 EPS estimate of $5.65 and discounting at 35%.

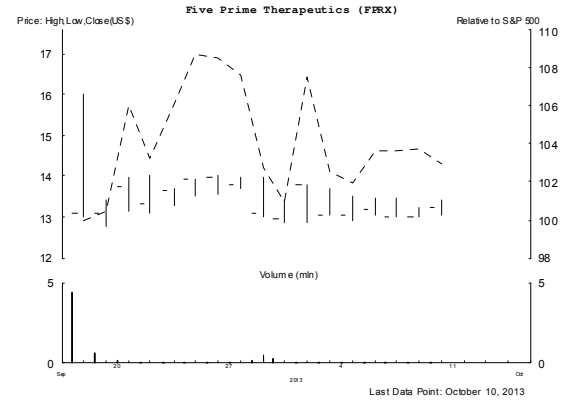
Recommendation

We rate FPRX shares at Outperform.

Securities Info

Price (11-Oct)	**$13.19**	**Target Price**	**$24.00**
52-Wk High/Low	$16/$13	Dividend	--
Mkt Cap (mm)	$221	Yield	--
Shs O/S (mm, BASIC)	16.7	Float O/S (mm)	5.5
Options O/S (mm)	na	ADVol (30-day, 000s)	377

Price Performance



Valuation/Financial Data

(FY-Dec.)	2011A	2012A	2013E	2014E
EPS GAAP	$0.47	-$2.50	-$2.17	-$0.71
P/E			nm	nm
First Call Cons.				
FCF	NA	NA	NA	NA
P/FCF			na	na
EBITDA ($mm)	$20	-$28	-$31	-$13
EV/EBITDA			nm	nm
Rev. ($mm)	$65	$10	$13	$32
EV/Rev			10.1x	4.0x

Quarterly EPS	**1Q**	**2Q**	**3Q**	**4Q**
2012A	NA	NA	NA	NA
2013E	-$0.64A	-$0.64A	-$0.44	-$0.44

Balance Sheet Data (30-Jun)

Net Debt ($mm)	-$94	Total Debt/EBITDA	nm
Total Debt ($mm)	$0	EBITDA/IntExp	na
Net Debt/Cap.	na	Price/Book	33.0x

Notes: All values in US$.
Source: BMO Capital Markets estimates, Bloomberg, Thomson Reuters, and IHS Global Insight.

Please refer to pages 67 to 70 for Important Disclosures, including the Analyst's Certification.

Investment Thesis

We believe that Five Prime Therapeutics has a best-in-class biologics discovery effort and multiple opportunities to drive value across an emerging pipeline in oncology and immunology. With primary focus on FGFR inhibitor FP-1039 in FGFR amplified tumors, we believe that upside potential could emerge from multiple indications, including breast cancer, lung cancer, and head and neck cancer. Option value beyond FP-1039 exists from more targeted development of FGFR2b inhibitor FP144A in gastric cancer and CSF1R inhibitor FPA008 in rheumatoid arthritis.

Key Investment Highlights

- FPRX has a broad protein library in biotech covering all 5,600+ extracellular proteins for purposes of high throughput biologics screening.
- Productivity of the FPRX biologic drug discovery platform has been evidenced by a pipeline of 3 biologic drug candidates targeting diverse cancer and immunology indications.
- Lead biologic drug candidate FP-1039 could represent a best-in-class FGFR inhibitor in a large group of cancer patients with FGFR amplification.
- Opportunity for FP-1039 in FGFR amplified tumors could approach $1.7B in peak sales across lung cancer, head and neck cancer and breast cancer.
- Data flow from a multi-group phase 1b study in patients with FGFR amplified tumors should support proof-of-concept for FP-1039 over the next 12 months.
- More targeted development of FGFR2b antibody FPA144 in FGFR2b amplified gastric cancer could drive additional value as phase 1 study is initiated in 2H14.
- Initiation of a third program by year-end 2013 with CSF1R antibody FPA008 in rheumatoid arthritis (RA) should attract large pharma interest, given prior validation of macrophage targeting therapeutics in RA.
- We expect additional biologic drug candidates to emerge from the company's high through-put screening and believe that novel targets could emerge to compliment existing cancer immunotherapies like YERVOY and PD-1 antibodies.

Review of Financials

We estimate initial launch of FP-1039 in the US in 2016 with $58M in estimated sales and $8.7M in FP-1039 royalties, assuming a 15% royalty rate on future partnership. We estimate US sales increasing to $865M in 2025 with royalties of $129.7M. We estimate peak sales for FP-1039 in the EU of $865M with $129.7M in royalties. We forecast initial profitability for FPRX in 2017 at $0.38 with 2018 to 2025 EPS estimates of $2.28, $3.85, $5.65, $7.12, $8.49, $8.59, $8.70 and $8.80, respectively.

Valuation

We base our valuation of FPRX on a relative value P/E multiple on future earnings. We arrive at our $24 price target by applying a 20x multiple to 2020 EPS estimate of $5.65 and discounting at 35% per year.

Exhibit 1: Five Prime Therapeutics Comps

EARLY STAGE ONCOLOGY COMPANIES						
Company	**Ticker**	**Market Cap (M)**	**Cash (M)**	**EV (M)**	**Stage of Development**	**Therapeutic Focus**
Ambit Biosciences	AMBI	$310.3	$85.3	$226.8	Phase 2	Oncology
Array BioPharma	ARRY	$672.4	$108.2	$663.2	Phase 2	Oncology
Curis	CRIS	$319.8	$57.1	$293.2	Phase 1	Oncology
Epizyme	EPZM	$1,009.9	$148.7	$861.2	Phase 1	Oncology
KaloBios	KBIO	$139.9	$63.7	$86.1	Phase 1	Oncology
OncoMed Pharmaceuticals	OMED	$421.1	$60.2	$360.8	Phase 1	Oncology
Stemline Therapeutics	STML	$468.3	$92.7	$375.6	Phase 2	Oncology
Verastem	VSTM	$254.4	$57.5	$197.0	Phase 2	Oncology
Mean		$449.5		$383.0		
Median		370.5		327.0		
Five Prime Therapeutics	FPRX	$209.8	$28.2	$181.6	Phase 1	Oncology

FIVE PRIME THERAPEUTICS SUM-OF-THE-PARTS					
Target	**Indication**	**Launch Year**	**Peak Sales ($M)**	**Probability**	**NPV ($M)**
FP-1039	NSCLC	2016	$120.3	25%	$107.7
FP-1039	SCCHN	2016	45.9	25%	41.8
FP-1039	Breast CA	2016	74.3	25%	67.6
FP-1039	SCLC	2016	18.9	25%	17.2
Total					$234.4

Source: Company reports, Thomson Reuters, and BMO Capital Markets

Overview

Five Prime Therapeutics is a development-stage biotechnology company focused on discovering and developing novel protein therapeutics for the treatment of cancer and inflammatory diseases. Five Prime's growth potential lies in an industry-leading protein therapeutics discovery platform, as well as three protein therapeutic candidates: FP-1039, FPA008, and FPA144.

Five Prime has developed the industry's most comprehensive protein library consisting of more than 5,600 human extracellular proteins, representing nearly all of the potential protein therapeutic targets in the body. To fully leverage this unique library, Five Prime has also developed high-throughput *in vitro* and *in vivo* screening technologies and technologies for identifying ligand-receptor interactions. Together, these components make up a powerful protein drug discovery engine. Through this platform, Five Prime has identified novel therapeutic targets and developed a pipeline of product candidates for cancer and inflammatory diseases.

The company's most advanced product candidate, FP-1039, or GSK3052230, is a ligand trap developed in partnership with GlaxoSmithKline (GSK). FP-1039 "traps" and neutralizes fibroblast growth factors (FGFs), a family of proteins that normally regulate cell proliferation but can also promote cancer development. FP-1039 is designed to only block FGF family members with cancer-promoting activities, and therefore may have a better tolerability profile than other FGF-targeting agents that indiscriminately block all FGFs. In preclinical studies, FP-1039 demonstrated anti-tumor activity in a variety of different xenograft models and enhanced anti-tumor activity of cytotoxic or targeted anti-cancer drugs. In a phase 1 study in 39 patients with various tumors (not selected for FGFR1 gene amplification), 17 patients had stabilization of tumor growth (stable disease) and one patient with hormone-resistant prostate cancer who had progressed rapidly on chemotherapy achieved tumor reduction of 20% following treatment with FP-1039, with a stable disease duration of approximately seven months. FP-1039 was described as well tolerated in phase 1 experience and was not associated with hyperphosphatemia as was observed in clinical trials of small-molecule pan-FGFR inhibitors.

In March 2011, Five Prime entered into a license and collaboration agreement that granted Human Genome Sciences (HGS), which was later acquired by GSK, an exclusive license to develop and commercialize FP-1039 and other FGFR1 fusion proteins in the US, the EU and Canada. In July 2013, GSK initiated an open-label phase 1b multi-arm clinical trial of FP-1039 either as a monotherapy or in combination with chemotherapies in cancer patients with abnormally high levels of FGFR1. Preliminary data from the phase 1b trial are expected in 2H14. GSK is responsible for the development and commercialization of FP-1039 in the US, the EU and Canada, and Five Prime has an option to co-promote FP-1039 in the US.

Exhibit 2: Five Prime Pipeline

Program	Product	Indication	Research	Pre-Clinical	Phase 1	Phase 1b	Phase 2
FP-1039 GSK 3052230	FGF Ligand Trap	Multiple Solid Tumors	Phase 1b start expected Q3:13				
FPA008	CSF1R Antibody	Autoimmune Disease	Phase 1 start planned Q4:13				
FPA144	FGFR2b Antibody	Gastric Cancer	Phase 1 start planned H2:14				
Multiple Candidates	Antibodies & Ligand Traps	Cancer Immunotherapy, Steroid Resistant Asthma					

Companion diagnostic strategy for each program

Source: Five Prime Therapeutics

FPA144 is an antibody against FGF receptor 2b (FGFR2b) and is currently under development for the treatment of gastric cancer. In preclinical studies, FPA144 was highly effective in inhibiting the growth of gastric tumors that expressed high levels of FGFR2b. Five Prime plans to initiate in 2H14 a phase 1 clinical trial of FPA144 in patients with tumors expressing high levels of FGFR2b, with preliminary data expected by year-end 2015.

FPA008 is an antibody against colony stimulating factor-1 receptor (CSF1R), currently in development for the treatment of rheumatoid arthritis. CSF1R plays an important role in the survival and function of certain immune cells known as monocytes and macrophages. By inhibiting CSF1R activation, FPA008 prevents the production of multiple proinflammatory cytokines, such as tumor necrosis factor (TNF), interleukin-6 (IL-6) and interleukin-1 (IL-1). Each of these proinflammatory cytokines has been successfully targeted by approved therapeutics for various inflammatory disorders such as rheumatoid arthritis (RA). Therefore FPA008 could potentially be effective in treating multiple inflammatory conditions. In addition, FPA008 may have advantage over existing therapies for RA because FPA008 directly inhibits bone-destroying cells known as osteoclasts. In preclinical studies, incubation with FPA008 reduced levels of inflammatory cytokines TNFα, IL-6 and IL-1β in biopsy samples of inflamed joints from RA patients. Five Prime plans to initiate a phase 1 trial for FPA008 by year-end 2013 and expect preliminary data by year-end 2014.

For all three lead product candidates, Five Prime is pursuing companion diagnostics to allow selection of patients most likely to benefit from treatment, a strategy that could allow acceleration of clinical development.

Five Prime's technology platform has attracted numerous partnerships with leading biopharmaceutical companies and generated $220 million in funding since 2006. Ongoing

collaborations include the FP-1039 partnership with GSK with contingent payments up to $435 million, and additional discovery collaborations with GSK in muscle and respiratory diseases and with UCB Pharma in fibrosis and CNS diseases.

Five Prime's key strategies for future development include focus on protein therapeutics to treat cancer and inflammatory diseases, pursuing indications and patients populations in which drug candidates can be assessed early in clinical development (potentially in phase 1 trials), pursuing the use of companion diagnostics, retaining rights and eventually building commercial capabilities in targeted specialty markets, and pursuing additional licensing and collaboration activities to generate funding.

Platform Focused on High-Throughput Biologics Screening and Large Protein Library

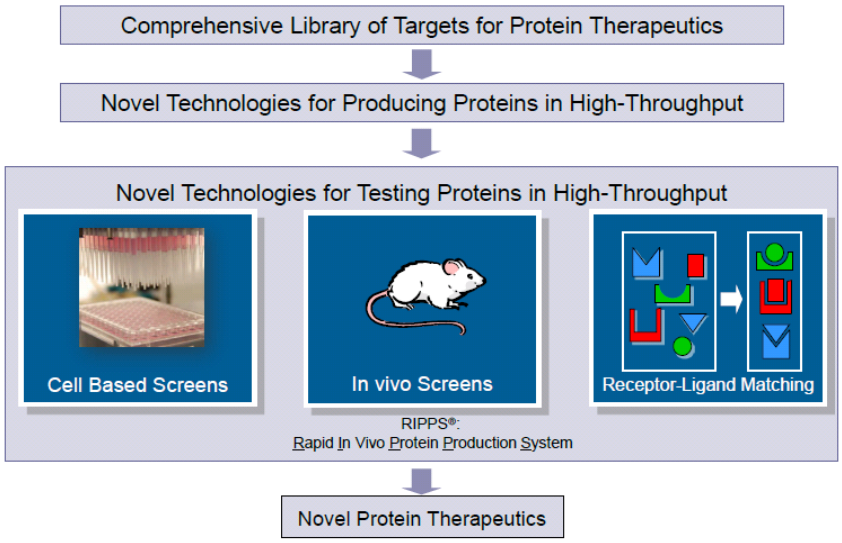
Five Prime's platform technology is among the most impressive in the industry, consisting of a large extracellular protein library and proprietary high-throughput *in vitro* and *in vivo* screening technologies. The power of this unique platform has been demonstrated by the discovery of a novel cytokine, successful identification of novel therapeutic targets, and a number of discovery collaborations with leading pharmaceutical companies.

There are more than 5,600 extracellular proteins in the body that can potentially be targeted by protein therapeutic agents. Of these, only 30 or so are targeted by currently marketed protein drugs in cancer and inflammatory diseases. The traditional protein drug discovery process is characterized by studying a single or a small number of proteins at a time. Five Prime has developed over a period of seven years a platform that improved the speed and throughput of the protein drug discovery process. The platform consists of two components: a protein library and a set of proprietary technologies for producing and testing thousands of proteins at a time (Exhibit 3).

Five Prime's proprietary library of fully functional human extracellular proteins is the most comprehensive in the industry. The library comprises more than 5,600 human proteins derived from more than 100 distinct human tissues. The members of the library span a wide spectrum in terms of degree of characterization, ranging from those with well known functions (such as TNFα and HER2, which are the targets of REMICADE and HERCEPTIN, respectively) to novel protein variants that are not disclosed in the public domain.

Five Prime has attributed its success in making a comprehensive library to a proprietary technology that allows efficient capturing of full length cDNAs. cDNAs are copies of genes. Just as genes can direct production of protein in the body, cDNAs can be used to reproduce the same protein in the laboratory. However, traditional technologies are not efficient in copying the full length of genes, such that one end of the gene can be missing in the cDNA copy. Of the two ends, termed 5 prime and 3 prime, the 5 prime end is more critical and without it the protein cannot be made (whereas cDNAs missing the 3 prime end could still generate proteins in truncated forms). Five Prime has developed a proprietary technology that can capture more cDNAs with intact 5 prime ends, resulting in a collection of more complete cDNAs, which can then be used to generate a more complete collection of proteins.

Exhibit 3: Five Prime's Protein Drug Discovery Platform

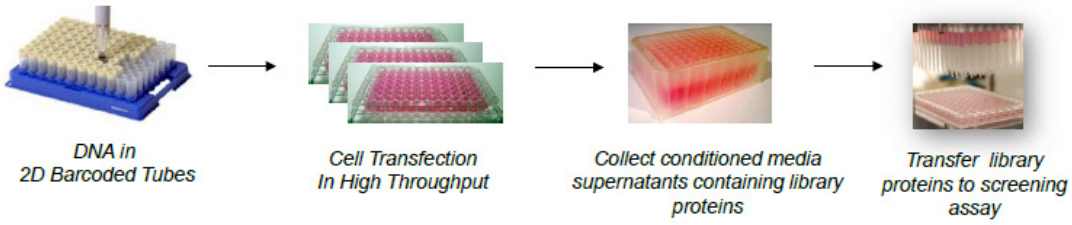


Source: Five Prime Therapeutics

In addition to the protein library, Five Prime has also developed a suite of technologies for high-throughput screening. These technologies encompass *in vivo* and *in vitro* protein production, cell-based screening assays, and receptor-ligand matching technologies.

Five Prime's protein production system, which was developed over several years, has the capacity to produce approximately 2,000 proteins per week at therapeutically relevant amounts and with a high level of consistency, far surpassing traditional throughput levels (Exhibit 4). Five Prime can produce its entire protein library in less than three weeks, whereas in comparison, it would take years to produce the same library using typical methods producing one or a few proteins at a time.

Exhibit 4: Five Prime's High Throughput Protein Production System



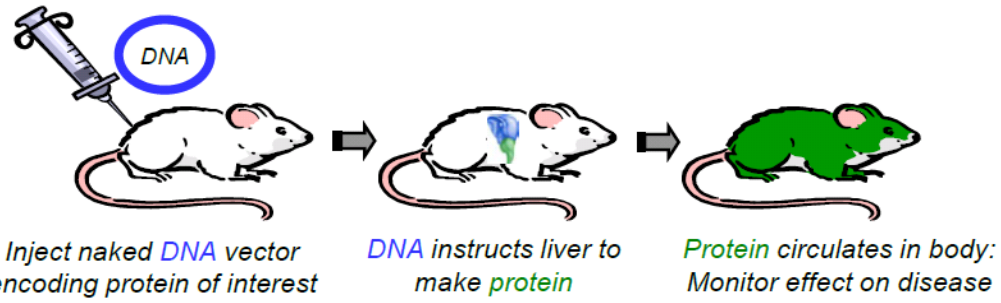
Source: Five Prime Therapeutics

To identify therapeutic targets among the proteins in the library, Five Prime has designed complex cell-based screens that best mimic the biological processes underlying the disease of interest. For example, the screens could involve diseased primary human cells or even rare stem cells. To date, Five Prime has performed approximately 100 cell-based screens using approximately 50 different cell types. The screening process is automated using equipment and

software that were designed and built in-house. Using these cell-based screens, Five Prime has discovered the target that forms the basis of the FPA008 program and numerous other targets for diseases including severe asthma, pulmonary fibrosis, muscle disease, and cancer.

Five Prime has also developed the Rapid In Vivo Protein Production System, or RIPPS, whereby DNA vectors are injected into mice to instruct liver cells to make the protein, which is then released into circulation (Exhibit 5). According to Five Prime, RIPPS could produce high blood levels of protein for weeks after just one injection. RIPPS has several obvious advantages over traditional screening methods. RIPPS bypasses several costly and time-consuming steps required for conventional *in vivo* testing of efficacy and safety, including protein expression, scale up, purification, characterization and formulation of each protein. RIPPS also enables direct testing of protein therapeutics in virtually any rodent model of disease, including those without corresponding cell-based screening assays. Using RIPPS, Five Prime has identified and validated dozens of new targets and protein drug candidates in rodent models of cancer, inflammatory disorders, muscle disease and other conditions.

Exhibit 5: Rapid In Vivo Protein Production System (RIPPS)



Source: Five Prime Therapeutics

Five Prime's protein library contains both protein ligands and extracellular domains of cell surface receptors. Five Prime is therefore uniquely positioned to identify unknown ligand-receptor pairs. Such information is highly valuable for elucidating disease pathways and for developing targeted therapies. Using this technology, Five Prime has identified the target for FPA008 and several new ligands, including two new hormones. In addition, Five Prime has gathered an extensive database of protein function as it tests each of the proteins in the library in numerous screens on different cell types. By documenting how each protein behaves in different screens, this database provides insights into whether a protein is involved in a specific disease process or has a broader set of activities.

In summary, the strength of Five Prime's drug discovery platform lies in (1) its ability to identify novel protein targets and therapeutics that may have little or no previously known biological functions; (2) its ability to determine the best protein target for a particular disease by screening and comparing nearly all medically important targets; and (3) its ability to test thousands of proteins at a time. The discovery platform and the scientific expertise accumulated in building such a platform represent a significant barrier to entry for any potential competitors.

Pipeline Focus on Cancer and Autoimmune Disease

Five Prime's most advanced pipeline candidates include FP-1039 for the treatment of FGFR1 gene-amplified tumors, such as squamous non-small cell lung cancer, FPA144 for the treatment of FGFR2 gene-amplified tumors, such as gastric cancer, and FPA008 for the treatment of rheumatoid arthritis (RA) and other inflammatory diseases.

FP-1039 is a FGF ligand trap developed in partnership with GlaxoSmithKline (GSK). FP-1039 is designed to only block FGF family members with cancer-promoting activities, and therefore may have a better tolerability profile than other FGF-targeting agents that indiscriminately block all FGFs. In preclinical studies, FP-1039 demonstrated anti-tumor activity in a variety of different xenograft models and enhanced anti-tumor activity of cytotoxic or targeted anti-cancer drugs. In a phase 1 study in 39 patients with various tumors (not selected for FGFR1 gene amplification), 17 patients had stabilization of tumor growth (stable disease) and one patient with hormone-resistant prostate cancer who had progressed rapidly on chemotherapy achieved tumor reduction of 20% following treatment with FP-1039, with a stable disease duration of approximately seven months. FP-1039 was described as well tolerated in phase 1 experience and was not associated with hyperphosphatemia as was observed in clinical trials of small molecule FGFR inhibitors. In July 2013, Partner GSK initiated an open-label phase 1b multi-arm clinical trial of FP-1039 either as a monotherapy or in combination with chemotherapies in cancer patients with abnormally high levels of FGFR1. Preliminary data from the phase 1b trial are expected in 2H14. GSK is responsible for the development and commercialization of FP-1039 in the US, the EU and Canada. Five Prime has rights to FP-1039 in the rest of world and has an option to co-promote FP-1039 in the US.

FPA144 is an antibody against FGF receptor 2b (FGFR2b) and is currently under development for the treatment of gastric cancer. In preclinical studies, FPA144 was highly effective in inhibiting the growth of gastric tumors that expressed high levels of FGFR2b. Five Prime plans to initiate in 2H14 a phase 1 clinical trial of FPA144 in patients with tumors expressing high levels of FGFR2b, with preliminary data expected by year-end 2015.

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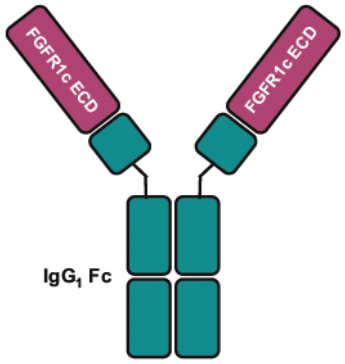
FGF Ligand Trap FP-1039 Progressing In Phase 1b for Multiple Tumor Types

FP-1039, or GSK3052230, is a so-called ligand trap, consisting of the extracellular domains of human FGF receptor 1 (FGFR1) linked to the Fc region of human IgG1 (Exhibit 6). FP-1039 binds to and sequesters multiple fibroblast growth factors (FGFs), and prevents them from activating multiple FGF receptors (FGFRs).

Exhibit 6: Domain Structure of FP-1039



Source: Five Prime Therapeutics

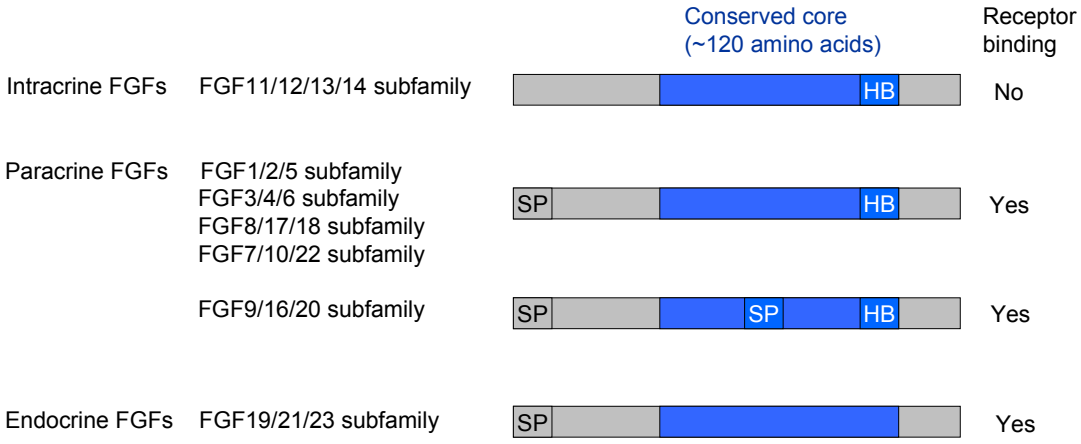
FGF Signaling Is Complex and Involves Multiple Ligands, Receptors and Pathways

The FGF-FGFR signaling pathway is fundamentally involved in early development as well as in the pathophysiology of certain cancers. Targeting of FGFR-FGF signaling has been thwarted by the complex nature of the pathway and the sheer number of ligands and receptors (22 ligands and 4 receptors in humans) involved as well as the myriad of over-lapping ligand-receptor interactions. Drug developers face the difficult task of tackling significant redundancy on the one hand (which requires a broad-acting agent that targets multiple ligands or receptors) and on the other hand the potential of "collateral damage" by unintended blockade of certain ligand-receptor interactions. In this regard, Five Prime's FP-1039 seems to have struck the right balance and therefore holds a favorable competitive position in relation to other classes of FGF-targeted therapies.

FGFs and their receptors (FGFRs) play an important role in a wide range of biological processes, including developmental events such as brain patterning, morphogenesis and limb development, as well as angiogenesis, wound repair and endocrine functions in adults. Perturbations of the FGF signaling pathway are associated with various diseases, including developmental, metabolic, neurological diseases, and cancer.

In humans, FGFs make up a large family of 22 proteins: FGF1 – FGF14 and FGF16 - FGF23. (There is no human FGF15, which is an ortholog of human FGF19 in some other species). FGF polypeptides are 150 to 300 amino acids long and have a conserved core of approximately 120 amino acids. Like other growth factors, FGFs act over certain distance to trigger signaling events in other cells. According to the distance of their reach, FGFs can be classified as intracrine (inside the cell), paracrine (nearby cells and tissues), and endocrine (long distance).

Exhibit 7: Schematic Representations of FGF Family Proteins and Their Structures



SP: Signal Peptide (for extracellular secretion); HB: Heparin-binding Domain

Source: BMO Capital Markets; adapted from Itoh N and Ornitz D, *J Biochem* 2011, 149:121

Endocrine FGFs (also called hormonal FGFs) include FGF19, FGF21, and FGF23, and these FGFs travel long distances to influence far away tissues. For example, FGF23 is made in bone and circulates through the blood to regulate vitamin D and phosphate metabolism in kidney. In contrast, intracrine FGFs, including FGF11-FGF14, are not secreted extracellularly and act as intracellular molecules instead. The principal targets of intracrine FGFs are the intracellular domains of voltage gated sodium channels.

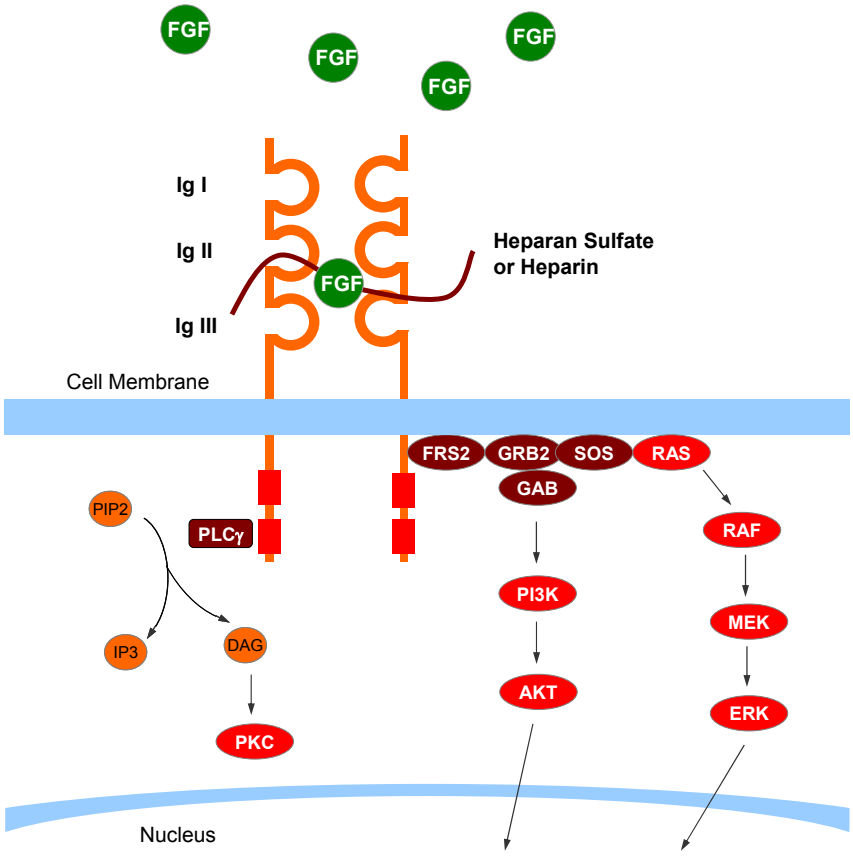
Paracrine FGFs mediate biological responses in nearby cells and tissues. These molecules cannot travel long distance because they bind readily to heparan sulfate proteoglycans (HSPG), which is abundant in the extracellular matrix. This is in contrast to endocrine FGFs, which do not bind to HSPG with high affinity.

Paracrine FGFs can be further classified into five subfamilies: FGF1/2/5; FGF3/4/6; FGF7/10/12; FGF8/17/18; and FGF9/16/20. Most paracrine FGFs are secreted proteins, although FGF1 and FGF2 lack the amino acid sequence that marks a protein for secretion (signal peptide) and are likely to be released only from damaged cells.

There are four FGF receptors, FGFR1-FGFR4, and they are receptor tyrosine kinases. Each FGFR consists of an extracellular ligand-binding domain with three immunoglobulin-like (Ig) domains (I, II, and III), a transmembrane domain, and a split intracellular tyrosine kinase domain. The first Ig domain, absent in certain receptor isoforms, is thought to have an auto-inhibitory function, whereas the second and third Ig domains are critical for ligand binding and specificity. Alternative splicing in the second half of immunoglobulin-like domain III of FGFR1-3 yields two major isoforms (IIIb and IIIc). Therefore there are a total of 7 major FGFR proteins: FGFRs 1b, 1c, 2b, 2c, 3b, 3c and 4. Each of these FGFRs binds to a selective but overlapping set of FGF ligands.

Activation of FGFRs requires binding of the FGF ligand and heparan sulfate (HS), which acts as a cofactor. Binding of endocrine FGFs (FGF19, FGF21, and FGF23) to FGFRs require an additional co-receptor, αKlotho or βKlotho. Once the FGF-FGFR-HS complex forms, FGFR undergo dimerization, followed by transphosphorylation of FGFR's intracellular tyrosine kinase domain and carboxy-terminal tail. Activated FGFRs subsequently phosphorylate two main substrates, phospholipase Cγ (PLCγ) and FGFR substrate 2 (FRS2). These substrates in turn activate the RAS-RAF-MAPK pathway, the PI3K-AKT pathway, and the STAT-dependent pathway.

Exhibit 8: The FGF-FGFR Signaling Pathway



Source: BMO Capital Markets; adapted from Beenken A. and Mohammadi M, *Nat Rev Drug Discov* 2009, 8:235

The binding specificities between the 15 paracrine FGFs and the 7 major FGFRs are complex and depend on both the primary sequences of these molecules and the temporal and spatial expression patterns of FGFs, FGFRs, and heparan sulfate. The expression of alternative-splice isoforms of FGFRs follows a general pattern: the b isoforms are mainly expressed in epithelial tissues and the c isoforms in mesenchymal tissues. In physiological conditions, ligand-receptor interactions seem to occur across tissue types, such that a ligand expressed in the epithelium will activate a mesenchymal receptor, and vice versa. Several ligands, including FGF1, can bind promiscuously to both b and c isoforms of certain FGFRs. In addition, in cancers in which FGFs are overexpressed, binding specificities may be altered.

Exhibit 9: FGF-FGFR Binding Specificities

			FGFR1b	FGFR1c	FGFR2b	FGFR2c	FGFR3b	FGFR3c	FGFR4
Paracrine	FGF1/2/5 Subfamily	FGF1	x	x	x	x	x	x	x
		FGF2	x	x		x		x	x
		FGF5		x		x			
	FGF3/4/6 Subfamily	FGF3	x		x				
		FGF4		x		x		x	x
		FGF6		x		x			x
	FGF7/10/22 Subfamily	FGF7			x				
		FGF10	x		x				
		FGF22	x		x				
	FGF8/17/18 Subfamily	FGF8		x		x		x	x
		FGF17		x		x		x	x
		FGF18				x		x	x
	FGF9/16/20 Subfamily	FGF9				x	x	x	x
		FGF16				x	x	x	x
		FGF20		x	x	x	x	x	x
Intracrine		FGF11							
		FGF12							
		FGF13							
		FGF14							
Endocrine		FGF19*		x		x		x	x
		FGF21*		x		x		x	x
		FGF23*		x		x		x	x

* Binding of endocrine FGFs to FGFRs requires co-receptor, alpha- or beta-Klotho

Source: BMO Capital Markets; adapted from Ornitz D et al., J Biol Chem, 271:15292 and Zhang X et al., J Biol Chem 281:16694

Rationale for Targeting the FGF-FGFR Signaling Pathway for Cancer Treatment

Given the role that FGF-FGFR signaling plays in cell proliferation and survival, it should come as no surprise that mutation and overexpression of FGFs and FGFRs are observed in cancer. Indeed, FGFRs have been identified as among the most frequently mutated kinases in human cancers. Dysregulation of FGF pathway typically involves activating mutations, gene amplifications, and to a lesser extent, chromosomal translocations and altered splicing patterns.

The genomic locus encompassing the *FGFR1* gene is frequently amplified in lung cancer tumors, and subtype analyses have suggested that *FGFR1* gene amplification occurs in about 22% of squamous non-small cell lung cancers (NSCLCs) and 6% of small cell lung cancers

(SCLCs). Studies with lung cancer cell lines has demonstrated that amplification of the *FGFR1* gene is associated with overexpression of FGFR1 protein and increased signaling, which in turn lead to uncontrolled cell proliferation and survival. In breast cancer, amplification of the genomic locus of *FGFR1* has been reported in approximately 10% of predominantly estrogen receptor-positive patients. *FGFR1* amplification has also been reported in 17% of head and neck cancer patients.

Activating mutations in the *FGFR2* gene occur in approximately 12% of endometrial carcinomas. *FGFR2* gene amplification occurs in 3%-9% of gastric cancers. The co-expression of FGFR2b and its ligand, FGF7, in pancreatic cancer and gastric cancer is associated with poor prognosis, likely due to aberrant signaling through the formation of an autocrine activation loop. (Autocrine activation refers to the process whereby ligands interact with receptors on the same cell.) Genome-wide association studies have also identified *FGFR2* as a breast cancer susceptibility gene.

FGFR3 mutations have been reported in approximately 50% to 60% of non-muscle invasive bladder cancers and 17% of high-grade bladder cancers, and these mutations cause constitutive FGFR3 dimerization and activation. FGFR3 mutations are also present in cervical cancer (5%), prostate cancer (3%), and spermatocytic seminoma (7%).

Dysregulation of FGF ligand expression has also been observed in cancer. In ovarian cancer, frequent amplification of *FGF1* has been reported and is associated with poor survival. The levels of FGF1 expression correlated with microvessel density, suggesting that aberrantly expressed FGF1 promotes angiogenesis. In prostate cancer, several FGFs, including FGF2 and FGF6, are upregulated. In hepatocellular carcinomas (HCC), the upregulation of FGF2, 8, 17 and 18 promotes tumor growth, survival, and angiogenesis.

Given the strong evidence of the involvement of FGF-FGFR signaling in cancer, attenuation of this pathway represents a sound strategy for cancer therapeutics development.

Exhibit 10: Expression Profile of FGF and FGFR in Various Cancer Types

Cancer Type	Receptor Over-expression	Ligand Over-Expression	
		Targeted by FP-1039	Not Targeted By FP-1039
NSCLC	FGFR1; FGFR4	FGF-2	FGF-19
Glioblastoma	FGFR1; FGFR4	FGF-1, FGF-2, FGF-5, FGF-9	
Prostate	FGFR1c; FGFR4	FGF-1, -2, -6, -8, -9, -17	FGF-10
Pancreatic	FGFR1; FGFR2; FGFR4	FGF-1, FGF-2, FGF-5	FGF-10
Melanoma	FGFR1; FGFR2	FGF-2	
Breast	FGFR1; FGFR2	FGF-1, FGF-2, FGF-8	
Colorectal	FGFR1; FGFR4	FGF-2, FGF-18	FGF-19
Ovarian	FGFR2	FGF-1, FGF-2, FGF-8, FGF-9	FGF-7, FGF-23
Liver	FGFR1; FGFR3; FGFR4	FGF-1, FGF-3, FGF-4	FGF-19
AML, CML, B-CLL, ALL	FGFR1	FGF-2	
Bladder	FGFR1; FGFR3	FGF-2	
Endometrial	FGFR2	FGF-1, FGF-2, FGF-9	
Gastric	FGFR2	FGF-2	

Source: Five Prime Therapeutics

Challenges in Developing FGF Pathway-Targeted Therapies

Despite significant effort in research and development, a safe and efficacious FGF pathway-targeting therapy for cancer treatment has been elusive. The main challenge in developing such a therapy stems from the large number of ligands and receptors, with complex and overlapping binding specificities, and the diverse biological processes regulated by different pathway members.

Broad blockade of FGFR signaling using small molecule kinase inhibitors has been associated with perturbations of phosphate metabolism. In a study published in 2005, treatment of rats with PD176067, a compound that inhibits FGFR1 tyrosine kinase activity, resulted in a 47% to 166% increase in serum phosphate levels (hyperphosphatemia) and calcium-phosphorus deposition in various organs. Hyperphosphatemia has also been reported for two FGFR kinase inhibitors currently in phase 1 clinical trials: AstraZeneca's AZD4547 and Novartis' BGJ398. In the AZD4547 phase 1 trial, elevated phosphate blood levels were observed in 35% of patients, including six patients who required chelation therapy. In the BGJ398 phase 1 trial, it was reported that hyperphosphatemia could be managed with chelation therapy and diuretics.

The effect of FGFR kinase inhibitors on phosphate metabolism are most likely on-target toxicities mediated by FGF23, which is a key regulator of phosphate homeostasis. FGF23 is an endocrine (or hormonal) FGF. It is most highly expressed in bone, and circulates through the blood to regulate vitamin D and phosphate metabolism in kidney. FGF23 signaling downregulates the expression of sodium-phosphate cotransporters in the proximal tubule and

decreases the reabsorption of phosphate in the kidney. FGF23 also downregulates the absorption of phosphate from the gut and the uptake of phosphate from bone; these effects were achieved indirectly through FGF23-dependent modulation of vitamin D activation and parathyroid hormone (PTH) secretion. Mutation of the FGF23 gene is associated with a disease condition known as hypophosphatemic rickets.

Monoclonal clonal antibodies (mAbs) are also being developed to target the FGF pathway. An mAb against FGFR1c caused severe weight loss in rats and monkeys, an effect correlated with accumulation of the antibody in the hypothalamus. (Of note, although the blood-brain barrier generally prevents large molecules such as antibodies from entering interstitial spaces in the brain, areas of hypothalamus are among the few CNS sites where this barrier is absent.) These findings suggested that FGF signaling through FGFR1c may play a role in the "feeding circuit" in the hypothalamus that regulates satiety, again highlighting the wide range of tissues and biological processes in which FGF signaling plays a role and the difficulty of targeting this pathway.

Rationale and Design of FP-1039 as a Novel Cancer Therapy

Five Prime's FP-1039 is constructed by coupling the extracellular domain of the FGFR1c isoform with the Fc domain of the immunoglobulin G1 (IgG1). The Fc portion of the molecule serves to improve the stability and increase half-life of the molecule. The FGFR1c portion of the molecule serves as a "ligand trap," binding to various FGF ligands and preventing them from activating FGFRs on cell surface.

Five Prime has documented the binding affinity of FP-1039 to all paracrine and endocrine FGFs, as summarized in Exhibit 11. Among the paracrine FGFs, FP-1039 binds with very high affinity to FGF1, 2, 4, 6, 8b, 9, 16, 17, and 18 (Kd < 0.1 nM), with moderate affinity to FGF3, FGF5, and FGF20 (1nM < Kd < 7nM), and does not have measurable affinity for FGF7, 10, and 22. FP-1039 binds to FGF23, one of the endocrine FGFs, with low affinity (Kd = 32.8nM), and does not bind to the other two endocrine FGFs, FGF19, and FGF21.

Exhibit 11: FGF Binding Profile of FP-1039

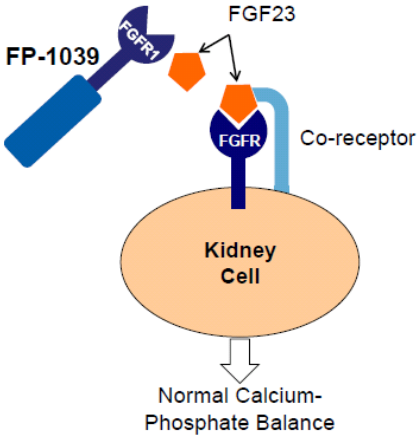
Ligand	Affinity to FP-1039 (M)
FGF1	3.10×10^{-11}
FGF2	4.62×10^{-10}
FGF3	2.92×10^{-9}
FGF4	6.75×10^{-10}
FGF5	2.95×10^{-9}
FGF6	3.40×10^{-10}
FGF7	0
FGF8b	4.56×10^{-10}
FGF9	9.19×10^{-10}
FGF10	0
FGF12	0
FGF16	4.04×10^{-10}
FGF17	5.94×10^{-10}
FGF18	2.99×10^{-10}
FGF19	0
FGF20	1.51×10^{-9}
FGF21	0
FGF22	0
FGF23	6.70×10^{-8}

FP-1039 ligand binding affinity was determined in the presence of heparin sulfate.

Source: Adapted from Harding T et al., *Sci Transl Med* 5:178ra39

The lack of high affinity binding of FP-1039 to the endocrine FGFs is not surprising, because binding of endocrine FGFs to FGFRs is known to require a co-receptor, αKlotho or βKlotho. The low binding affinity of FP-1039 to FGF23 may be associated with better tolerability compared with small molecule FGFR tyrosine kinase inhibitors, because FP-1039 is less likely to disturb FGF23-mediated phosphate homeostasis.

Exhibit 12: FP-1039 Unlikely to Disturb Phosphate Metabolism

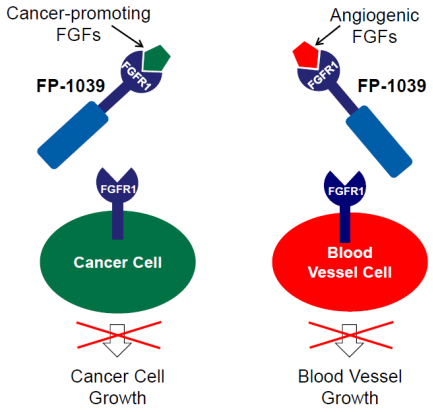


Source: Five Prime Therapeutics

Five Prime intends to use FP-1039 to treat tumors that contain an excessive number of *FGFR1* genes. As discussed earlier, *FGFR1* gene amplification has been reported in a wide variety of cancer types, including in 22% of patients with squamous non-small cell lung cancer (NSCLC), 6% of patients with small cell lung cancer (SCLC), 17% of patients with head and neck cancer, and 7%-15% of patients with breast cancer. FGFR1 amplification is associated with worse diagnosis in SCLC and breast cancer. Treatment with FP-1039, which is a fusion of FGFR1c and Fc, may compete with endogenous FGFR1 for FGF ligand binding and therefore attenuate signaling through these receptors.

In addition to promoting cell proliferation and survival, FGF signaling can also promote growth of blood vessel tissue. This process, known as angiogenesis, can accelerate the growth of tumors by increasing the supply of oxygen and nutrients to the tumor cells. The FGFs that promote angiogenesis are often present in renal cell carcinoma (a type of kidney cancer) and in hepatocellular carcinoma (a type of liver cancer). Targeting FGFR1 signaling in these cancers therefore could block tumor angiogenesis and inhibit tumor growth.

Exhibit 13: Proposed Dual Mechanism of Action of FP-1039



Source: Five Prime Therapeutics

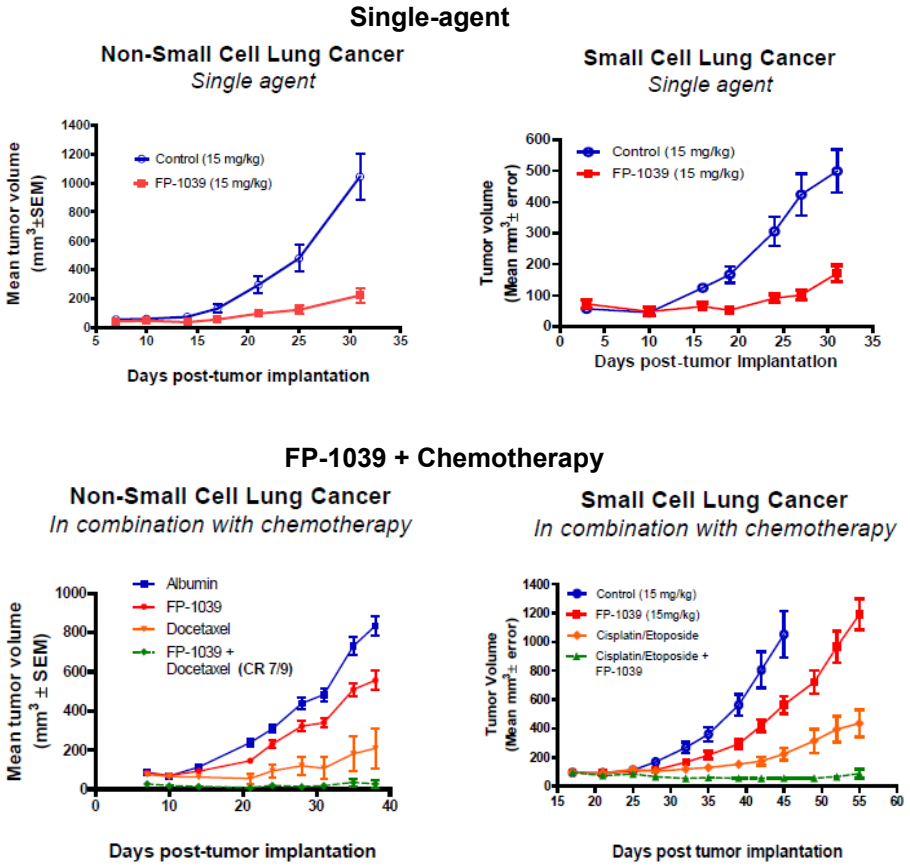
Pre-clinical Data for FP-1039

In preclinical studies, FP-1039 inhibited FGF-stimulated cell proliferation in vitro, blocked FGF-induced angiogenesis in vivo, and inhibited in vivo growth of a broad range of tumor types, particularly those with FGFR1 gene amplification.

Five Prime identified lung cancer cell lines (including both squamous NSCLC and SCLC) that had FGFR1 gene amplification. FP-1039 demonstrated single-agent anti-tumor activity and inhibited the growth of these cell lines in cell culture (in vitro) and in subcutaneous xenograft models in vivo (Exhibit 14, top row).

Furthermore, when combined with standard chemotherapy, such as docetaxel for squamous NSCLC and cisplatin/etoposide for SCLC, FP-1039 treatment demonstrated greater tumor growth inhibition than either FP-1039 or chemotherapy alone (Exhibit 14, bottom row).

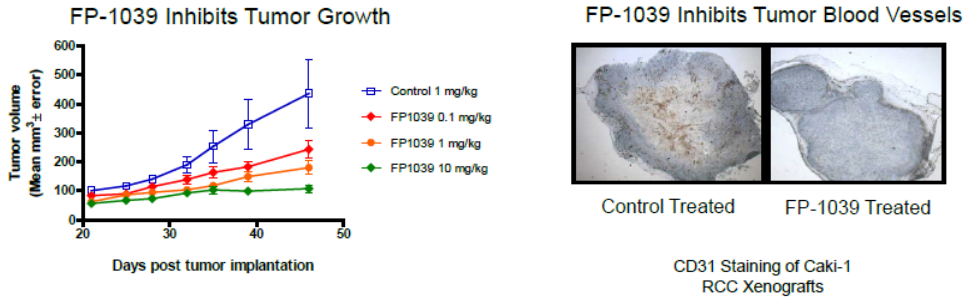
Exhibit 14: Anti-tumor Activity of FP-1039 in FGFR1-amplified Squamous NSCLC and SCLC Models



Source: Five Prime Therapeutics

The FGF pathway has been implicated in the progression of renal cell carcinoma (RCC). In some preclinical models of RCC, FGFs are found at high levels and promote tumor growth and angiogenesis. Treatment of these RCC tumors with FP-1039 as a single agent resulted in inhibition of tumor growth (Exhibit 15, left panel). Antiangiogenic activity was observed directly in the RCC tumor xenografts by staining blood vessels with anti-CD31 (a protein expressed on endothelial cells and platelets). Reduced vasculature staining was observed following treatment with FP-1039 (Exhibit 15, right panel).

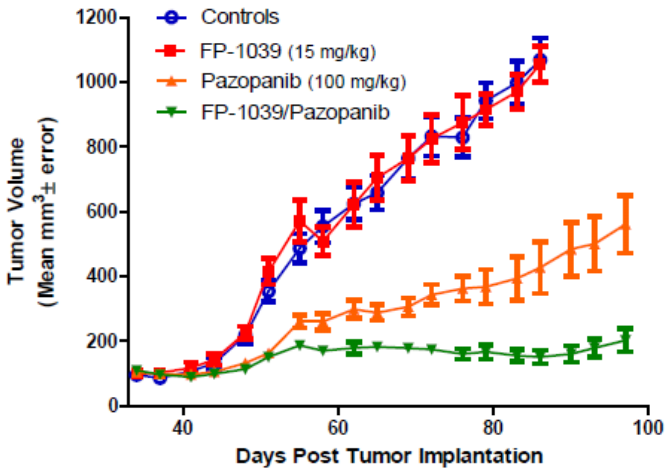
Exhibit 15: Anti-tumor Activity of FP-1039 in a Mouse Model of RCC



Source: Five Prime Therapeutics

In most cases of human RCC, angiogenesis is promoted by an abnormally high level of vascular endothelial growth factor (VEGF). VOTRIENT (pazopanib) is a small molecule VEGF inhibitor approved for the treatment of RCC. Although tumor growth may be controlled by anti-VEGF therapy initially, RCC tumors eventually progress because other factors, including FGFs, replace VEGF in stimulating angiogenesis. Therefore a combination therapy of VOTRIENT + FP-1039 may be effective in treating RCC. In preclinical models of RCC with abnormally high VEGF levels, adding FP-1039 to VOTRIENT resulted in greater inhibition of tumor growth than VOTRIENT alone (Exhibit 16).

Exhibit 16: Anti-tumor Activity of FP-1039 + VOTRIENT in a Mouse Model of RCC with high VEGF Levels

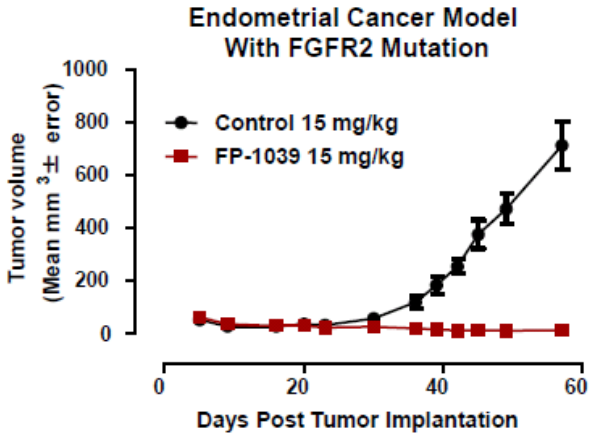


Source: Five Prime Therapeutics

Because of the overlapping ligand binding specificities of different FGFRs, FP-1039 can potentially target additional FGFRs besides FGFR1. To test this hypothesis, Five Prime studied a human endometrial carcinoma cell line bearing a common FGFR2 mutation (the S252W mutation, which occurs in 7% of all endometrial carcinoma cases). FP-1039 treatment

demonstrated a 95% inhibition of tumor growth in mice bearing xenograft of the FGFR2 S252W mutated endometrial carcinoma cell line (Exhibit 17).

Exhibit 17: Anti-tumor Activity of FP-1039 in a Endometrial Cancer Model With FGFR2 Mutation



Source: Five Prime Therapeutics

Review of Phase 1 Data for FP-1039

From July 2008 to June 2011, Five Prime conducted a phase 1 clinical trial of FP-1039 in patients with metastatic or locally advanced unresectable solid tumors for which standard curative or supportive measures did not exist or were no longer effective. In this phase 1 trial, FP-1039 treatment was associated with stabilization of tumor growth (stable disease) in 43.6% of the patients, including one patient with hormone-resistant prostate cancer who achieved tumor reduction of 20% and a stable disease duration of approximately seven months. FP-1039 was described as well tolerated and was not associated with hyperphosphatemia as was observed in clinical trials of small molecule pan-FGFR inhibitors.

The open-label, non-randomized, ascending-dose phase 1 study enrolled 39 patients with a variety of tumors, including advanced or metastatic breast cancer, lung cancer, colon/rectal cancer, prostate cancer, head and neck cancers, or uterine cancer. Patients received FP-1039 as a 30-minute, once weekly intravenous infusion for four weeks, followed by a two-week observation period. Patients without progressive disease were provided the option to continue on FP-1039 on a weekly basis.

Exhibit 18: Patients Demographics for the FP-1039 Phase 1 Trial

Characteristic	Number of Patients (n=39)	
Age	Median:	64
Gender	Female:	24
	Male:	15
Screening ECOG Performance Status	ECOG 0	9
	ECOG 1	27
	ECOG 2	3
Number of Prior Regimens	Median:	4
	Mean:	4.8
	Range:	0 - 11
Race	Caucasian	30
	African American	3
	Hispanic/Latino	6

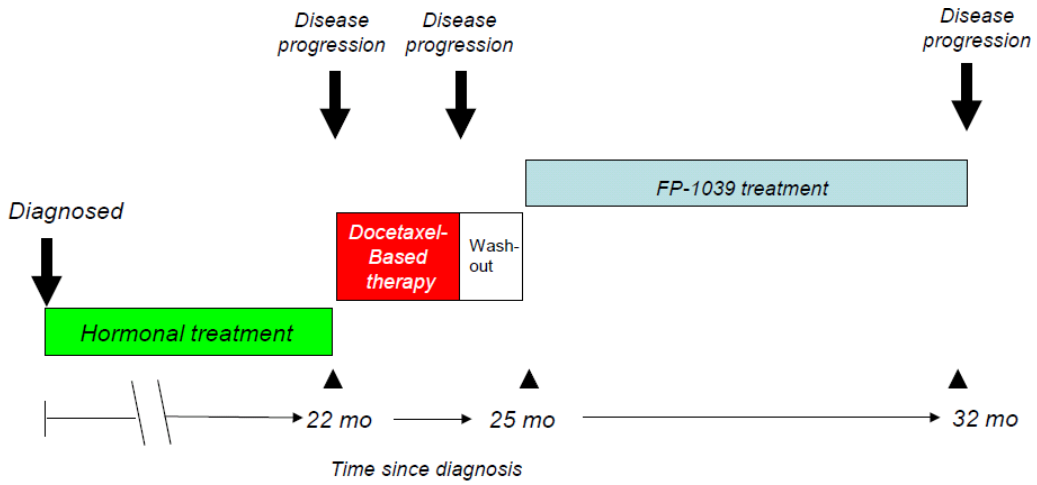
Tumor Type	Number of Subjects (n=39)
[a]Sarcoma	9
Breast Cancer	7
[b]Colorectal Cancer	6
Prostate Cancer	5
[c]Head and Neck	4
Lung Cancer	3
[d] Hepatobiliary	2
Endometrial Cancer	1
Carcinoid	1
Pancreatic Cancer	1

[a] Combines: liposarcoma, sarcoma of leg, leiomyosarcoma, angiosarcoma, chondrosarcoma, mixed Mullerian Duct sarcoma, rhabdomyosarcoma, and osteosarcoma of jaw
[b] Combines: adenocarcinoma of colon and rectal carcinoma and adenocarcinoma of small bowel
[c] Combines:cancer of salivary gland, laryngeal cancer, adenoid cystic carcinoma, and squamous cell carcinoma
[d] Combines: hepatocellular carcinoma and cholangiosarcoma

Source: Five Prime Therapeutics

Five Prime did not require patients to have tumors with FGFR1 gene amplification because the primary objectives of the study were to assess safety and pharmacokinetics of FP-1039. In this unselected patient population, 17 patients had stable disease (stabilization of tumor growth), and no major tumor shrinkage was observed. One patient with hormone-resistant prostate cancer who had progressed rapidly on chemotherapy achieved tumor reduction of 20% following treatment with FP-1039, with a stable disease duration of approximately seven months.

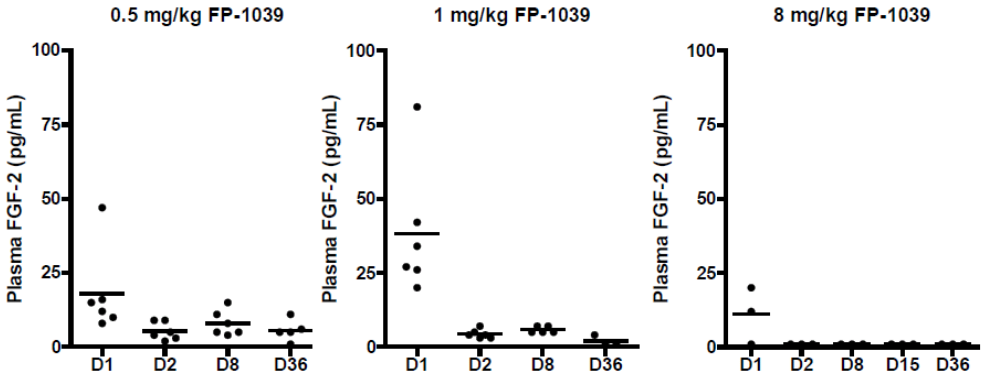
Exhibit 19: Treatment History of a Patient with Prostate Cancer Who Achieved Stable Disease on FP-1074



Source: Five Prime Therapeutics

Five Prime reported a significant decrease in the blood levels of FGF2, which is an important cancer-promoting FGF, in all patients tested (Exhibit 20). Plasma levels of FGF2 remained low throughout the dosing schedule (weekly dosing) and at 2 weeks after the last dose.

Exhibit 20: FP-1039 Lowered FGF2 Plasma Levels



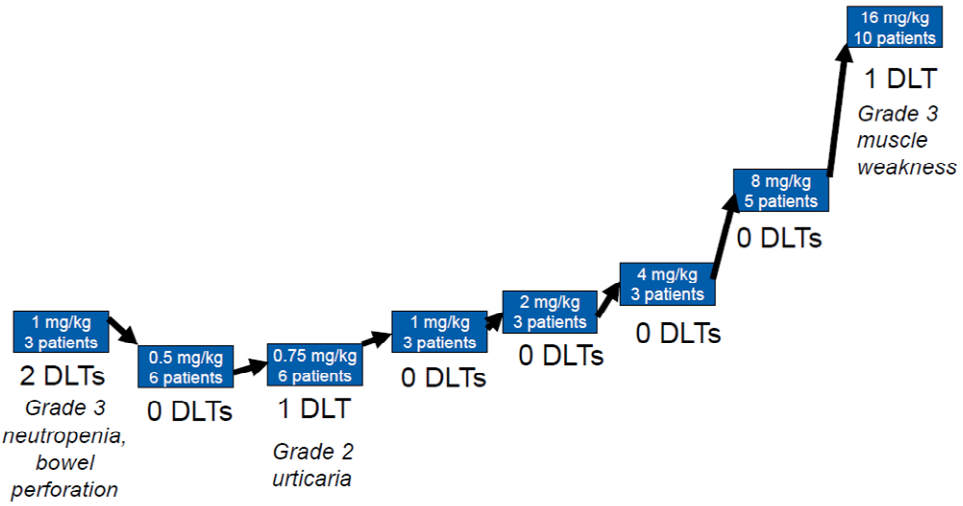
D1 = Day 1 (Pre-dose); D2 = 24 hr post 1st dose; D8 = 7 Days post 1st dose; D36= 2 weeks post last (4th) dose.

Source: Five Prime Therapeutics

In the phase 1 study, FP-1039 was described as well tolerated over the dose range studied (0.5mg/kg to 16mg/kg), and no maximum tolerated dose (MTD) was observed. Early in the trial, dose limiting toxicities (DLTs) were observed at 1mg/kg and 0.75mg/kg, but no DLTs were observed at subsequent doses of 2mg/kg, 4mg/kg, and 8 mg/kg (Exhibit 21). FP-1039 treatment was not associated with hyperphosphatemia, as observed in clinical trials of small molecule pan-FGFR inhibitors. Five Prime expects FP-1039 to be well tolerated in combination with standard of care chemotherapy.

Exhibit 21: Safety and Tolerability Results From the Phase 1 Trial

Dose Escalation and Dose Limiting Toxicities (DLTs)



Adverse Events

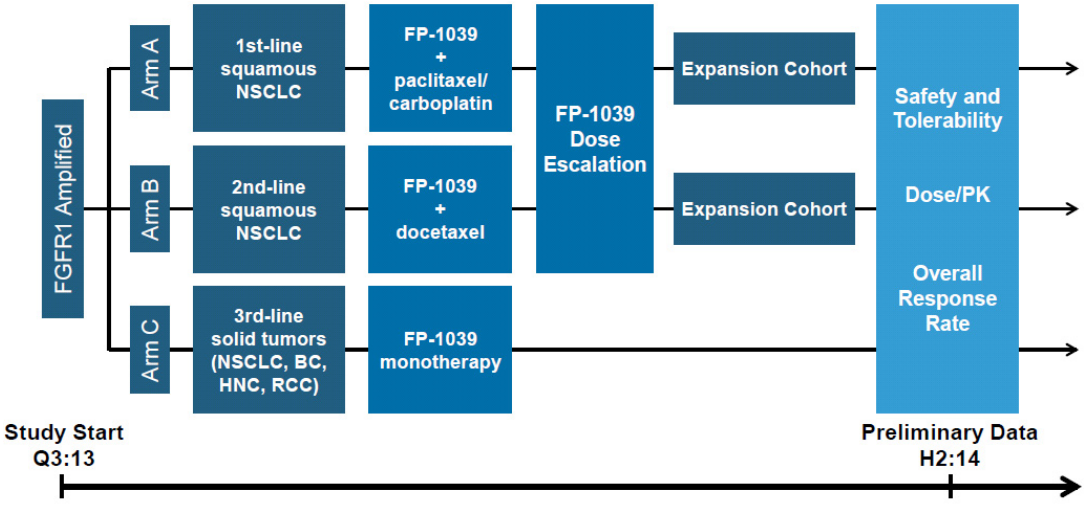
		Dosing Cohort (mg/kg)																					
		0.5 (n=6)			0.75 (n=6)			1 (n=6)			2 (n=3)			4 (n=3)			8 (n=5)			16 (n=10)		
System/Terms	CTC Grade	2	3	4	2	3	4	2	3	4	2	3	4	2	3	4	2	3	4	2	3	4
Blood																						
	Anemia							1														
	Leukopenia																			1		
	Neutropenia								1													
Gastrointestinal																						
	Diarrhea							1														
	Intestinal Perforation								1													
	Nausea							1														
	Vomiting																			1		
General																						
	Fatigue	2																		2		
	Infusion Related Reaction																			1		
	Peripheral Edema							1														
Metabolic																						
	Hypocalcemia	1																				
	Hyponatremia																				1	
Musculoskeletal/Nervous System																						
	Weakness																				1	
Skin																						
	Urticaria				1																	

Source: Five Prime Therapeutics

Review of Phase 1b Design and Timelines for Data

In July 2013, GlaxoSmithKline (GSK), Five Prime's partner for FP-1039 development, initiated an open-label, uncontrolled phase 1b clinical trial to evaluate FP-1039 in combination with chemotherapies and as a monotherapy in patients with FGFR1 gene-amplified tumors. This phase 1b trial includes three arms: (1) FP-1039 + paclitaxel + carboplatin in patients with previously untreated metastatic squamous NSCLC (Stage IV disease); (2) FP-1039 + docetaxel in patients with documented tumor progression or intolerability after receiving only one prior line of platinum-containing combination chemotherapy for Stage IV disease; or (3) FP-1039 monotherapy in patients with metastatic cancers, such as squamous NSCLC, RCC, breast or head and neck, who have exhausted all lines of standard therapies or for whom there is no standard treatment.

Exhibit 22: Study Design of the FP-1039 Phase 1b Trial



Source: Five Prime Therapeutics

Approximately 70 patients will be enrolled in the study, with possible expansion to approximately 104 patients. All patients are required to have documented FGFR1 gene amplification or FGF over-expression. A third-party central lab tests tumor samples from prospective subjects to identify those with FGFR1 gene-amplified tumors. Five Prime and GSK have not yet engaged any third party to develop a companion diagnostic for future clinical trials of FP-1039 or for the registration and approval of FP-1039.

Primary outcome measures of the phase 1b study are safety and tolerability evaluated up to 6 months, including rate and severity of adverse events (AEs), discontinuation due to AEs, dose-limiting toxicity (DLT), electrocardiogram and echocardiogram measures, and efficacy outcomes evaluated up to six months, including best response and overall response rate (ORR) according to RECIST 1.1.

Preliminary data from the phase 1b trial are expected in 2H14. In addition to the phase 1b program, Five Prime is also exploring the feasibility of conducting a study of FP-1039 in combination with a VEGF inhibitor in certain tumors, possibly RCC or HCC.

Five Prime had previously initiated a phase 2 clinical trial of FP-1039 in endometrial cancers in 2011, which was later terminated because no qualified patients were identified after screening the first 70 patients. The study intended to enroll patients with advanced and/or recurrent endometrial cancers bearing the S252W or the P243R mutation in the FGFR2 gene, and it had been assumed that at least 5% of patients screened would qualify.

Review of Competitive Landscape for FGF Targeted Drugs

Broadly speaking, FGF pathway-targeted therapies can be divided into five classes: mixed kinase inhibitors with weak FGFR activity; FGFR selective tyrosine kinase inhibitors; FGF antibodies; FGFR antibodies; and FGF ligand traps. Each class has its inherent advantages and disadvantages. After taking stock of the overall safety and efficacy profiles of each class, we believe FGF ligand traps, of which FP-1039 is the sole member, occupy a favorable competitive position relative to other classes of FGF targeted agents. This is because the design of FP-1039 seems to have struck the right balance in fulfilling two opposing goals: tackling the redundancy of the pathway caused by over-lapping binding specificities between 22 ligands and 4 receptors, and at the same time steering clear of certain ligand-receptor interactions that are involved in important biological processes.

Among the five classes of FGF-targeted therapies, mixed kinase inhibitors are the most clinically advanced. These inhibitors typically have higher potency against VEGF and/or PDGF receptors versus FGFRs. Examples of this class include dovitinib (Novartis), lenvatinib (Eisai), brivanib (Bristol-Myers Squibb), ICLUSIG (Ariad Pharmaceuticals), lucitanib (Servier), and nintedanib (Boehringer Ingelheim).

Novartis's dovitinib is an FGF, VEGF, and PDGF receptor kinase inhibitor. In a recently completed phase 3 trial in 564 patients with metastatic renal cell carcinoma (RCC) after failure of anti-angiogenic therapies (with VEGF- and mTOR- inhibitors), dovitinib failed to meet the primary endpoint of progress-free survival in a head-to-head comparison with Onyx Pharmaceuticals' NEXAVAR. Dovitinib is currently under phase 1/2 development for three solid cancer indications: breast cancer, endometrial cancer, and hepatocellular carcinoma (HCC).

BMS's brivanib is a FGFR and VEGFR dual kinase inhibitor. M.D. Anderson Cancer Center and BMS are currently conducting a phase 2 trial of brivanib + irinotecan in metastatic colorectal cancer enriched for elevated levels of plasma FGF. The primary completion date for the trial is September 2015 according to clinicaltrial.gov. Brivanib has failed two prior phase 3 trials in HCC, both as a first-line treatment in a head-to-head comparison with NEXAVAR and as a second-line treatment in patients who failed or cannot tolerate NEXAVAR.

Ariad Pharmaceuticals' ICLUSIG (ponatinib) is a multi-targeted tyrosine kinase inhibitor that primarily targets BCR-ABL, an abnormal tyrosine kinase found in chronic myeloid leukemia

(CML) and Philadelphia-chromosome positive acute lymphoblastic leukemia (Ph+ ALL). ICLUSIG can also inhibit a broad panel of naturally occurring mutant variants of FGFRs, in particular variants of FGFR2 that have been observed in endometrial cancer and squamous cell carcinomas of the lung. A phase 2 investigator-sponsored trial is currently evaluating ponatinib in patients with squamous cell lung and head and neck cancers, with a primary completion date of December 2014.

The newer generation small molecule FGFR inhibitors have higher selectivity for FGFR versus VEGFR and other tyrosine kinases. Examples in this class include AZD4547 (AstraZeneca), BGJ398 (Novartis), and LY2874455 (Eli Lilly). In preclinical studies, all three agents demonstrated antitumor activity in xenograft models with FGFR dysregulation, such as FGFR2 gene amplification. Furthermore, efficacious doses of AZD4547 and LY2874455 did not induce elevations in blood pressure in tumor xenograft models.

AstraZeneca is conducting a three-part phase 1 study of AZD4547 in patients with advanced tumors. Part A and Part B of the trial represented the dose-escalation and dose-expansion phases, enrolling patients unselected for FGFR amplification. Part C1 assessed the safety and clinical activity of AZD4547. In Part C1, 21 patients with diverse tumor types and with FGFR1- or FGFR2- gene amplification received AZD4547 at a dose of 80mg BID. Data presented at the 2013 American Association for Cancer Research (AACR) annual meeting demonstrated single agent activity of AZD4547. One squamous NSCLC patient in Part C who had high copy number FGFR1 amplification and who had received two previous lines of anti-cancer therapy, achieved a partial response lasting 12 weeks (patient discontinued at 24 weeks due to disease progression). An additional four patients in Part C, including one with breast cancer, one with squamous NSCLC, and two with transitional cell carcinoma, experienced disease stabilization of greater than 24 weeks. Besides the phase 1 trial, AZD4547 is also being evaluated in several phase 1/2 and phase 2 studies for the treatment of solid tumors, either as monotherapy or in combination with chemotherapy. The solid tumor indications being studied include advanced gastric or gastro-oesophageal junction cancer, ER+ breast cancer, recurrent NSCLC, and FGFR1 or FGFR2 amplified gastric, oesophageal, breast, and squamous cell lung cancers. Primary completion dates of these trials range from 3Q14 to 2Q16.

BGJ398 is currently in phase 1 development in patients with advanced solid tumors with FGFR1 and FGFR2 amplification or FGFR3 mutation, with a primary completion date of August 2013 as listed on clinicaltrials.gov. Preliminary data from this trial were presented at the 2012 AACR annual meeting. A total of 21 patients, including 10 patients with FGFR1-amplified breast cancer and 3 patients with FGFR1-amplified squamous cell lung cancer were treated with BGJ398 in a 28-day cycle in escalating dose cohorts. The dose was escalated from 5mg to 150mg over 7 dose cohorts. One lung cancer patients with FGFR1-amplification responded to 100mg BGJ398 and achieved a 33% reduction in target lesion by CT scan at 8 weeks, with confirmation at 12 weeks and a substantial standardized uptake value (SUV) decrease on PET.

LY2874455 is currently in a phase 1 trial in patients with advanced solid tumors, lymphoma, or chronic lymphocytic leukemia, with a primary completion date of November 2014.

As mentioned earlier, a well documented issue with small molecule FGFR inhibitors is their effect on phosphate metabolism, an on-target toxicity mediated by inhibition of signaling through FGF23, a key regulator of phosphate homeostasis. In phase 1 experience, treatment with AZD4547 was associated with elevated phosphorous blood levels in 35% of patients, including 6 patients who required chelation therapy. In addition, 20% of patients discontinued study due to Grade 1/2 oral toxicity. In the BGJ398 phase 1 trial, hyperphosphatemia was also reported.

Exhibit 23: Comparison of FP-1039 With Other FGF-targeted Therapeutics

Approach	Stage	Known Issues	FP-1039 Advantages
Antibody to FGF2 (Roche)	Preclinical	Only targets 1 FGF	Targets multiple cancer-promoting ligands
Antibody to FGFR1 (Genentech, Imclone)	No development reported	Severe weight loss	No weight loss
Selective FGFR TKIs (Novartis, AZ, J&J, Lilly)	Phase 1/2	Hyperphosphatemia Retinal toxicities	Similar toxicities not seen
Multikinase TKIs (Novartis, Servier/EOS, etc)	Various	VEGFR-related toxicities limit FGFR pathway inhibition	No VEGFR-related toxicities seen

Source: Five Prime Therapeutics

Monoclonal clonal antibodies (mAbs) against both FGF ligands and FGFRs have been under development for several years. At the 2011 AACR-NCI-EORTC International Conference, Genentech (now part of Roche) presented pre-clinical data demonstrating efficacy of an mAb against FGFR3, named R3Mab, in bladder tumor xenograft models. FGFR3 overexpression occurs frequently in bladder cancers and activating mutations of FGFR3 have been identified in approximately 60%-70% of papillary and 16%-20% of muscle-invasive bladder carcinomas. Binding of R3Mab to FGFR3 blocked ligand binding, prevented receptor dimerization, and induced substantial conformational changes in the receptor. As a result, R3Mab was capable of inhibiting both WT and various cancer-associated mutant forms of FGFR3. R3Mab suppressed the growth of multiple bladder tumor xenografts in mice as a single agent, and enhanced anti-tumor activity of chemotherapeutics, including gemcitabine and carboplatin. In addition, R3Mab also inhibited the growth of t(4;14)-positive multiple myeloma xenografts via antibody-dependent cell-mediated cytotoxicity (ADCC).

Genentech's anti-FGFR3 mAb, MFGR1877S (RG7444), was the first anti-FGFR antibody to enter human clinical trials. Two phase 1 trials have been completed, one in patients with relapsed or refractory t(4;14)-positive multiple myeloma, and the other in advanced solid tumors. The data from the multiple myeloma trial were presented at the 2012 American Society

of Hematology (ASH) annual meeting. Fourteen patients with a median number of 5 prior therapies received anti-FGFR3 mAb weekly for 3 weeks, followed by q28-day dosing. The median number of doses received was 3.5 doses (range 1-7 doses). No objective response was observed. Six patients had stable disease, with 2 patients up to 4 cycles, 1 patient up to 3 cycles, and 3 patients up to 1 cycle. No DLT was observed through the highest dose tested (15mg/kg). The anti-FGFR3 therapy was described as well-tolerated, with fatigue (7%) being the only Grade ≥3 related AE observed. It is unclear whether MFGR1877S is still under active development. Clinicaltrials.gov does not list any ongoing trials of MFGR1877S.

ImClone Systems (now part of Eli Lilly) developed a fully human mAb against FGFR1c, named IMC-A1. The antibody was developed as a cancer treatment but was found to cause profound weight loss in rats and monkeys in pre-clinical testing. The weight loss was primarily a result of reduced energy intake (hypophagia) and not a result of illness or increased energy expenditure. Systemically administered antibody was found to accumulate in the hypothalamus in a region with enriched FGFR1 expression. Furthermore, a single intracerebroventricular administration of IMC-A1 in mice caused an approximately 36% reduction in food intake within the ensuring 24 hours. These data suggested that FGF signaling through FGFR1c may play a role in the hypothalamic feeding circuit and blocking it could lead to hypophagia and weight loss.

AVEO Pharmaceuticals developed an mAb against FGFR2b, named GP369. According to preclinical data published in 2010, the administration of GP369 in mice significantly inhibited the growth of xenografts of human gastric cancer cell line harboring GFGR2 amplification. The current development status of GP369 at AVEO is unclear.

Development of antibodies against FGF ligands has been less active compared with development of antibodies against FGFRs, presumably because of the large number of ligands and the high degree of redundancy. Genentech published pre-clinical studies of an mAb against FGF19, an endocrine FGF that acts on distant tissues. This antibody selectively blocked the interaction between FGF19 and FGFR4. In preclinical studies, Genentech's anti-FGF19 mAb inhibited growth of colon tumor xenografts and prevented hepatocellular carcinomas in FGF19 transgenic mice.

Opportunity for FP-1039 in FGFR1 Amplified Tumors (NSCLC, SCLC, SCCHN, BC, RCC and HCC)

Five Prime and partner GSK are developing FP-1039 for the treatment of several solid tumor types that are associated with frequent FGFR1 gene amplification, including squamous non-small cell lung cancer, or NSCLC, with an FGFR1-amplification frequency of 22%, small cell lung cancer, or SCLC (6% amplification), head and neck cancer (17% amplification), and breast cancer (7-15% amplification). In addition to FGFR1-amplified tumors, tumors with high angiogenesis, such as renal cell carcinoma (RCC) and hepatocellular carcinoma (HCC), are also suitable indications for FP-1039 development.

Exhibit 24: Patient Number for FP-1039's Potential Indications

Advanced or metastatic tumors with *FGFR1* gene amplification			
	Amplification Frequency	Prevalence of *FGFR1* Amplification	
Tumor Type		US	Europe & Asia
Squamous NSCLC	22%	11,000	51,000
Head and Neck Cancer	17%	17,000	132,000
Breast Cancer	7-15%	32,000	148,000
SCLC	6%	2,000	10,000
Total		62,000	341,000

Advanced or metastatic tumors with high angiogenesis		
	Tumor Prevalence	
Tumor Type	US	Europe & Asia
Kidney (RCC)	61,000	172,000
Liver (HCC)	9,000	250,000
Total	70,000	422,000

Based on 2012 estimated prevalence

Source: Five Prime Therapeutics

NSCLC and SCLC

The National Cancer Institute (NCI) estimates that approximately 228,000 new cases of lung cancer will be diagnosed in the US in 2013, accounting for approximately 14% of all cancer diagnosis. The average age at the time of diagnosis is 70 years. The prevalence of lung cancer in the US was approximately 399,000 in 2010.

Lung cancer is classified as small cell lung cancer (SCLC), which accounts for 15% of lung cancer cases, and non-small cell lung cancer (NSCLC), which accounts for 84%. The distinction between SCLC and NSCLC was based on differential clinical features and sensitivities to cytotoxic therapies. SCLC was characterized by widespread metastatic spread at diagnosis and demonstrated partial or complete response to conventional cytotoxic therapies, whereas NSCLC was less likely to spread at diagnosis and typically failed to demonstrate objective responses to cytotoxic agents.

NSCLC is further divided into three histological subtypes: squamous cell carcinoma, adenocarcinoma, and large cell carcinoma. Squamous cell cancer accounts for approximately 30% of NSCLC. Squamous cell cancer often develops from epithelial cells near the center of the lung in one of the main airways (the left or right bronchus). Often a result of smoking, squamous cell cancer used to be the most common form of lung cancer, but its incidence has decreased significantly in the past four decades. Adenocarcinoma is a cancer of epithelial cells that make fluids (mucus) to keep the lung moist and is often found in the peripheral compartments of the lung, near respiratory bronchioles, and the alveoli. Adenocarcinoma is the most common form of lung cancer in North America. Large-cell lung carcinomas lack features to be classified as any other carcinoma, and this type of lung cancer tends to grow quickly.

Lung cancer is the leading cause of cancer death; NCI estimates that 160,000 lung cancer deaths will occur in 2013, more than colon, breast, and prostate cancer deaths combined and accounting for approximately 27% of all cancer deaths. The one-year relative survival for lung cancer is 44%, and the five-year survival for lung cancer is 16%. The five-year survival for SCLC (6%) is significantly lower than that for NSCLC (18%). Exhibit 25 provides detailed five-year survival rates for NSCLC and SCLC by stage.

Exhibit 25: Five-Year Survival Rate of NSCLC and SCLC by Stage

Stage	5-Year Survival Rate
NSCLC	
IA	49%
IB	45%
IIA	30%
IIB	31%
IIIA	14%
IIIB	5%
IV	1%
SCLC	
I	31%
II	19%
III	8%
IV	2%

Source: National Cancer Institute's Surveillance, Epidemiology, and End Results (SEER) database.

A Stage IA lung tumor is 3cm or smaller, and a Stage IB tumor is 5cm or smaller or may have grown into the bronchus or visceral pleura. A Stage IIA tumor is 3cm or smaller with cancer in the lung's lymph nodes, or between 5cm and 7cm with no cancer in lymph nodes, whereas a Stage IIB tumor can be 7cm or smaller with cancer in the nodes, or those larger than 7cm without cancer in nodes, or those that have invaded chest wall or bronchus, or those with secondary tumors in the same lobe. Stage III tumor may be of any size; Stage IIIA is often defined by cancer that has spread to mediastinal nodes, presence of secondary tumors, or tumor growth into the mediastinum, neck or spine; Stage IIIB is often defined by cancer that has spread to lymph nodes in or near the other lung or above the collarbone. In Stage IV lung cancer, malignant tumors are found in both lungs, or the cancer has spread to other parts of the body, such as the brain, bones, liver, or in the fluid between the two layers of pleura.

The frontline regimen recommended by the National Comprehensive Cancer Network (NCCN) for squamous NSCLC is a doublet of gemcitabine and cisplatin, although doublet combinations of a platinate with another class of agent, other than ALIMTA are options. NCCN also notes that the addition of ERBITUX to cisplatin/vinorelbine is reasonable for subjects with good ECOG performance status. Following 4-6 cycles, NCCN notes that maintenance therapy may be indicated with options including continuation of the current regimen, or if ERBITUX was used continuation of single agent ERBITUX; NCCN also notes that switching to TARCEVA or docetaxel is also an option for maintenance. Following failure of front line chemotherapy, options become more limited for additional therapy. Options noted by NCCN are restricted to

docetaxel or TARCEVA. In addition neither Alimta nor Avastin are indicated for use in the treatment of squamous histology NSCLC.

For extensive stage SCLC, NCCN recommends either the combination of cisplatin with etoposide or irinotecan as a frontline chemotherapy; irinotecan can also be paired with carboplatin. While response rates to frontline therapy are high, relapse is inevitable. Beyond a clinical trial, choice of second-line therapy depends on the duration of response to frontline therapy. If the duration of response is longer than six months, the frontline regimen can be repeated, while for a rapid relapse 2-3 months single agent chemotherapy is recommended, largely for palliation. The majority of patients will relapse in the two-three- to six-month range and for these patients, single agent HYCAMTIN is recommended by NCCN. NCCN identifies a number of alternative less preferred agents to HYCAMTIN including single agent taxanes, irinotecan, gemcitabine, vinorelbine, etoposide and the combination of cyclophosphamide, doxorubicin and vincristine. In patients with methylated methylguanine-DNA methyltransferase (MGMT), TEMODAR may have useful activity.

SCCHN

Head and neck cancer is a term used to describe a number of cancers that develop in or around the areas of mouth, throat, larynx (voice box), nose, and sinuses. Most head and neck cancers begin in the flat epithelial cells (squamous cells) that line the mucosal surfaces, and are referred to as squamous cell carcinoma of the head and neck (SCCHN). Head and neck cancers that start in the salivary glands are classified as adenocarcinoma.

Head and neck cancers account for approximately 3% of all cancers in the US, and are nearly twice as common among men as among women. It is estimated that 53,000 Americans develop head and neck cancers annually and 11,500 die from the disease. The five-year survival rate of patients with head and neck cancers varies depending on factors such as the specific cancer type. Exhibit 26 summarizes the five-year survival rate of oral cavity and pharynx cancers, which accounts for more than 70% of head and neck cancers.

Exhibit 26: Five-Year Survival Rate of Oral Cavity and Pharynx Cancers

Stage	5-Year Survival Rate
Localized (confined to primary site)	83%
Regional (spread to regional lymph nodes)	59%
Distant (metastatic)	36%
Unknown (unstaged)	49%

Source: National Cancer Institute's Surveillance, Epidemiology, and End Results (SEER) database.

Although SCCHN comprises tumors initiating in a wide variety of structures, chemotherapy options are universal with the exception of nasopharyngeal tumors. In the advanced stage, a platinate combined with 5FU with ERBITUX for non-nasopharyngeal tumors is the regimen of choice according to the NCCN guidelines. Again for non-nasopharyngeal tumors, cisplatin with ERBITUX is an option while for nasopharyngeal tumors carboplatin/ERBITUX or

gemcitabine/vinorelbine are options and for all advanced SCCHN tumors, doublets or a platinate with a taxane or cisplatin with 5FU are options. On a case-by-case basis, the use of surgery or radiation therapy is also an option in advanced incurable disease for palliation. Single agent chemotherapy is also listed as an option in the NCCN guidelines for good ECOG performance status patients with a broad range of options, and single agent chemotherapy is the only therapeutic option for ECOG PS2. For patients with a good ECOG PS progressing on a front line regimen, NCN recommends a clinical trial, single agent chemotherapy, or best supportive care.

Breast Cancer

Excluding cancers of the skin, breast cancer is the most frequently diagnosed cancer in women. The NCI estimates that approximately 232,300 women in the US will be diagnosed with breast cancer in 2013, and approximately 39,600 will die of the disease. Breast cancer is the second leading cause of cancer death in women (after lung cancer). Currently there are more than 2,800,000 breast cancer survivors in the US.

The stages of breast cancer are determined based on the size of the tumor and whether it has spread beyond the breast. Stage 0 and Stage I tumors are non-invasive. In Stage 0, atypical cells have not spread outside the ducts or lobules into the surrounding breast tissue. In Stage I, the tumor is no more than 2cm in diameter and has not spread to lymph nodes or outside the breast. In Stage II, the tumor is growing, but it is still contained in the breast or growth has only extended to nearby lymph nodes. In Stage III, also known as locally or regionally advanced breast cancer, the breast cancer has extended to beyond the immediate region of the tumor and may have invaded nearby lymph nodes and muscles, but has not spread to distant organs. In Stage IV, or metastatic breast cancer, the cancer has spread to other areas of the body, such as the brain, bones, lung, and liver.

Death rates from breast cancer have been declining since 1989, owing to earlier detection through screening and increased awareness. Five-year survival rates of various stages of breast cancer are summarized in Exhibit 27.

Exhibit 27: Five-Year Survival Rate of Breast Cancer by Stage

Stage	5-Year Survival Rate
0	100%
I	100%
II	93%
III	72%
IV	22%

Source: National Cancer Institute's Surveillance, Epidemiology, and End Results (SEER) database.

The greatest unmet need in the treatment of metastatic breast cancer is in patients with triple negative disease. Triple-negative breast cancer (TNBC) is an aggressive subtype of breast cancer defined by an absence of estrogen and progesterone receptors, and HER2. Consequently, neither endocrine-based nor HER2-based therapies, which are the cornerstones for targeted

therapy-based treatment for non-TNBC are used. There is no established standard of care regimen for advanced metastatic disease, and choice is driven by the duration of benefit from anthracycline/docetaxel adjuvant/neo-adjuvant therapy. In patients with a greater than six-month benefit from adjuvant therapy re-use of the same chemotherapy doublet is appropriate with data from one large phase 3 trial suggesting a 10-month DFS benefit for sequential use of these agents, versus 7 months for docetaxel monotherapy. In subjects with a more rapid pace of disease progression, treatment with non-cross resistant, NCCN lists the preferred chemotherapeutic agents as: an anti-metabolite gemcitabine or XELODA, a microtubule inhibitor such as gemcitabine or ERIBULIN or ABRAXANE.

Renal Cell Carcinoma

Renal cell carcinoma (RCC) is a type of kidney cancer that develops within the kidney's microscopic filtering systems, or more specifically, in the lining of the proximal convoluted tubules. RCC is the most common type of kidney cancer, making up 85% of cases. Kidney cancer is among the 10 most common cancers in the US. The NCI estimated that 65,150 new cases of kidney cancer are expected to be diagnosed in the US in 2013, and 13,680 patients will die of the disease.

A Stage I kidney tumor is 7cm or smaller and is limited to the kidney. In Stage II, the tumor is greater than 7cm in size, but is still limited to the kidney. In Stage III, the tumor has invaded the adrenal gland (which sits atop each kidney), tissue surrounding the kidney or major nearby veins such as the vena cava. Stage III also includes patients with enlarged abdominal lymph nodes. In Stage IV, patients have large tumors extending to surrounding tissues and/or metastases to other distant organs.

The five-year survival rate for kidney cancer is 71%. Exhibit 28 summarizes the five-year survival rates for kidney cancer by stage.

Exhibit 28: Five-Year Survival Rate of Kidney Cancer by Stage

Stage	5-Year Survival Rate
I	81%
II	74%
III	53%
IV	8%

Source: National Cancer Institute's Surveillance, Epidemiology, and End Results (SEER) database.

The most common histology for renal cell carcinoma is clear cell, and for front line therapy NCCN recommends single agent anti-VEGF TKIs SUTENT or VOTRIENT, the combination of AVASTIN with interferon alpha or a clinical trial. Only one therapy, the m-TOR inhibitor TORISEL has been tested specifically in poor risk patients, and NCCN recommends this agent for poor risk clear cell carcinoma. NCCN also notes that for selected patients, NEXAVAR or high dose IL-2 are appropriate first line choices. In the recurrent disease setting, NCCN

recommends a second m-TOR inhibitor, AFINITOR or the VEGF-TKI INLYTA after failing SUTENT or VOTRIENT front-line. Other options include NEXAVAR, TEMODAR, AVASTIN, and the alternate front -line agent, i.e., VOTRIENT in SUTENT failures and vice versa. After failure of IL-2, any of the four approved TKI's are recommended with TEMODAR or AVASTIN as alternates. For non-clear cell tumors, NCCN recommends TEMODAR for poor risk patients and then either TEMODAR, NEXAVAR or SUTENT as preferred options with VOTRIENT, TARCEVA or INLYTA as alternatives.

Hepatocellular Carcinoma

Hepatocellular carcinoma (HCC) is the most common type of liver cancer, accounting for more than 80% of the cases. HCC originates from hepatocytes, which is the predominant liver cell type. The NCI estimates that approximately 30,640 new cases of liver cancer will be diagnosed in the US in 2013, and approximately 21,670 patients will die of the disease. Liver cancer incidence rates are three times higher in men than in women. Liver cancer is much more common in countries in Southeast Asia and sub-Saharan African, and is often the most common type of cancer in those countries. Worldwide, more than 700,000 people are diagnosed with liver cancer annually. The five-year survival rate for liver cancer overall is 15%. Exhibit 29 summarizes the five-year survival rates for liver cancer by stage.

Exhibit 29: Five-Year Survival Rate of Liver Cancer by Stage

Stage	5-Year Survival Rate
Localized (confined to the liver)	28%
Regional (spread to nearby organs or lymph nodes)	7%
Distant (metastatic)	2%

Source: National Cancer Institute's Surveillance, Epidemiology, and End Results (SEER) database.

The only pharmacotherapy recommended for inoperable or metastatic hepatocellular carcinoma is NEXAVAR. NCCN gives the highest weighting of recommendation to subjects with Child-Pugh Class A liver function, but NCCN also recognizes that NEXAVAR can be used in patients with Child-Pugh Class B liver function; however, no data exist for NEXAVAR use in Child-Pugh Class C. The only other options noted by NCCN are a clinical trial or supportive care. Given the poor results with chemotherapy, NCCN recommends that systemic or locally delivered chemotherapy be evaluated in the context of a clinical trial. There are no recommended second-line regimens following failure of NEXAVAR.

FP-1039 Development and Commercialization Agreement With GSK

In March 2011, Five Prime entered into a license and collaboration agreement with Human Genome Sciences (HGS), which was later acquired by GSK. According to the collaboration agreement, GSK/HGS obtained an exclusive license to develop and commercialize FP-1039 and other FGFR1 fusion proteins in the US, the EU and Canada. Five Prime retains rights to develop and commercialize FP-1039 in territories outside the US, the EU and Canada.

Five Prime and HGS agreed to mutually disclose FP-1039 preclinical and clinical data from future trials or studies. Either party may use, at no cost, such exchanged data in regulatory filings. For example, after GSK completes its phase 1b clinical trial of FP-1039, Five Prime would be able to use the clinical data from that filing in regulatory filings in Japan, which is outside of GSK's territory.

In entering into the agreement, GSK/HGS paid Five Prime an upfront license fee of $50 million. GSK-HGS is also obligated to pay contingent payments of up to $435 million, including $70 million for the pre-specified development criteria, up to $195 million for the pre-specified regulatory criteria, and up to $170 million for the pre-specified commercial criteria. According to the pre-specified development criteria, Five Prime could receive a $5 million contingent payment upon GSK's completion of its phase 1b clinical trial and a $15 million contingent payment if GSK initiates a phase 2 clinical trial. The aggregate potential contingent payments could total up to $310 million, instead of $435 million, if certain manufacturing criteria are not met.

Five Prime is also eligible to receive tiered royalty payments on a country-by-country basis from low double-digits to high teens based on net sales of FP-1039 for the longer of patent life of FP-1039, or 12 years after the first commercial sale of FP-1039 in the country.

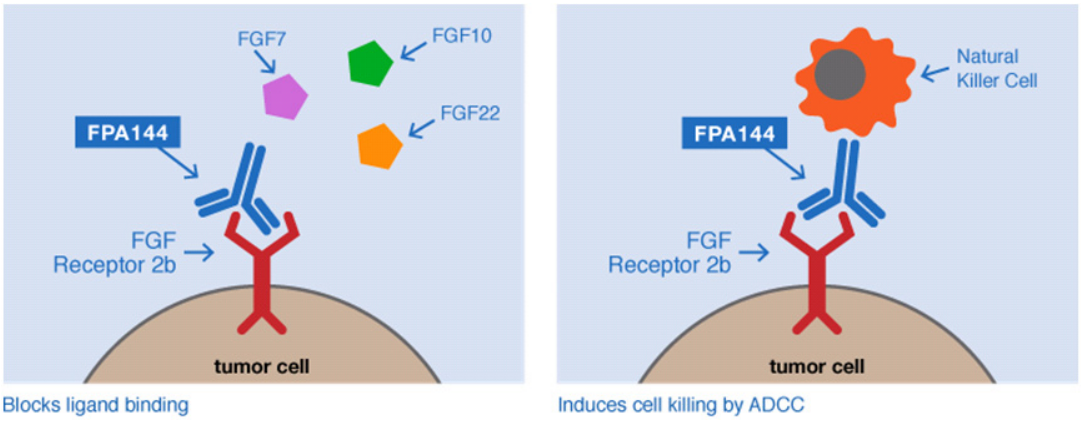
Five Prime has a minority co-promote option for FP-1039 in the US. To exercise this option, Five Prime must notify GSK prior to the later of (i) five days after the BLA filing for FP-1039 or (ii) six months after GSK notifies Five Prime of the anticipated BLA filing. If Five Prime exercises its right to co-promote FP-1039, it would receive a low single-digit increase in the royalty rate relating to net sales in the US.

Selective FGFR2b Antibody FPA144 for Patients with Gastric Cancer

Five Prime is developing, FPA144, a monoclonal antibody directed against the b isoform of FGFR2, or FGFR2b, to treat patients with gastric cancer and potentially other solid tumors such as esophageal, breast, and ovarian cancer. FPA144 was highly effective in blocking the growth of gastric tumors with FGFR2b overexpression. Five Prime plans to develop and commercialize FPA144 on its own, and intends to initiate a phase 1 trial in gastric cancer in 2H14 with preliminary data expected by year-end 2015.

FPA144 binds to FGFR2b and blocks the receptor's interaction with FGF7, FGF10, and FGF22 (Exhibit 30). Note that FGF7, 10 and 22 are among the few ligands that are not targeted by the more broadly acting FP-1039 (refer to Exhibit 11 for FP-1039's specificity). Therefore, the spectrum of anti-tumor activity for FPA144 is different than that of FP-1039. But similar to FP-1039, FPA144 does not affect FGF23 mediated phosphate homeostasis, because FPA144 does not block FGF23 signaling.

Exhibit 30: Schematic Depiction of FPA144's Mechanisms of Action



Source: Five Prime Therapeutics

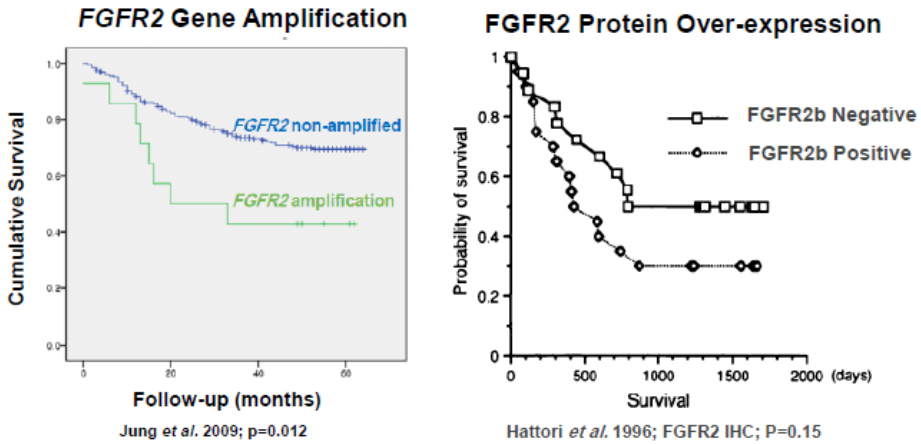
In addition to inhibition of FGF signaling, FPA144 also "marks" FGFR2b-expressing tumor cells and recruits certain immune cells (e.g., Natural Killer cells) to attack the tumor, in a process known as antibody-dependent cell-mediated cytotoxicity (ADCC).

Biologic Rationale for Targeting FGFR2b in Gastric Cancer

FGFR2 gene amplification has been observed in 3%-9% of patients with gastric cancer (Exhibit 31, top panel). In fact, the FGFR2 gene was originally identified as an amplified cDNA from a gastric cancer cell line. FGFR2 gene amplification and protein overexpression have been demonstrated to correlate with poor prognosis (Exhibit 31, bottom panels), likely because of the formation of an autocrine activation loop between FGF2R and its ligands.

Exhibit 31: FGFR2 Amplification in Gastric Cancer and Its Association With Poor Prognosis

Study	Technique	FGFR2 amplification frequency (%)	Clinical sub-type correlation; Comments
Deng et al. 2012	SNP 6.0	~21/233 (9.3%)	Correlation between qPCR and amplification, but some patients with high mRNA no amplification
Matsumoto et al. 2012	PCR (FISH confirmed in 7 cases)	11/267 (4.1%)	FGFR amplification >5 copies in all cases
Kilgour et al 2012	FISH	30/ 408 (7%) Caucasian 15/356 (4%) Korean	Her2+ overlap 2/26 in Caucasian, 0/15 in Korea Amplification correlated with lymph node status
Jung et al. 2012	FISH	14/312 (4.5%) Korean	Higher stage, metastasis, lower survival; The extent of amplification ranged from 3.0 to 30.0-fold
Tsuijimoto et al. 1997	Southern blot	3/57 (5.3%) Japanese	Un-differentiated sub-type
Hara et al. 1998	FISH	3/154 (2.9%) Japanese pts.	Poorly differentiated adenocarcinomas
Xie et al. 2011	FISH	8/195 (4%) Chinese 7/97 (7%) Caucasian	Not reported
Mor et al. 1993	Southern blot	5/139 (3.6%)	Poorly differentiated tumors; The extent of amplification ranged from 5- to 20-fold.

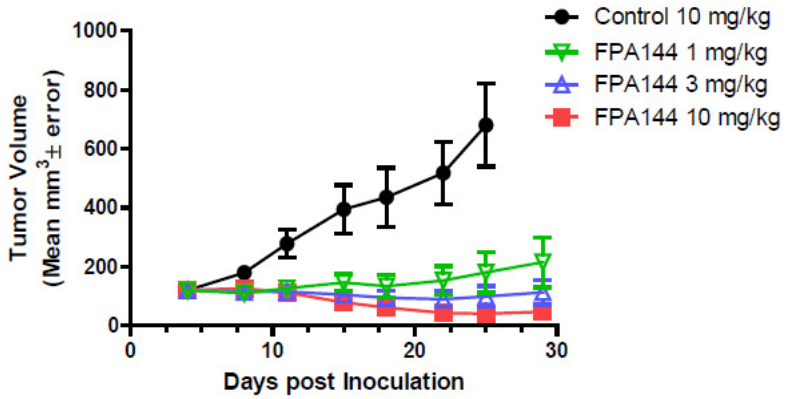


Source: Five Prime Therapeutics

Review of Preclinical Data for FPA144

In preclinical studies, FPA144 was highly effective in blocking the growth of gastric cancers that overexpressed FGFR2b. As shown in Exhibit 32, treatment of human gastric tumors with FGFR2 gene amplification with increasing doses of FPA144, but not with a control antibody, resulted in significant inhibition of tumor growth and led to tumor shrinkage.

Exhibit 32: Anti-tumor Activity of FPA144 in a Mouse Model of Gastric Cancer With FGFR2b Gene Amplification



Source: Five Prime Therapeutics

Five Prime also disclosed that FP-1039, which targets FGFR1c, was not effective against gastric cancer with abnormally high levels of FGFR2b, highlighting the differential anti-tumor activities of FPA144 versus FP-1039.

Future Plans for FPA144 Clinical Development

Five Prime plans to submit an Investigational New Drug (IND) application with FDA and initiate a phase 1 trial of FPA144 in the US and in Asia in 2H14, with preliminary phase 1 clinical data expected by year-end 2015. The phase 1 trial will enroll patients with gastric cancer that expresses abnormally high levels of FGFR2b. If the phase 1 trial demonstrates acceptable safety and evidence of clinical activity of FPA144, Five Prime plans to conduct a multinational phase 2 trial and will consider initiating a phase 1 trial in Japan.

If early evidence of a therapeutic effect is observed, Five Prime plans to meet with regulatory authorities to discuss the possibility of an expedited regulatory path for FPA144. Five Prime also plans to seek orphan drug designation for FPA144 before the end of the phase 1 clinical trial, as well as expedited review and approval programs, including breakthrough therapy and fast track designations, if eligible.

To identify patients most likely to respond to FPA144 treatment, Five Prime plans to screen patients with special staining tests that can identify overexpression of FGFR2b protein on the

surface of tumor cells. Five Prime plans to identify and engage a third party to develop a companion diagnostic that would be used in clinical trials or required for the registration and approval of FPA144.

Given the small patient population and poor survival rate of gastric cancer (see next section), Five Prime believes that gastric cancer will be an orphan indication in the US, and that the subset of patients with FGFR2 gene amplification constitutes an ultra-orphan indication.

Five Prime plans to develop and commercialize FPA144 in the US, and partner the drug for development outside the US. In our opinion, a niche indication with a significant unmet medical need, such as FGFR2 gene amplified gastric cancer, is highly suitable for Five Prime to develop in the US on its own, and an accelerated development timeline is possible.

Commercial Opportunity in FGFR2b Gastric Cancer

Gastric cancer is cancer that forms in tissues lining the stomach. The prevalence of gastric cancer is about 72,250 in the US in 2013, and the NCI estimates that approximately 21,600 new cases will be diagnosed and 10,990 will die of the disease in 2013. Globally, there are more than one million gastric cancer patients, according to the International Agency for Research on Cancer.

According to the literature, approximately 3%-9% of patients with gastric cancer have tumors with FGFR2 gene amplification. Therefore the prevalence rate of gastric cancer with FGFR2-amplifed tumor in the US is between 2,100 and 6,500, and the incidence rate for this population is between 600 and 1,900 per year.

In the US, the five-year survival rate for gastric cancer overall is 27.7%. Approximately 34% of patients have metastatic cancer at diagnosis, and for these patients the five-year survival rate is only 4% (Exhibit 33). Furthermore, patients with FGFR2 gene amplification have even worse prognosis than those without.

Exhibit 33: Five-Year Survival Rate of Gastric Cancer by Stage

Stage	5-Year Survival Rate
Localized (confined to primary site)	63%
Regional (spread to regional lymph nodes)	28%
Distant (metastatic)	4%

Source: National Cancer Institute's Surveillance, Epidemiology, and End Results (SEER) database.

The NCCN guidelines recommend HER2 testing in the case of adenocarcinoma, with the addition of HERCEPTIN to cisplatin and 5FU being the preferred regimen. Outside of HER2 positive disease, NCCN recommends doublet or triplet cytotoxic regimens based on performance status. The core regimen for gastric cancer is cisplatin with a fluoropyrimidine, either 5FU or XELODA with an option in the medically fit to add either docetaxel (DCF) or epirubicin (ECF). The other doublet regimen in the preferred category is 5FU with irinotecan.

Alternative regimens include a taxane plus a platinate, docetaxel plus irinotecan as well as single agent chemotherapy. In the relapsed setting, second-line therapy depends on the composition of the front line regimen and again performance status with single agent taxane or irinotecan preferred for poor performance and irinotecan-based doublets for those with better performance status. In addition to the aforementioned selection of agents, NCCN also recognizes that mitomycin and etoposide have activity in gastric cancer.

FPA008 for Inflammatory Disease

FPA008 is an antibody directed against colony stimulating factor-1 receptor (CSF1R) and is being developed for the treatment of inflammatory diseases, such as rheumatoid arthritis (RA). CSF1R plays an important role in the survival and function of macrophages, which mediates inflammatory responses by secreting proinflammatory cytokines, such as TNFα, IL-6 and IL-1β. A number of therapeutic agents targeting these cytokines, including approved drugs such as HUMIRA (anti-TNFα), ACTEMRA (anti-IL-6R), and KINERET (anti-IL-1Rα), have been demonstrated to be effective for the treatment of inflammatory disorders. FPA008 has the potential to have better efficacy than each of these approved drugs because it could inhibit the production of multiple inflammatory cytokines simultaneously. In addition, FPA008 may have advantage over existing therapies for RA because FPA008 directly inhibits bone-destroying cells known as osteoclasts.

In preclinical studies, incubation with FPA008 reduced levels of inflammatory cytokines TNFα, IL-6, and IL-1β in biopsy samples of inflamed joints from RA patients. Five Prime plans to initiate a phase 1 trial for FPA008 by year-end 2013 and expect preliminary data by year-end 2014.

Role of the Monocyte and Macrophage in Inflammatory Disorders

Macrophages are central to normal inflammation responsible for clearing pathogens as well as chronic inflammation that underlies many diseases. In particular, cytokines produced by activated macrophages have been recognized as important mediators in chronic inflammatory and autoimmune diseases, and approved therapeutics targeting these cytokines have changed the treatment landscape for RA, multiple sclerosis, and Crohn's disease (a form of inflammatory bowel disease).

Macrophages are phagocytes ("eater-cells") of the immune system. They reside in every tissue of the body, although sometimes disguised as specialized cells such as osteoclasts in bone and microglia in the brain. Macrophages constantly survey their surroundings for signs of invading organisms and tissue damage. They recognize danger signals through an array of specialized cell-surface receptors, such as the Toll-like receptors (TLRs). Upon recognizing a danger signal, macrophages can ingest the pathogen or foreign material and also produce immune effector molecules, such as cytokines and chemokines, to activate other cells of the immune system.

Monocytes are precursors of macrophages. Monocytes are generated in the bone marrow and are released into circulation. Monocytes differentiate into macrophages or dendritic cells (DCs) when they extravasate through the endothelium (lining of blood vessels) and take residence in local tissues throughout the body. (A pool of undifferentiated monocytes is also found in the spleen, which serves as a reservoir). In response to inflammation, the number of monocytes increases, leading to an increase in the numbers of tissue-resident macrophages and DCs.

In RA, a large number of macrophages infiltrate the synovial membrane (membrane that lines the cavity of a joint). Surface markers expressed on these macrophages (such as a high level of major histocompatibility, or MHC, class II molecules) indicate that these cells are activated and capable of promoting inflammation and tissue damage. Monocytes circulating in the blood of RA patients also appear to be markedly activated. In the joints of RA patients, a variety of macrophage-derived cytokines are present, including TNFα, IL-1, IL-6, IL-8, IL-15, IL-18, TGFβ, and MIF.

In addition to their role in inflammation, monocytes/macrophages also contribute to bone erosion in RA by differentiating into osteoclasts, a cell type that specializes in bone resorption. Excessive differentiation into osteoclasts is a key cause of pathological bone erosion in RA.

Rheumatoid Arthritis Primer

RA is a chronic autoimmune inflammatory disorder manifested by pain, swelling, stiffness and redness in multiple joints of the body followed by cartilage destruction, bone erosion, and subsequent deformities, and impaired physical function.

RA is a systemic autoimmune disease, even though it translates primarily into joint inflammation and joint destruction. In RA, both the innate and the adaptive branches of the immune system are activated: macrophages produce large amounts of proinflammatory cytokines; B cells mount autoantibody responses; and CD4+ T cells release T-cell-derived cytokines and provide stimulation to B cells and macrophage.

For reasons not fully understood, the systemic immune reaction in RA results in an inflammatory process primarily at the site of the synovium membrane. In normal state, the synovial membrane (also called the synovium) is a thin membrane that attaches to skeletal tissues at the bone-cartilage interface, forming a pocket to line the joint surface. The synovial membrane has two separate layers: the lining cell layer, which is in contact with the intraarticular cavity and normally composed of synovial lining cells (SLCs) arranged 1 to 3 cells thick, and the sublining layer, which contains blood vessels, lymphatics and nerves. In RA, several significant changes take place at the synovial membrane. The synovial lining cell layer becomes hyperplastic and may grow to 6 to 10 cells thick. A large number of inflammatory cells, comprised predominantly of lymphocytes, plasma cells and macrophages, infiltrate the sublining layer. As the disease progress, the hyperplastic synovial tissue becomes invasive and adheres to the surface of articular cartilage. The invasive tissue derived from synovial membrane is termed pannus. Cells within pannus produce proteinases, which degrade and destroy cartilage and underlying bone, leading to joint instability. In addition to causing joint

damage, RA sometimes can affect other organs as well, such as the skin, eyes, lungs, and blood vessels.

Several classes drugs are used to treat RA, including glucocorticoids, nonsteroidal antiinflammatory drugs (NSAIDs), and disease-modifying antirheumatic drugs (DMARDs), which include both traditional DMARDs and biologic agents. Glucocorticoids, such as prednisone and prednisolone, rapidly improve RA symptoms such as pain and stiffness. But because of many possible side effects, glucocorticoids are used only as short-term treatment until other slower-acting drugs with greater ability to prevent joint damage begin to work. NSAIDs, such as aspirin and ibuprofen, can relieve pain and reduce minor inflammation, but do not slow RA progression. DMARDs, on the other hand, can significantly reduce inflammation and joint damage and preserve joint structure and function. Traditional DMARDs include methotrexate, hydroxychloroquine, sulfasalazine, and leflunomide. Biologic agents are important additions to traditional small-molecule DMARDs. There are several types of biologic DMARDs, including anti-TNFα injectables (e.g., HUMIRA, REMICADE, ENBREL), anti-IL-6R mAb (ACTEMRA), anti-IL-1Rα mAb (KINERET). Biologic agents are often reserved for patients who do not respond to traditional DMARDs or those who cannot tolerate DMARDs in doses high enough to control inflammation. Biologics may be used alone or in combination with traditional DMARDs (e.g., methotrexate), NSAIDs, and/or glucocorticoids. In November 2012, XELJANZ (tofacitinib), the first of a new class of RA treatment called JAK kinase inhibitors, was approved by FDA.

Biologic Rationale for Targeting CSF1 and IL-34 in RA

CSF1 and IL-34 are essential soluble factors that regulate the survival, proliferation, and differentiation of monocytes/macrophages. Elevated levels of CSF1 and IL-34 have been observed in the synovium of RA patients, suggesting a role for these cytokines in the pathogenesis of RA.

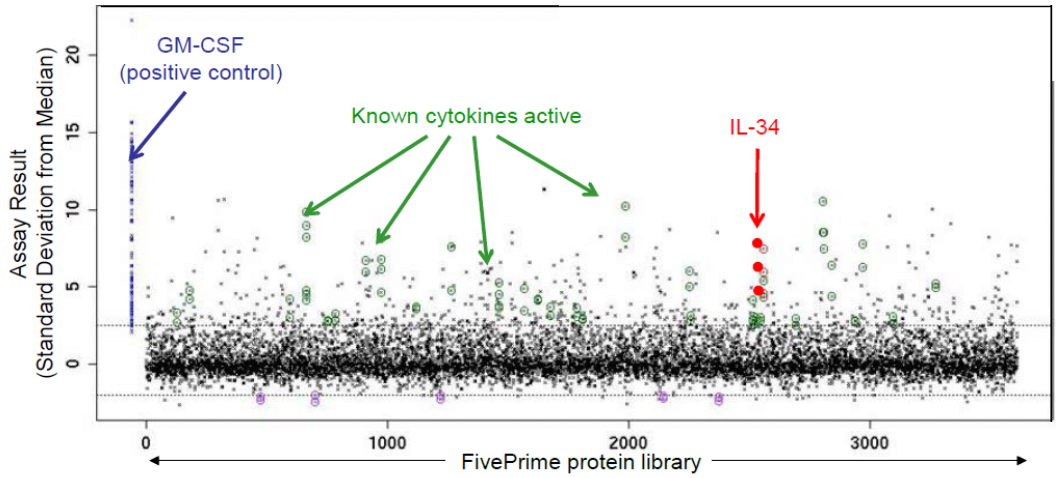
The critical role of CSF1 in controlling the number of macrophage can be appreciated from mice that carry a natural mutation in CSF1 (the op/op mice). These mice exhibit deficiency in macrophage, monocytes, microglia, and osteoclasts, all of which are cell types of the monocyte/macrophage linage.

CSF1 and IL-34 share little amino-acid homology, but they both bind to and signal through CSF1R (also know as C-FMS). CSF1R is a protein tyrosine kinase receptor expressed on monocytes/macrophages and signaling through this receptor promotes monocytes/macrophages proliferation and survival. Although both CSF1 and IL-34 trigger CSF1R signaling, the *in vivo* expression patterns of CSF1 and IL-34 may be different. CSF-1 is produced constitutively by a wide variety of cells and is present in circulation. IL-34, at least in mice, is mainly produced by keratinocytes (skin cells) and neurons, according to a recent gene-knock-in study.

Upon ligand binding, CSF1R undergoes dimerization and autophosphorylation of tyrosine residues in its intracytoplasmic domain. Phosphorylated tyrosines then behave as docking sites for adaptor molecules that mediate activation of signaling pathways, including ERK1/2 and PI3K/Akt pathways.

Notably, IL-34 was discovered by Five Prime scientists in 2008 as a novel cytokine that stimulated the proliferation of human primary monocytes. The discovery was made using the company's proprietary protein library, which included IL-34, and the high-throughput cell-based screen technology (Exhibit 34). Using another component of the company's technology platform, the ligand-receptor matching technology, Five Prime also identified CSF1R as the receptor for IL-34. These discoveries clearly demonstrated effectiveness of Five Prime's protein therapeutics discovery platform.

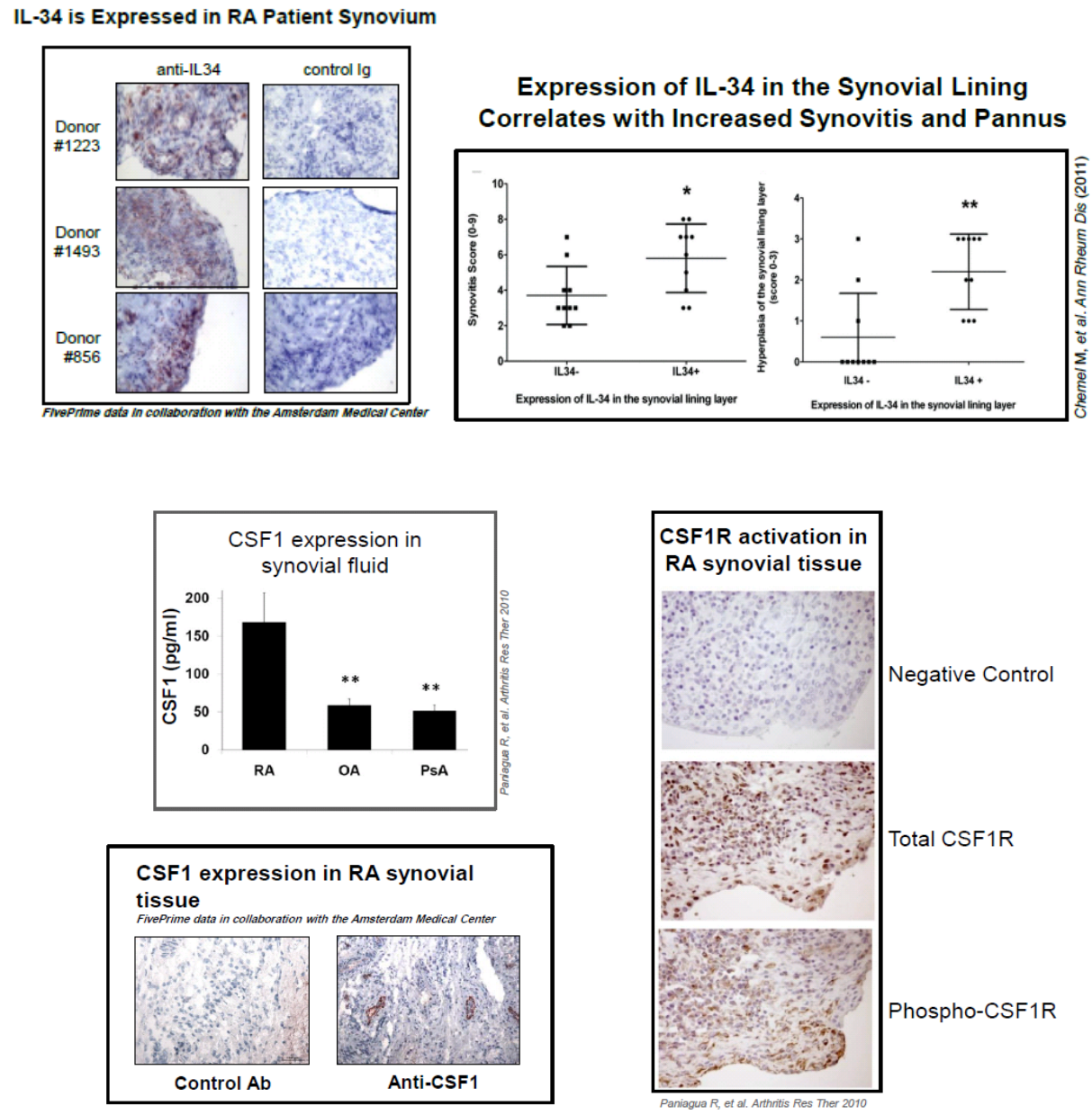
Exhibit 34: Discovery of IL-34 as a Novel Cytokine Using Five Prime's Protein Library and Cell-based Screen Technology Platform



Source: Five Prime Therapeutics

CSF1 levels are elevated under many disease conditions, including infections, cancer, and chronic inflammatory disease. In the synovium of RA patients, elevated levels of both CSF1 and IL-34 have been observed (Exhibit 35). More importantly, the levels of IL-34 expression in the synovial lining correlated with increased synovitis and pannus (Exhibit 35, upper right panel). (Pannus refers to the thickening synovial tissue that invades and destructs articular cartilage and bone).

Exhibit 35: Elevated Levels of IL-34 and CSF1 in RA Patients Synovium



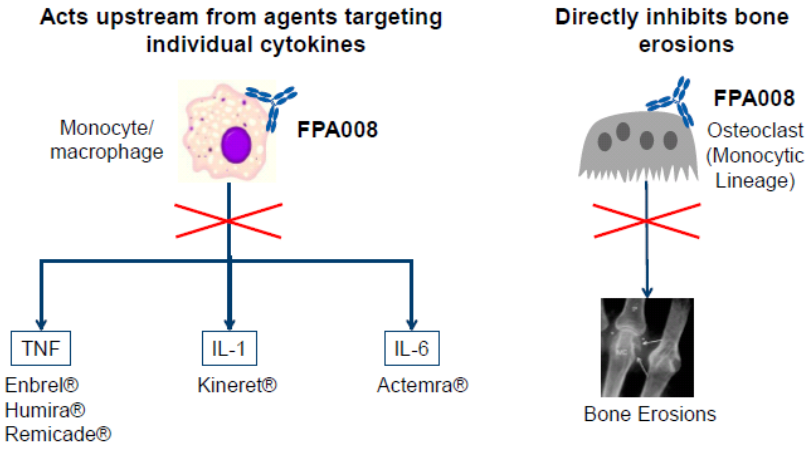
Source: Five Prime Therapeutics

Given the importance of CSF1R signaling in monocyte/macrophage homeostasis and function, inhibition of CSF1R signaling represents an attractive strategy for inflammatory diseases such as RA. A number of therapeutic agents targeting macrophage-derived cytokines, such as TNFα, IL-6, and IL-1, have been demonstrated to be effective for the treatment of RA, including approved drugs such as HUMIRA (anti-TNFα), ACTEMRA (anti-IL-6R), and KINERET (anti-IL-1Rα).

FPA008 is an anti-CSF1R antibody designed to block the ability of IL-34 and CSF1 to bind to and activate CSF1R. FPA008 reduces the numbers and activity of monocytes/macrophages that cause inflammatory disease and prevents the production and release of inflammatory factors. FPA008 may have better efficacy than the anti-cytokine therapeutics, such as anti-TNF, anti-IL-6R and anti-IL-1Rα, because FPA008 could inhibit the production of multiple inflammatory cytokines simultaneously.

Another advantage of blocking CSF1R, as opposed to blocking macrophage-derived cytokines, is that blocking of CSF1R signaling also inhibits the function of osteoclast, the specialized macrophage cell type that breaks down bone. Therefore FPA008 may directly suppress bone destruction in the joints of patients with RA.

Exhibit 36: Potential Advantages of FPA008 Over Current Therapies



Source: Five Prime Therapeutics

Validation Through Success of Other Macrophage Targeted Therapies

The approach of targeting macrophages for the treatment of RA has been recently validated by positive results from two phase 2 studies involving antibodies that inhibit the GM-CSF pathway, which is another signaling pathway that activates macrophage function. Results from these phase 2 studies may provide a frame of reference when evaluating FPA008.

Granulocyte-macrophage colony-stimulating factor (GM-CSF) stimulates the production of monocytes and granulocytes (including neutrophils) from stem cells, and also activates mature tissue macrophages, leading to an increased production of proinflammatory cytokines, chemokines and proteases. In RA patients, levels of GM-CSF in the synovial fluid and levels of GM-CSF receptor (GM-CSFR) expression in synovial tissue cells were both increased, suggesting a role for GM-CSF in the pathogenesis of RA.

MOR103 is a fully human anti-GM-CSF antibody developed by MorphoSys. In June 2013, GlaxoSmithKline (GSK) assumed responsibility for global development and commercialization of MOR103 through a license agreement with MorphoSys.

In a phase 1/2 study conducted by MorphoSys from 2009 to 2012, MOR103 demonstrated promising clinical activity in RA. In this randomized, double-blind, placebo-controlled trial, 96 patients with mild-to-moderate RA were enrolled at 26 centers in Germany, Netherlands, Poland, Bulgaria, and Ukraine, and received four weekly doses of 0.3mg/kg, 1.0mg/kg, or 1.5mg/kg MOR103. The majority of the subjects were on a stable regimen of disease modifying anti-rheumatic drugs. The primary endpoint of the trial was the safety and tolerability of multiple doses of MOR103 in patients with active RA. The secondary endpoints were pharmacokinetics, immunogenicity, and efficacy of MOR103 as measured by DAS28, ACR20/50/70 and EULAR response criteria, MRI imaging for synovitis and bone edema as well as patient reported outcomes.

ACR20/50/70 and DAS28 are disease activity scores that help measure the improvement of disease in RA clinical trials. American College of Rheumatology 20 (ACR20) is defined as the percentage of patients who achieved at least a 20% reduction in tender or swollen joint count as well as a 20% reduction in three of the following five parameters: physician's overall assessment of disease activity, C-reactive protein (CRP) or erythrocyte sedimentation rate in blood, degree of physical disability in Health Assessment Questionnaire (HAQ) score, patient's overall assessment of disease activity, and patient's assessment of pain. ACR50 and ACR70 are defined similarly to ACR20 but require 50% and 70% improvement, respectively. Disease Activity Score 28 (DAS28) is a European standard that measures the activity of the disease in RA patients based on 28 defined joints.

The best response in MOR103's phase 1/2 trial was achieved in the 1.0 mg/kg dose cohort, with an ACR20 score of 68% versus an ACR20 score of 7% in the placebo group at week 4 ($p<0.0001$). The onset of action of MOR103 was also remarkably rapid, with up to 40% of patients achieving an ACR20 score within 2 weeks in the 1.0 mg/kg dose cohort. MorphoSys also reported that DAS28 improvement was also rapid and significant over the treatment period of the study, and that MRI scans revealed a reduction of synovitis according to the Rheumatoid Arthritis MRI Scoring System (RAMRIS) system at week 4.

Exhibit 37: Clinical Efficacy of MOR103 in the Phase 1/2 Trial

	Placebo	MOR103 0.3 mg/kg	MOR103 1.0 mg/kg	MOR103 1.5 mg/kg
Patient Number	27	24	22	23
Proportion of Patients Achieving ACR20	7%	25%	68%	30%
Proportion of Patients Achieving ACR50	4%	4%	23%	9%

Source: MorphoSys AG

MOR103 was described as safe and well-tolerated. There were no treatment-related serious adverse events and no obvious differences in the rate of adverse events between the MOR103

and control groups. MorphoSys is also studying MOR103 for the treatment of multiple sclerosis in an ongoing phase 1b dose-escalation trial.

MedImmune (part of AstraZeneca) is developing mavrilimumab, a human mAb targeting the alpha subunit of GM-CSF receptor (GM-CSFRα). In a phase 2 study, mavrilimumab induced rapid and significant responses in RA patients, with an ACR20 rate of 69% vs. 40% placebo and an ACR50 rate of 31% vs. 12% placebo in the highest dose (100mg) cohort at week 12.

In the randomized, double-blind phase 2 study of mavrilimumab, 239 patients with moderate-to-severe RA on background methotrexate therapy were randomized to subcutaneous mavrilimumab 10mg, 30mg, 50mg, 100mg, or placebo every other week for 12 weeks. The primary endpoint was the proportion of patients achieving a ≥1.2 decrease from baseline in DAS28 with CRP measurement (DAS28-CRP) at week 12.

Overall, 55.7% of mavrilimumab-treated patients met the primary endpoint vs. 34.7% placebo (p=0.003) at week 12. Best response was observed in the 100mg dose cohort at 66.7% (p=0.001). Response was rapid and generally occurred within two weeks of treatment initiation. The 100mg dose also demonstrated a significant effect vs. placebo on all categories of the ACR criteria (ACR20 69% vs. 40%, p=0.005; ACR50: 31% vs. 12%, p=0.021; ACR70: 18% vs. 4%, p=0.03). The placebo response rate was noticeably high in this trial, but nevertheless significant improvements were achieved in the mavrilimumab arms.

Exhibit 38: Clinical Efficacy of Mavrilimumab in the Phase 2 Trial

	Placebo	Mavrilimumab			
		10 mg	30 mg	50 mg	100 mg
Patient Number	75	39	41	39	39
Proportion of Patients Achieving ACR20	40%	41%	56%	41%	69%
Proportion of Patients Achieving ACR50	12%	23%	29%	21%	31%
Proportion of Patients Achieving ACR70	4%	5%	10%	8%	18%

Source: BMO Capital Markets, adapted from Burmester G et al., *Ann Rheum Dis* 2013; 72:1445.

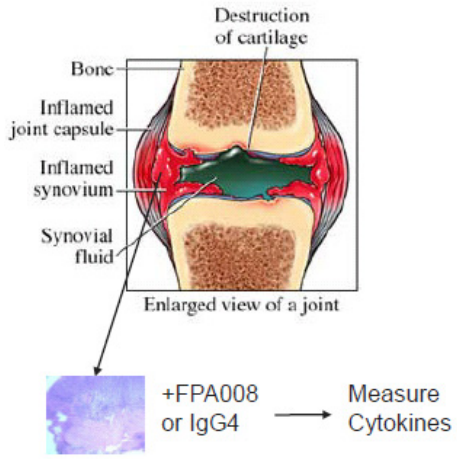
Adverse event were described as generally mild or moderate in intensity in mavrilimumab's phase 2 trial. The frequency or severity of adverse events (AEs) was not dependent on mavrilimumab dose. There was no death or treatment-related severe adverse events (SAEs). Anti-drug antibodies (ADAs) at titers greater than 4 were reported in 1.3% of patients in the placebo arm and 6.3% of patients in the mavrilimumab arms. Although the presence of ADAs was associated with somewhat reduced PK exposure, there was no apparent correlation between ADAs and reduced clinical responses.

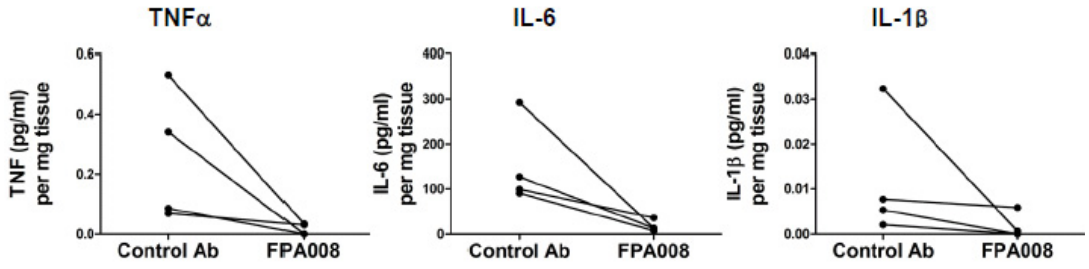
In summary, the phase 2 studies of MOR103 and mavrilimumab in RA have demonstrated that significant clinical benefit can be achieved through the blockade of the GM-CSF pathway. We expect that the blockade of the CSF1/IL-34 signaling pathway by FPA008, which similarly inhibits macrophage activation, could achieve a similar therapeutic effect in RA.

Preclinical Data of FPA008 Suggest Potential Benefit on Bone Erosions

Five Prime's own research as well as research from other groups demonstrated that both IL-34 and CSF1 were present at increased levels in the inflamed joints of RA patients. Incubation of biopsy samples of inflamed joints from RA patients with FPA008 ex vivo demonstrated reduced levels of the inflammatory cytokines TNFα, IL-6 and IL-1β compared with samples incubated with a control antibody (Exhibit 39). These results provided evidence that FPA008 could simultaneously inhibit the production of multiple cytokines that cause inflammation in RA.

Exhibit 39: Reduction of Cytokine Production in RA Patient Joint Tissue After FPA008 Treatment Ex Vivo



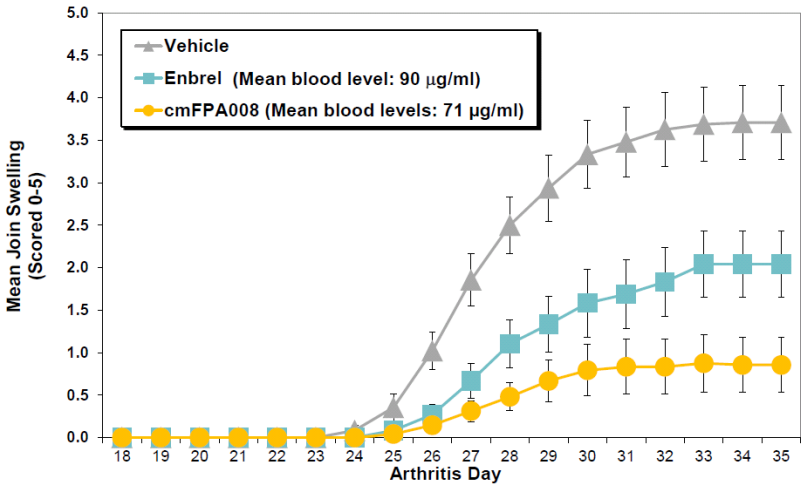


Each pair of linked dots corresponds to samples from the same patient treated with either a control Ab (which does not bind to CSF1R) or with FPA008.

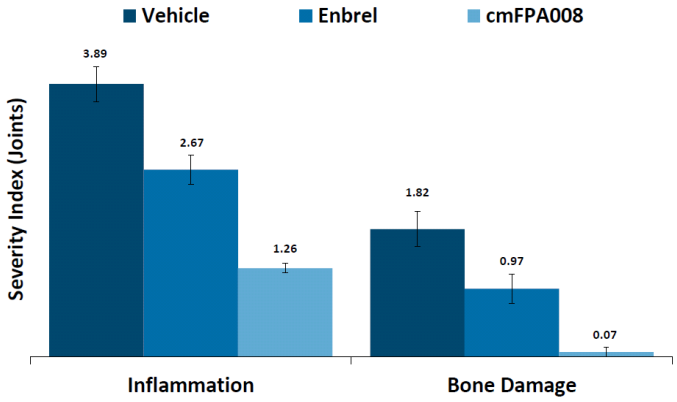
Source: Five Prime Therapeutics

Five Prime has also demonstrated beneficial effects of targeting CSF1R in a mouse model of RA. For this line of studies, Five Prime used a mouse form of FPA008, cmFPA008. In a mouse model of collagen-induced arthritis, cmFPA008 reduced the swelling of the joints and demonstrated better efficacy than ENBREL, an approved protein therapeutic for use in RA that blocks TNFα (Exhibit 40, top panel). Furthermore, cmFPA008 also demonstrated better efficacies than ENBREL in preventing inflammation and bone destruction in the mouse model of RA (Exhibit 40, bottom panel).

Exhibit 40: Treatment with cmFPA008 Prevented Development of Arthritis in a Collagen-Induced Arthritis Model



cmFPA008 and ENBREL were dosed to provide roughly equivalent drug levels in the blood.

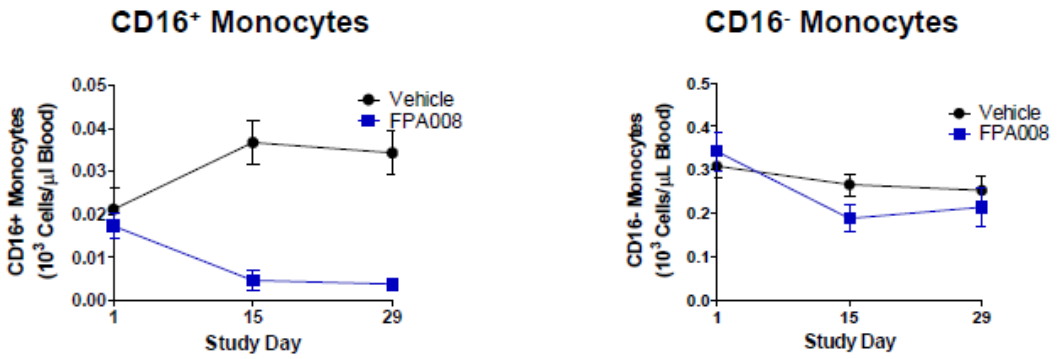


Source: Five Prime Therapeutics

Five Prime has also demonstrated that FPA008 selectively targets a subset of monocytes that are responsible for inflammation. Monocytes can be divided into two subsets: classical monocytes, which express the surface marker CD16 and a high level of CSF1R ($CD16^{+}$

CSF1R^{high}), and inflammatory monocytes, which do not express CD16 and express only low levels of CSF1R (CD16^{-} CSF1R^{low}). Compared to classical monocytes, inflammatory monocytes have a superior capacity to produce TNFα, IL-6 and IL-1. In addition, the percentages of CD16^{+} monocytes in peripheral circulation have been reported to be elevated in patients with RA. Because of their higher expression of CSF1R, CD16^{+} monocytes could be more sensitive to FPA008 treatment. As shown in Exhibit 41, treatment with FPA008 significantly reduced the number of CD16^{+} inflammatory monocytes in cynomolgus monkeys, while having a much smaller effect on CD16^{-} monocytes. In light of these findings, Five Prime plans to use CD16^{+} monocytes as a pharmacodynamic marker.

Exhibit 41: Selective Targeting of Inflammatory Monocytes by FPA008



Source: Five Prime Therapeutics

Five Prime has pointed out that because of its mechanism of action, treatment of FPA008 may lead to elevation in the blood level of creatine kinase (CK), a toxicological biomarker commonly used as an indicator for muscle damage. CK is normally removed from the blood by liver macrophages, and therefore its blood level could be affected by the number or function of liver macrophages. Similarly, liver macrophages also mediate the clearance of aspartate aminotransferase (AST) and alanine aminotransferase (ALT), which are commonly used biomarkers for liver damage.

Five Prime's research has demonstrated that CSF1R pathway inhibition in monkeys increased half-life of labeled CK and resulted in parallel increase of three CKs derived from muscle, heart and brain. Five Prime has suggested that CK elevation in this context is a result of slow clearance rather than muscle damage. Five Prime noted that there were no microscopic liver or muscle findings in these experiments, and that more sensitive markers of muscle and liver function were not affected, including skeletal and cardiac troponins and bilirubin.

Similar findings have been reported for an anti-CSF1 mAb, PD-0360324, developed by Pfizer. In a single ascending dose phase 1 trial of PD-0360324 in healthy volunteers, dose-related increases in the levels of AST and CK were observed largely in the absence of other signals of liver or muscle injury. (PD-0360324 is currently in a phase 2 study in cutaneous lupus erythematosus, an autoimmune disease, and a phase 1 study in chronic pulmonary sarcoidosis,

an inflammatory disease. A phase 1 trial of PD-0360324 + methotrexate in RA patients was completed in June 2009.)

Development Plans and Commercial Opportunity for FPA008 in Rheumatoid Arthritis

Five Prime plans to initiate a phase 1 clinical trial outside the US by year-end 2013 to assess the safety, tolerability and early efficacy of FPA008. The trial will commence in healthy volunteers and transition to testing in patients with RA. Preliminary results from the phase 1 study are expected by year-end 2014. Five Prime also plans to submit an IND for FPA008 in connection with a subsequent phase 2 clinical trial, which will be a randomized study in patients with RA.

Five Prime plans to analyze clinical data from the phase 1 trial to assess biomarkers that may identify subsets of RA patients who would benefit from FPA008 treatment more than other patients. Five Prime believes this approach could help streamline the clinical development process. Five Prime also plans to explore whether a companion diagnostic should be used in later clinical studies of FPA008.

Upon the completion of the phase 1 trial in RA, Five Prime may explore additional inflammatory disease indications for FPA008. Lupus nephritis and multiple sclerosis may be suitable indications, as these disease conditions involve elevated levels of activated monocytes/macrophages in inflamed tissues.

RA occurs in approximately 0.5%-1% of the population in the US as well as globally. The annual incidence of RA in the US has been reported to be approximately 40 per 100,000 persons. Women are about three times more likely than men to develop RA. RA usually develops in the fourth or fifth decades of life and is a major cause of disability. When inadequately treated, RA is associated with reduced life expectancy.

It is estimated that 1.8 million RA patients in the US were treated with a pharmacological agent. In 2012, the top three RA biologic products, HUMIRA, ENBREL, and REMICADE, generated over $25 billion in global sales. Despite the wide array of treatment options, significant portions of RA patients fail to achieve meaningful responses and are not adequately controlled. New drugs with novel mechanisms of action, such as FPA008, may potentially address the unmet medical need in RA.

Intellectual Properties

Five Prime's FP-1039 patent portfolio includes issued patents and pending patent applications covering compositions of matter, methods of use, including certain combination therapies and dosing regimens, and biomarkers relating to FP-1039. This patent portfolio includes patents issued in the US, Europe, Japan, Australia, New Zealand, and Hong Kong. The issued US patents covering composition of matter and methods for using FP-1039 expire in 2026 and 2031, respectively. The issued patents in Europe, Japan, Australia, New Zealand, and Hong Kong covering composition of matter for FP-1039 expire in 2026. Patents that may issue from

pending US and foreign patent applications would expire between 2026 and 2034. The FP-1039 patent portfolio also includes issued US and foreign patents exclusively licensed from the UC Regents that cover composition of matter and methods of producing FP-1039, with the US patents expiring between 2019 and 2020.

Five Prime's FPA144 patent portfolio includes patents and patent applications exclusively licensed from Galaxy, as well as a pending US patent application wholly owned by Five Prime. The patent portfolio licensed from Galaxy includes an issued US patent as well as pending US and foreign patent applications covering compositions of matter and methods of use of FPA144. The issued US composition of matter patent expires in 2029, and patents that may issue from the pending US and foreign applications would expire in 2029. Patents that may issue from the pending and wholly owned US patent application would expire in 2034.

Five Prime's FPA008 patent portfolio includes an issued US patent as well as pending US and foreign patent applications covering compositions of matter, methods of use and biomarkers relating to FPA008. The issued US composition of matter patent expires in 2031, and patents that may issue from these pending US and foreign applications would expire between 2031 and 2033.

Exhibit 42: Five Prime's Intellectual Properties

	Expiration*	Status in US
▪ **FP-1039**		
» Composition of matter	2026	Issued
» Specific dosage regimens	2031	Allowed
» Methods of treatment	2026-2034	Pending
» Methods of selecting patients	2032	Pending
▪ **FPA008**		
» Composition of matter	2031	Issued
» Methods of treatment	2031-2033	Pending
▪ **FPA144**		
» Composition of matter	2029	Issued
» Methods of treatment	2029	Pending

* Expiration dates do not reflect any potential patent term extensions or patent term adjustments under applicable law.

Source: Five Prime Therapeutics

Partnerships

Since 2006, Five Prime has entered into six discovery collaborations with Boehringer Ingelheim, Centocor Research and Development (Centocor), GSK, Pfizer, and UCB. These collaborations have involved identification and characterization of target proteins in several disease areas using Five Prime's protein discovery platform, including the protein library and the cell-based and in vivo screening technologies.

These discovery collaborations have provided Five Prime approximately $104 million in non-equity funding through June 30, 2013. Five Prime also sold shares of convertible preferred stock to Johnson & Johnson Development Corporation, an affiliate of Centocor, Pfizer, and GSK in connection with these discovery collaborations for total equity funding of $63 million.

The collaborations with Boehringer, Centocor, and Pfizer have ended. The collaboration with GSK on FP-1039 has been described in an earlier section titled *FP-1039 Development and Commercialization Agreement with GSK*. There are three other ongoing collaborations.

Exhibit 43: Five Prime's Active Discovery Collaborations

Collaborator	Subject Matter(s)	Protein Claiming (Option) Fees, License Fees and Contingent Milestone Payments	Patent Royalties
gsk	Muscle diseases (sarcopenia & cachexia)	up to $124 million per protein	Not disclosed
gsk	Respiratory diseases (refractory asthma & COPD)	up to $193.5 million per protein	Not disclosed, but higher for Track 2 products for which FivePrime demonstrates proof of mechanism
ucb	Fibrosis-related immunologic and CNS diseases	Not disclosed	Not disclosed

Source: Five Prime Therapeutics

GSK Muscle Diseases Collaboration

In July 2010, Five Prime and GSK entered into a three-year research collaboration and license agreement to identify potential drug targets and drug candidates to treat skeletal muscle diseases. In May 2011, the agreement was amended to include an additional cell-based screen and an in vivo screen using the RIPPS technology. Under the muscle diseases collaboration agreement, Five Prime is conducting three customized cell-based screens and one in vivo screen of the protein library. The original two cell-based screens ended in July 2013 and the other cell-based screen and the in vivo screen will end in May 2014.

Under the muscle diseases collaboration, GSK has the right to evaluate, for limited periods of time, proteins identified in the screens, and the right to obtain an exclusive global license to develop and commercialize products that incorporate or target the selected protein. In December 2012, GSK selected a protein for further evaluation.

Five Prime is eligible to receive up to $124.3 million in potential option exercise fees and contingent payments for each protein target that GSK elects to obtain rights, including aggregate target evaluation and selection fees of up to $1.8 million, preclinical and development-related contingent payments of up to $28.5 million, regulatory-related contingent

payments of up to $40.0 million and commercial-related contingent payments of up to $54.0 million. In addition, Five Prime is also eligible for tiered low- to mid-single digit royalties on net sales for each product that incorporates or targets a licensed protein target.

GSK UK Respiratory Diseases Collaboration

In April 2012, Five Prime and Glaxo Group Limited (GSK UK) entered into a four-year research collaboration and license agreement to identify new therapeutic approaches to treat refractory asthma and chronic obstructive pulmonary disease (COPD). Five Prime plans to conduct up to six customized cell-based screens of its protein library under the respiratory diseases collaboration.

In connection with the agreement, GSK UK made an upfront payment of $7.5 million and purchased shares of Five Prime preferred stock for $10.0 million. Five Prime has received $2.6 million of research funding and is eligible to receive up to an additional $7.9 million through the remainder of the research term, which ends in April 2016.

Under the respiratory diseases collaboration, GSK UK has the right to evaluate, for limited periods of time, proteins identified in the screens, and the right to obtain an exclusive global license to develop and commercialize products that incorporate or target the selected protein.

Before GSK UK exercises its right to obtain an exclusive license to a protein target, GSK UK and Five Prime will decide which protein targets GSK UK will have sole responsibility for the further development and commercialization (referred to as Track 1 Targets) and which protein targets Five Prime will develop through clinical proof of mechanism in either a phase 1 clinical trial or phase 2 clinical trial (referred to as Track 2 Targets).

For Track 1 Targets, GSK UK would have sole responsibility for the further development and commercialization at GSK UK's cost and expense. For Track 2 Targets, Five Prime would have sole responsibility for further development, including preclinical studies, clinical development and manufacturing, at Five Prime's cost and expense through agreed-upon proof-of-mechanism endpoints in a phase 1 or phase 2 clinical trial.

Five Prime is eligible to receive up to $124.3 million in potential target evaluation and selection fees and contingent payments with respect to each Track 1 Target, including per target evaluation and selection fees of up to $1.8 million, preclinical and development-related contingent payments of up to $28.5 million, regulatory-related contingent payments of up to $40.0 million and commercial-related contingent payments of up to $54.0 million. In addition, GSK UK is also obliged to pay Five Prime tiered low- to mid-single digit royalties on net sales for each product that incorporates or targets a Track 1 Target.

Five Prime is eligible to receive up to $193.8 million in potential target evaluation and selection fees and contingent payments with respect to each Track 2 Target, including per target evaluation and selection fees of up to $1.8 million, a clinical proof of mechanism option exercise fee of up to $23.0 million, preclinical and development-related contingent payments of up to $36.5 million, regulatory-related contingent payments of up to $53.0 million and commercial-related contingent payments of up to $79.5 million. In addition, GSK UK is also

obliged to pay Five Prime tiered high-single to low-double digit royalties on net sales for each product that incorporates or targets a Track 2 Target.

UCB Fibrosis and CNS Collaboration

In March 2013, Five Prime entered into a research collaboration and license agreement with UCB to identify innovative biologics targets and therapeutics in the areas of fibrosis-related immunologic diseases and central nervous system (CNS) disorders. Five Prime plans to conduct five customized cell-based and in vivo screens of its protein library under the fibrosis and CNS collaboration. Five Prime expects the completion of the initial research activities under the collaboration agreement by March 2016. Upon the completion of these initial research activities, UCB has up to a two-year evaluation period and may request additional services from Five Prime.

In connection with the agreement, UCB made upfront payments of $8.2 million. Five Prime is eligible to receive up to an additional $6.4 million of technology access fees and research funding under the fibrosis and CNS collaboration from March 2014 to March 2016. In addition, Five Prime may be eligible to receive up to $1.3 million if UCB requests a third fibrosis screen.

UCB has the right to evaluate, for limited periods of time, protein targets identified in the screens, and the right to obtain an exclusive worldwide license to develop and commercialize products that incorporate or target the protein. If UCB elects to obtain an exclusive license to a protein it has evaluated, Five Prime is eligible to receive up to $92.2 million in potential evaluation and selection fees and contingent payments for each protein target, comprising aggregate target evaluation and selection fees of up to $0.4 million, preclinical and development-related contingent payments of up to $11.8 million, regulatory-related contingent payments of up to $20.0 million and commercial-related contingent payments of up to $60.0 million. In addition, UCB is also obliged to pay Five Prime tiered low- to mid-single digit royalties on net sales of each product that incorporates or targets a licensed protein target.

Going forward, Five Prime plans to continue to engage in discussions with pharmaceutical and biotech companies regarding potential new discovery collaborations. Given the continued need for biopharmaceutical companies to replenish pipelines and the scarcity of high-quality drug discovery platforms, we believe Five Prime's unique technology platform will continue to attract partnership interests and provide opportunity for monetization through product and discovery collaboration.

Management

Lewis T. "Rusty" Williams, M.D., Ph.D. founded the company in December 2001 and has been a member of board of directors since January 2002, president and chief executive officer since August 2011, and was executive chairman from July 2003 to January 2012. Previously, Dr. Williams spent seven years at Chiron Corporation (now Novartis Vaccines and Diagnostics), most recently as its chief scientific officer. He also served on Chiron's board of directors from 1999 to 2001. Prior to joining Chiron, Dr. Williams was a professor of medicine at the University of California, San Francisco and served as director of the University's Cardiovascular Research Institution and Daiichi Research Center. Dr. Williams also has served on the faculties of Harvard Medical School and Massachusetts General Hospital and co-founded COR Therapeutics, a biotechnology company focused on cardiovascular disease. He is a member of the National Academy of Sciences and a fellow of the American Academy of Arts and Sciences. Dr. Williams was previously a member of the boards of directors of COR Therapeutics and Beckman Coulter, which are public companies. Dr. Williams holds a BS from Rice University and an MD and a PhD from Duke University.

Marc L. Belsky has been vice president, finance since October 2009. From December 2006 to October 2009, Mr. Belsky was vice president, finance, and chief accounting officer of Cell Genesys, a biotechnology company acquired by BioSante Pharmaceuticals. Prior to 2006, Mr. Belsky served as vice president, Global Visa Commerce at Visa, chief financial officer at Active Aero Group, and chief financial officer at DataWave Systems. Prior to these positions, he spent 15 years at Michigan National Corporation, a holding company for Michigan National Bank which was acquired by BANA Holding Corporation, in positions of increasing responsibility, most recently as senior vice president, U.S. Payment Products and Services. Mr. Belsky started his career as an auditor with Coopers & Lybrand. Mr. Belsky holds a BS in accounting from Wayne State University and an MBA from University of Michigan. He is a certified public accountant, a chartered global management accountant, and a certified treasury professional.

Julie Hambleton, M.D. has been senior vice president and chief medical officer since December 2012. From April 2010 to December 2012, Dr. Hambleton was vice president, clinical development, at Clovis Oncology. From 2003 to April 2010, Dr. Hambleton served at Genentech (now part of Roche) in positions of increasing responsibility, most recently as group medical director, global clinical development. Prior to 2003, Dr. Hambleton served for 10 years in academic positions in the Division of Hematology/Oncology at the University of California, San Francisco, most recently as associate professor of Clinical Medicine. Dr. Hambleton holds a BS from Duke University and an MD from Case Western Reserve University School of Medicine, and is board certified in Hematology and Internal Medicine.

W. Michael Kavanaugh, M.D. has been senior vice president and chief scientific officer since January 2013, and was senior vice president, research and development from February 2009 to January 2013. Previously, Dr. Kavanaugh served at Novartis Vaccines and Diagnostics in positions of increasing responsibility, most recently as vice president of Novartis Vaccines & Diagnostics and executive director of Novartis Institutes of Biomedical Research from 2006 to

February 2009. Dr. Kavanaugh also currently serves as an attending staff physician, Coronary Intensive Care Unit at the San Francisco Veterans Administration Medical Center and as an associate clinical professor of Medicine at the University of California, San Francisco. Dr. Kavanaugh holds a BS in molecular biochemistry and biophysics from Yale University and an MD from Vanderbilt University. He trained in Internal Medicine, Cardiovascular Disease and Molecular and Cellular Biology at the University of California, San Francisco and the Cardiovascular Research Institute. He is board certified in cardiovascular disease and internal medicine.

Aron M. Knickerbocker has been senior vice president and chief business officer since April 2012, and was vice president, business development, from September 2009 to April 2012. From 2001 to September 2009, Mr. Knickerbocker served at Genentech in positions of increasing responsibility, most recently as senior director, business development. Prior to 2001, Mr. Knickerbocker served as director of commercial development at ALZA Corporation (acquired by Johnson & Johnson), as senior manager, corporate development at Amgen, and as a scientist at Bristol-Myers Squibb. Mr. Knickerbocker holds an AB in biology from Washington University in St. Louis and an MBA from the University of Michigan.

Francis W. Sarena has been senior vice president since January 2013, general counsel and secretary since December 2010, and was vice president from December 2010 to January 2013. From December 2008 to July 2010, Mr. Sarena served as vice president, general counsel and secretary of Facet Biotech, a public biotechnology company that was spun off from PDL BioPharma and was later acquired by Abbott Laboratories. From April 2006 to December 2008, Mr. Sarena served at PDL BioPharma in positions of increasing responsibility, most recently as vice president, general counsel and secretary. Prior to 2006, Mr. Sarena was an associate at Bingham McCutchen LLP where he represented public and private life science and high tech clients primarily in merger and acquisition transactions, corporate and securities law matters and equity financing transactions. Mr. Sarena holds a BS in finance from San Francisco State University and a JD from University of California, Berkeley.

Exhibit 44: FPRX Income Statement 2012A-2020E

INCOME STATEMENT ($M)	2012A	1Q13A	2Q13A	3Q13E	4Q13E	2013E	2014E	2015E	2016E	2017E	2018E	2019E	2020E	2021E	2022E	2023E	2024E	2025E
REVENUES																		
Product Revenue	$ -	$ -	$ -	$ -	$ -	$ -	$ -	$ -	$ 12.2	$ 45.1	$ 86.6	$ 128.1	$ 169.6	$ 210.4	$ 246.3	$ 257.9	$ 259.3	$ 259.3
Collaboration Revenue	10.0	3.3	3.3	3.0	3.0	12.5	12.0	12.0	12.0	12.0	12.0	12.0	12.0	12.0	12.0	12.0	12.0	12.0
Other Revenue	-	-	-	-	-	-	20.0	10.0	-	-	-	-	-	-	-	-	-	-
TOTAL REVENUES	**$ 10.0**	**$ 3.3**	**$ 3.3**	**$ 3.0**	**$ 3.0**	**$ 12.5**	**$ 32.0**	**$ 22.0**	**$ 24.2**	**$ 57.1**	**$ 98.6**	**$ 140.1**	**$ 181.6**	**$ 222.4**	**$ 258.3**	**$ 269.9**	**$ 271.3**	**$ 271.3**
EXPENSES (GAAP)																		
Cost of Goods Sold (COGS)	$ -	$ -	$ -	$ -	$ -	$ -	$ -	$ -	$ 0.6	$ 2.3	$ 5.2	$ 12.8	$ 12.7	$ 15.8	$ 12.3	$ 12.9	$ 13.0	$ 13.0
R&D Expense	28.8	8.3	8.3	8.5	8.5	33.5	34.4	34.8	35.2	35.6	36.0	36.4	36.8	37.2	37.6	38.0	38.4	38.8
SG&A Expense	9.0	2.4	2.4	2.5	2.5	9.8	10.4	10.8	11.2	11.6	12.0	12.4	12.8	13.2	13.6	14.0	14.4	14.8
Other	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
TOTAL EXPENSES	37.8	10.6	10.6	11.0	11.0	43.3	44.8	45.6	47.0	49.5	53.2	61.6	62.3	66.2	63.5	64.9	65.8	66.6
Operating Income	**(27.8)**	**(7.4)**	**(7.4)**	**(8.0)**	**(8.0)**	**(30.8)**	**(12.8)**	**(23.6)**	**(22.8)**	**7.6**	**45.3**	**78.5**	**119.2**	**156.3**	**194.8**	**205.0**	**205.6**	**204.8**
Depreciation and amortization	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
EBIT	(27.8)	(7.4)	(7.4)	(8.0)	(8.0)	(30.8)	(12.8)	(23.6)	(22.8)	7.6	45.3	78.5	119.2	156.3	194.8	205.0	205.6	204.8
Interest and other income	0.1	0.0	0.0	0.0	0.0	0.1	0.1	0.1	0.1	0.1	0.2	0.3	0.5	0.8	1.1	1.6	2.0	2.4
Interest and other expense	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Other Income (Expense)	0.1	0.2	0.2	-	-	0.4	-	-	-	-	-	-	-	-	-	-	-	-
Interest and Other Income (Expense)	0.2	0.2	0.2	0.0	0.0	0.5	0.1	0.1	0.1	0.1	0.2	0.3	0.5	0.8	1.1	1.6	2.0	2.4
Pre-Tax Income	(27.6)	(7.2)	(7.2)	(8.0)	(8.0)	(30.3)	(12.7)	(23.5)	(22.8)	7.7	45.5	78.7	119.7	157.0	196.0	206.6	207.6	207.2
Income Taxes	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Net Income (GAAP)	**$ (27.6)**	**$ (7.2)**	**$ (7.2)**	**$ (8.0)**	**$ (8.0)**	**$ (30.3)**	**$ (12.7)**	**$ (23.5)**	**$ (22.8)**	**$ 7.7**	**$ 45.5**	**$ 78.7**	**$ 119.7**	**$ 157.0**	**$ 196.0**	**$ 206.6**	**$ 207.6**	**$ 207.2**
EPS (GAAP) (basic)	$ (2.50)	$ (0.64)	$ (0.64)	$ (0.48)	$ (0.48)	$ (2.24)	$ (0.76)	$ (1.39)	$ (1.30)	$ 0.41	$ 2.44	$ 4.12	$ 6.05	$ 7.63	$ 9.09	$ 9.20	$ 9.32	$ 9.43
EPS (GAAP) (diluted)	**$ (2.50)**	**$ (0.64)**	**$ (0.64)**	**$ (0.44)**	**$ (0.44)**	**$ (2.17)**	**$ (0.71)**	**$ (1.29)**	**$ (1.21)**	**$ 0.38**	**$ 2.28**	**$ 3.85**	**$ 5.65**	**$ 7.12**	**$ 8.49**	**$ 8.59**	**$ 8.70**	**$ 8.80**
Total of Reconciliation Items	1.7	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Net Income (Non-GAAP)	$ (14.9)	$ (7.2)	$ (7.2)	$ (8.0)	$ (8.0)	$ (30.3)	$ (12.7)	$ (23.5)	$ (22.8)	$ 7.7	$ 45.5	$ 78.7	$ 119.7	$ 157.0	$ 196.0	$ 206.6	$ 207.6	$ 207.2
Impact of Adjustments to EPS	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
EPS (Non-GAAP) (basic)	$ (12.63)	$ (0.64)	$ (0.64)	$ (0.48)	$ (0.48)	$ (2.24)	$ (0.76)	$ (1.39)	$ (1.30)	$ 0.41	$ 2.44	$ 4.12	$ 6.05	$ 7.63	$ 9.09	$ 9.20	$ 9.32	$ 9.43
EPS (Non-GAAP) (diluted)	$ (12.63)	$ (0.64)	$ (0.64)	$ (0.44)	$ (0.44)	$ (2.17)	$ (0.71)	$ (1.29)	$ (1.21)	$ 0.38	$ 2.28	$ 3.85	$ 5.65	$ 7.12	$ 8.49	$ 8.59	$ 8.70	$ 8.80
Weighted average shares outstanding (basic)	11.0	11.2	11.2	16.7	16.7	14.0	16.7	16.9	17.5	18.4	18.7	19.1	19.8	20.6	21.5	22.5	22.3	22.0
Weighted average shares outstanding (diluted)	11.0	11.2	11.2	18.0	18.0	14.6	18.0	18.1	18.8	19.7	20.0	20.4	21.2	22.0	23.1	24.1	23.9	23.5

Source: Company reports and BMO Capital Markets

Exhibit 45: FPRX Balance Sheet 2012A-2020E

BALANCE SHEET (M)	2012A	2013E	2014E	2015E	2016E	2017E	2018E	2019E	2020E
Current Assets									
Cash and cash equivalents	$ 11.4	$ 42.9	$ 30.3	$ 6.8	$ 34.0	$ 41.7	$ 87.2	$ 166.0	$ 285.7
Short-term investments	26.6	19.3	19.3	19.3	19.3	19.3	19.3	19.3	19.3
Total cash, cash equivalents, and short-term investments	$ 38.0	$ 62.2	$ 49.6	$ 26.1	$ 53.3	$ 61.0	$ 106.5	$ 185.3	$ 305.0
Accounts Receivables	0.4	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1
Restricted Cash	-	-	-	-	-	-	-	-	-
Inventories	-	-	-	-	-	-	-	-	-
Prepaid and other current assets	0.7	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0
Total Current Assets	$ 39.1	$ 63.4	$ 50.7	$ 27.2	$ 54.5	$ 62.2	$ 107.7	$ 186.4	$ 306.2
Leasehold improvements	-	-	-	-	-	-	-	-	-
Property and equipment, net	4.6	4.3	4.3	4.3	4.3	4.3	4.3	4.3	4.3
Patents and licensed technology	-	-	-	-	-	-	-	-	-
Intangibles, net	-	-	-	-	-	-	-	-	-
Other assets	0.4	1.7	1.7	1.7	1.7	1.7	1.7	1.7	1.7
TOTAL ASSETS	**$ 44.1**	**$ 69.4**	**$ 56.7**	**$ 33.2**	**$ 60.5**	**$ 68.2**	**$ 113.7**	**$ 192.4**	**$ 312.2**
Current Liabilities									
Accounts payable	2.5	3.3	3.3	3.3	3.3	3.3	3.3	3.3	3.3
Accrued payroll	2.3	1.7	1.7	1.7	1.7	1.7	1.7	1.7	1.7
Accrued expenses	0.3	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7
Accrued interest	-	-	-	-	-	-	-	-	-
Payables to related parties	-	-	-	-	-	-	-	-	-
Income taxes payable	-	-	-	-	-	-	-	-	-
Current portion of other long-term obligations	-	-	-	-	-	-	-	-	-
Current portion of deferred rent	-	-	-	-	-	-	-	-	-
Current portion of deferred revenue	7.5	9.0	9.0	9.0	9.0	9.0	9.0	9.0	9.0
Other current liabilities	0.6	0.3	0.3	0.3	0.3	0.3	0.3	0.3	0.3
Total Current Liabilities	$ 13.1	$ 15.0	$ 15.0	$ 15.0	$ 15.0	$ 15.0	$ 15.0	$ 15.0	$ 15.0
Convertible notes payable	-	-	-	-	-	-	-	-	-
Accrued interest on convertible notes payable	-	-	-	-	-	-	-	-	-
Other long-term obligations, less current portion	-	-	-	-	-	-	-	-	-
Deferred revenue, less current portion	7.3	10.0	10.0	10.0	10.0	10.0	10.0	10.0	10.0
Deferred rent	2.4	2.4	2.4	2.4	2.4	2.4	2.4	2.4	2.4
Other liabilities	0.9	0.8	0.8	0.8	0.8	0.8	0.8	0.8	0.8
TOTAL LIABILITIES	$ 23.7	$ 28.2	$ 28.2	$ 28.2	$ 28.2	$ 28.2	$ 28.2	$ 28.2	$ 28.2
Shareholder's Equity									
Series A convertible preferred stock	84.6	84.6	84.6	84.6	84.6	84.6	84.6	84.6	84.6
Series A1 convertible preferred stock	11.0	11.0	11.0	11.0	11.0	11.0	11.0	11.0	11.0
Series A2 convertible preferred stock	33.9	33.9	33.9	33.9	33.9	33.9	33.9	33.9	33.9
Series A3 convertible preferred stock	6.8	6.8	6.8	6.8	6.8	6.8	6.8	6.8	6.8
Common stock, at par	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Additional paid-in capital	6.8	57.9	57.9	57.9	107.9	107.9	107.9	107.9	107.9
Accumulated other comprehensive income	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Accumulated deficit	(122.7)	(153.0)	(165.6)	(189.2)	(211.9)	(204.2)	(158.7)	(79.9)	39.8
TOTAL SHAREHOLDER'S EQUITY (DEFICIT)	$ 20.4	$ 41.3	$ 28.6	$ 5.1	$ 32.3	$ 40.0	$ 85.5	$ 164.3	$ 284.0
TOTAL LIABILITIES AND SHAREHOLDER'S EQUITY	**$ 44.1**	**$ 69.4**	**$ 56.7**	**$ 33.2**	**$ 60.5**	**$ 68.2**	**$ 113.7**	**$ 192.4**	**$ 312.2**

Source: Company reports and BMO Capital Markets

Exhibit 46: FPRX Cash Flow Statement 2012A-2020E

CASH FLOW STATEMENT (M)	2012A	2013E	2014E	2015E	2016E	2017E	2018E	2019E	2020E
Cash Flow From Operating Activities									
Net Income	$ (27.6)	$ (8.0)	$ (8.2)	$ (8.4)	$ (3.8)	$ 5.6	$ 14.9	$ 23.2	$ 33.6
Adjustments to reconcile net income to cash from operations									
Depreciation & amortization	1.6	0.9	0.9	0.9	0.9	0.9	0.9	0.9	0.9
Amortization of premium on investments, net	0.5	-	-	-	-	-	-	-	-
Gain on disposal of property and equipment	(0.0)	-	-	-	-	-	-	-	-
Stock-based compensation	1.7	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0
Deferred income taxes	-	-	-	-	-	-	-	-	-
Other	(0.1)	(1.0)	(1.0)	(1.0)	(1.0)	(1.0)	(1.0)	(1.0)	(1.0)
Working Capital Adjustments									
Prepaids and other assets	0.4	-	-	-	-	-	-	-	-
Accounts payable	0.0	-	-	-	-	-	-	-	-
Accrued payroll	(0.0)	-	-	-	-	-	-	-	-
Accrued expenses	-	-	-	-	-	-	-	-	-
Accrued interest	-	-	-	-	-	-	-	-	-
Receivable from collaborative partners	0.4								
Payable to related parties	(3.0)	-	-	-	-	-	-	-	-
Income taxes payable	-	-	-	-	-	-	-	-	-
Deferred revenue	7.4	-	-	-	-	-	-	-	-
Deferred rent	0.5	-	-	-	-	-	-	-	-
Other assets and liabilities, net	(0.2)	-	-	-	-	-	-	-	-
Total Working Capital Increase (Decrease)	$ 5.4	$ -	$ -	$ -	$ -	$ -	$ -	$ -	$ -
TOTAL CASH FROM OPERATIONS	**$ (18.4)**	**$ (7.1)**	**$ (7.3)**	**$ (7.5)**	**$ (3.0)**	**$ 6.5**	**$ 15.8**	**$ 24.1**	**$ 34.4**
Cash From Investing Activities									
Purchases of short-term investments	(45.4)	-	-	-	-	-	-	-	-
Maturities and sales of short-term investments	64.6	-	-	-	-	-	-	-	-
Purchases of property and equipment	(0.7)	-	-	-	-	-	-	-	-
Acquisitions of patents	-	-	-	-	-	-	-	-	-
Acquisitions of licenses	-	(0.9)	(0.9)	(0.9)	(0.9)	(0.9)	(0.9)	(0.9)	(0.9)
Increase in patents, deposits and other assets	0.0	-	-	-	-	-	-	-	-
TOTAL CASH FROM INVESTING	$ 18.5	$ (0.9)	$ (0.9)	$ (0.9)	$ (0.9)	$ (0.9)	$ (0.9)	$ (0.9)	$ (0.9)
Cash From Financing Activities									
Proceeds from long-term debt borrowings	6.8	-	-	-	-	-	-	-	-
Repayment of borrowings	-	-	-	-	-	-	-	-	-
Payments of financing costs for an initial public offering	-	-	-	-	-	-	-	-	-
Proceeds from exercise of common stock options	-	-	-	-	-	-	-	-	-
Payments under capital lease obligation	(0.0)	-	-	-	-	-	-	-	-
Common stock issuance	0.1	-	-	-	-	-	-	-	-
TOTAL CASH FROM FINANCING	$ 6.9	$ -	$ -	$ -	$ -	$ -	$ -	$ -	$ -
Increase (decrease) in cash and cash equivalents	7.0	(8.0)	(8.2)	(8.4)	(3.8)	5.6	14.9	23.2	33.6
Cash and cash equivalents at beginning of year	4.4	50.9	38.4	15.1	37.8	36.1	72.3	142.8	252.2
Cash and cash equivalents at end of year	$ 11.4	$ 42.9	$ 30.3	$ 6.8	$ 34.0	$ 41.7	$ 87.2	$ 166.0	$ 285.7

Source: Company reports and BMO Capital Markets

FP-1039 NSCLC Market

SOLID TUMORS	2012A	2013E	2014E	2015E	2016E	2017E	2018E	2019E	2020E
U.S. MARKET									
Lung Cancer Incidence	180,000	180,000	180,000	180,000	180,000	180,000	180,000	180,000	180,000
Non-Small Cell Lung Cancer (NSCLC) Incidence	135,000	135,000	135,000	135,000	135,000	135,000	135,000	135,000	135,000
% Squamous of NSCLC	30%								
Squamous NSCLC Patients		40,500	40,500	40,500	40,500	40,500	40,500	40,500	40,500
% FGFR1 Positive									
FGFR1 Positive Patients		8,910	8,910	8,910	8,910	8,910	8,910	8,910	8,910
FP-1039 Squamous NSCLC Penetration		0.0%	0.0%	0.0%	11.0%	23.0%	35.0%	47.0%	59.0%
FP-1039 Squamous NSCLC Patients		0	0	0	579	1,648	2,718	3,787	4,856
Dosing Duration (months)									
Pricing (per month)		$ 10,000	$ 10,000	$ 10,000	$ 10,000	$ 10,000	$ 10,000	$ 10,000	$ 10,000
Price per Patient		$ 240,000	$ 240,000	$ 240,000	$ 240,000	$ 240,000	$ 240,000	$ 240,000	$ 240,000
FP-1039 Squamous NSCLC U.S. Sales ($M)		$ -	$ -	$ -	$ 34.7	$ 98.9	$ 163.1	$ 227.2	$ 291.4
Royalty Rate 15%									
FP-1039 Squamous NSCLC U.S. Royalties ($M)		**$ -**	**$ -**	**$ -**	**$ 5.2**	**$ 14.8**	**$ 24.5**	**$ 34.1**	**$ 43.7**
EX-U.S. MARKET									
Lung Cancer Incidence	180,000	180,000	180,000	180,000	180,000	180,000	180,000	180,000	180,000
Non-Small Cell Lung Cancer (NSCLC) Incidence	135,000	135,000	135,000	135,000	135,000	135,000	135,000	135,000	135,000
% Squamous of NSCLC	30%								
Squamous NSCLC Patients		40,500	40,500	40,500	40,500	40,500	40,500	40,500	40,500
% FGFR1 Positive									
FGFR1 Positive Patients		8,910	8,910	8,910	8,910	8,910	8,910	8,910	8,910
FP-1039 Squamous NSCLC Penetration		0.0%	0.0%	0.0%	0.0%	11.0%	23.0%	35.0%	47.0%
FP-1039 Squamous NSCLC Patients		0	0	0	0	579	1,648	2,718	3,787
Dosing Duration (months)									
Pricing (per month)		$ 10,000	$ 10,000	$ 10,000	$ 10,000	$ 10,000	$ 10,000	$ 10,000	$ 10,000
Price per Patient		$ 240,000	$ 240,000	$ 240,000	$ 240,000	$ 240,000	$ 240,000	$ 240,000	$ 240,000
FP-1039 Squamous NSCLC Ex-U.S. Sales ($M)		$ -	$ -	$ -	$ -	$ 34.7	$ 98.9	$ 163.1	$ 227.2
Royalty Rate 15%									
FP-1039 Squamous NSCLC Ex-U.S. Royalties ($M)		**$ -**	**$ -**	**$ -**	**$ -**	**$ 5.2**	**$ 14.8**	**$ 24.5**	**$ 34.1**

Source: Company reports and BMO Capital Markets

FP-1039 SCCHN Market

SOLID TUMORS	2012A	2013E	2014E	2015E	2016E	2017E	2018E	2019E	2020E
U.S. MARKET									
Head and Neck Cancer (SCCHN) Incidence	30,000	30,000	30,000	30,000	30,000	30,000	30,000	30,000	30,000
% FGFR1 Positive	17%								
FGFR1 Positive Patients		5,100	5,100	5,100	5,100	5,100	5,100	5,100	5,100
FP-1039 SCCHN Penetration		0.0%	0.0%	0.0%	4.0%	12.0%	20.0%	28.0%	36.0%
FP-1039 SCCHN Patients		-	-	-	128	459	867	1,275	1,683
Dosing Duration (months)									
Pricing (per month)		$ 10,000	$ 10,000	$ 10,000	$ 10,000	$ 10,000	$ 10,000	$ 10,000	$ 10,000
Price per Patient		$ 240,000	$ 240,000	$ 240,000	$ 240,000	$ 240,000	$ 240,000	$ 240,000	$ 240,000
FP-1039 SCCHN U.S. Sales ($M)		$ -	$ -	$ -	$ 7.7	$ 27.5	$ 52.0	$ 76.5	$ 101.0
Royalty Rate 15%									
FP-1039 SCCHN U.S. Royalties ($M)		**$ -**	**$ -**	**$ -**	**$ 1.1**	**$ 4.1**	**$ 7.8**	**$ 11.5**	**$ 15.1**
EX-U.S. MARKET									
Head and Neck Cancer (SCCHN) Incidence	30,000	30,000	30,000	30,000	30,000	30,000	30,000	30,000	30,000
% FGFR1 Positive	17%								
FGFR1 Positive Patients		5,100	5,100	5,100	5,100	5,100	5,100	5,100	5,100
FP-1039 SCCHN Penetration		0.0%	0.0%	0.0%	4.0%	12.0%	20.0%	28.0%	36.0%
FP-1039 SCCHN Patients		-	-	-	128	459	867	1,275	1,683
Dosing Duration (months)									
Pricing (per month)		$ 10,000	$ 10,000	$ 10,000	$ 10,000	$ 10,000	$ 10,000	$ 10,000	$ 10,000
Price per Patient		$ 240,000	$ 240,000	$ 240,000	$ 240,000	$ 240,000	$ 240,000	$ 240,000	$ 240,000
FP-1039 SCCHN Ex-U.S. Sales ($M)		$ -	$ -	$ -	$ 7.7	$ 27.5	$ 52.0	$ 76.5	$ 101.0
Royalty Rate 15%									
FP-1039 SCCHN Ex-U.S. Royalties ($M)		**$ -**	**$ -**	**$ -**	**$ 1.1**	**$ 4.1**	**$ 7.8**	**$ 11.5**	**$ 15.1**

Source: Company reports and BMO Capital Markets

FP-1039 Breast Cancer Market

SOLID TUMORS	2012A	2013E	2014E	2015E	2016E	2017E	2018E	2019E	2020E
U.S. MARKET									
Breast Cancer Incidence	75,000	75,000	75,000	75,000	75,000	75,000	75,000	75,000	75,000
% FGFR1 Positive	11%								
FGFR1 Positive Patients		8,250	8,250	8,250	8,250	8,250	8,250	8,250	8,250
FP-1039 Breast Cancer Penetration		0.0%	0.0%	0.0%	4.0%	12.0%	20.0%	28.0%	36.0%
FP-1039 Breast Cancer Patients		-	-	-	206	743	1,403	2,063	2,723
Dosing Duration (months)									
Pricing (per month)		$ 10,000	$ 10,000	$ 10,000	$ 10,000	$ 10,000	$ 10,000	$ 10,000	$ 10,000
Price per Patient		$ 240,000	$ 240,000	$ 240,000	$ 240,000	$ 240,000	$ 240,000	$ 240,000	$ 240,000
FP-1039 Breast Cancer U.S. Sales ($M)		$ -	$ -	$ -	$ 12.4	$ 44.6	$ 84.2	$ 123.8	$ 163.4
Royalty Rate 15%									
FP-1039 Breast Cancer U.S. Royalties ($M)		**$ -**	**$ -**	**$ -**	**$ 1.9**	**$ 6.7**	**$ 12.6**	**$ 18.6**	**$ 24.5**
EX-U.S. MARKET									
Breast Cancer Incidence	75,000	75,000	75,000	75,000	75,000	75,000	75,000	75,000	75,000
% FGFR1 Positive	11%								
FGFR1 Positive Patients		8,250	8,250	8,250	8,250	8,250	8,250	8,250	8,250
FP-1039 Breast Cancer Penetration		0.0%	0.0%	0.0%	4.0%	12.0%	20.0%	28.0%	36.0%
FP-1039 Breast Cancer Patients		-	-	-	206	743	1,403	2,063	2,723
Dosing Duration (months)									
Pricing (per month)		$ 10,000	$ 10,000	$ 10,000	$ 10,000	$ 10,000	$ 10,000	$ 10,000	$ 10,000
Price per Patient		$ 240,000	$ 240,000	$ 240,000	$ 240,000	$ 240,000	$ 240,000	$ 240,000	$ 240,000
FP-1039 Breast Cancer Ex-U.S. Sales ($M)		$ -	$ -	$ -	$ 12.4	$ 44.6	$ 84.2	$ 123.8	$ 163.4
Royalty Rate 15%									
FP-1039 Breast Cancer Ex-U.S. Royalties ($M)		**$ -**	**$ -**	**$ -**	**$ 1.9**	**$ 6.7**	**$ 12.6**	**$ 18.6**	**$ 24.5**

Source: Company reports and BMO Capital Markets

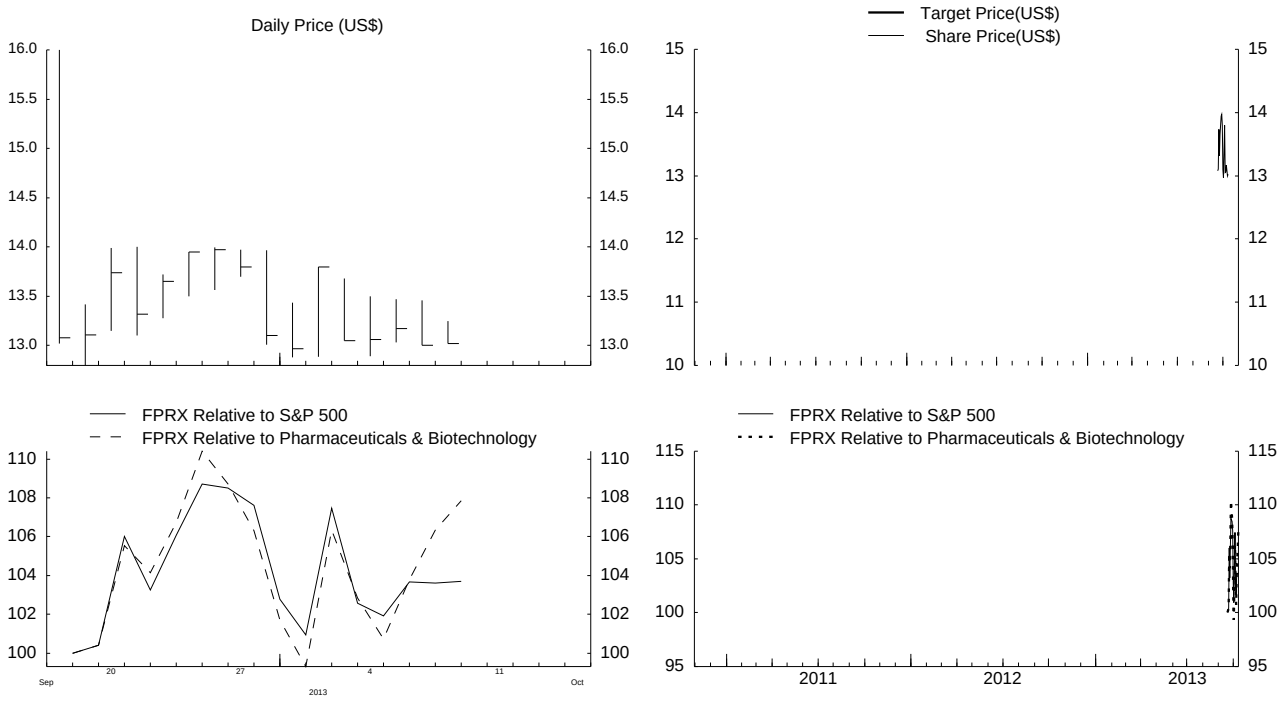
FP-1039 SCLC Market

SOLID TUMORS	2012A	2013E	2014E	2015E	2016E	2017E	2018E	2019E	2020E
U.S. MARKET									
SCLC Incidence	35,000	35,000	35,000	35,000	35,000	35,000	35,000	35,000	35,000
% FGFR1 Positive	6%								
FGFR1 Positive Patients		2,100	2,100	2,100	2,100	2,100	2,100	2,100	2,100
FP-1039 SCLC Penetration		0.0%	0.0%	0.0%	4.0%	12.0%	20.0%	28.0%	36.0%
FP-1039 SCLC Patients		-	-	-	53	189	357	525	693
Dosing Duration (months)									
Pricing (per month)		$ 10,000	$ 10,000	$ 10,000	$ 10,000	$ 10,000	$ 10,000	$ 10,000	$ 10,000
Price per Patient		$ 240,000	$ 240,000	$ 240,000	$ 240,000	$ 240,000	$ 240,000	$ 240,000	$ 240,000
FP-1039 SCLC U.S. Sales ($M)		$ -	$ -	$ -	$ 3.2	$ 11.3	$ 21.4	$ 31.5	$ 41.6
Royalty Rate 15%									
FP-1039 SCLC U.S. Royalties ($M)		**$ -**	**$ -**	**$ -**	**$ 0.5**	**$ 1.7**	**$ 3.2**	**$ 4.7**	**$ 6.2**
EX-U.S. MARKET									
SCLC Incidence	35,000	35,000	35,000	35,000	35,000	35,000	35,000	35,000	35,000
% FGFR1 Positive	6%								
FGFR1 Positive Patients		2,100	2,100	2,100	2,100	2,100	2,100	2,100	2,100
FP-1039 SCLC Penetration		0.0%	0.0%	0.0%	4.0%	12.0%	20.0%	28.0%	36.0%
FP-1039 SCLC Patients		-	-	-	53	189	357	525	693
Dosing Duration (months)									
Pricing (per month)		$ 10,000	$ 10,000	$ 10,000	$ 10,000	$ 10,000	$ 10,000	$ 10,000	$ 10,000
Price per Patient		$ 240,000	$ 240,000	$ 240,000	$ 240,000	$ 240,000	$ 240,000	$ 240,000	$ 240,000
FP-1039 SCLC Ex-U.S. Sales ($M)		$ -	$ -	$ -	$ 3.2	$ 11.3	$ 21.4	$ 31.5	$ 41.6
Royalty Rate 15%									
FP-1039 SCLC Ex-U.S. Royalties ($M)		**$ -**	**$ -**	**$ -**	**$ 0.5**	**$ 1.7**	**$ 3.2**	**$ 4.7**	**$ 6.2**

Source: Company reports and BMO Capital Markets

Other companies mentioned (priced as of the close on October 11, 2013):
Ariad Pharmaceuticals (ARIA, $4.26, rated Market Perform)
AstraZeneca (AZN, $50.96, not rated)
Bristol-Myers Squibb (BMY, $47.68, rated Market Perform by Alex Arfaei)
Eli Lilly (LLY, $48.88, rated Market Perform by Alex Arfaei)
GlaxoSmithKline (GSK, $49.80, not rated)
Johnson & Johnson (JNJ, $89.45, not rated)
Novartis (NVS, $74.95, not rated)
Pfizer (PFE, $28.72, rated Outperform by Alex Arfaei)
Roche (RHHBY-5, $65.74, not rated)

Five Prime Therapeutics (FPRX)



FPRX - Rating as of 23-Sep-13 = NR

Last Daily Data Point: October 9, 2013

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Hold	Market Perform	59.4%	13.1%	51.1%	56.9%	50.2%	41.7%
Sell	Underperform	4.9%	3.4%	1.1%	6.4%	1.5%	5.6%

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