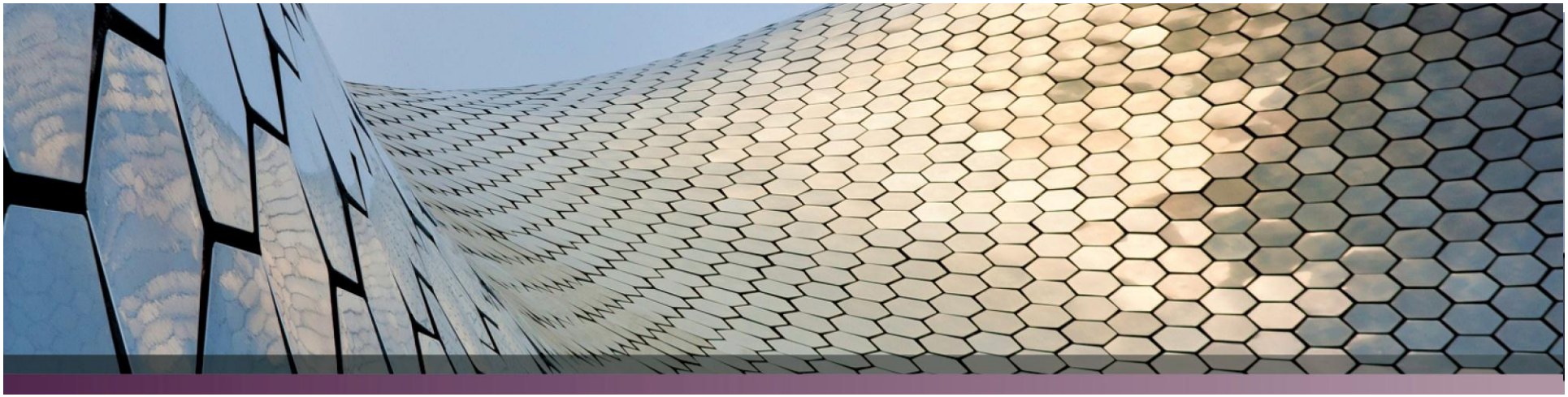

Five Prime Therapeutics (FPRX, $13.19): Promising Oncology/Inflammatory Disease Pipeline with Impressive Platform Technology; Initiating Coverage with BUY (PT $20)

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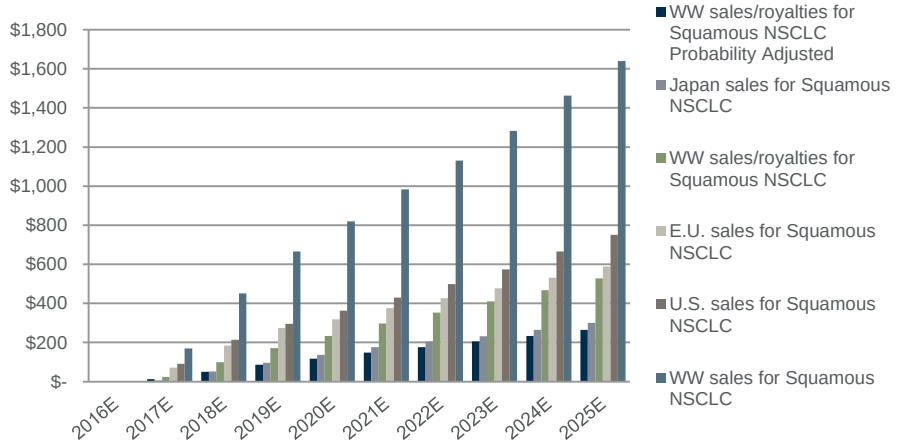
FPRX – Company Background

COMPANY DESCRIPTION

Five Prime Therapeutics (FPRX) is focused on antibody, ligand trap, and protein therapeutics for cancer and inflammatory diseases. The company's drug discovery platform is based on a proprietary library of >5,600 human proteins that represent a majority of the targets available for large molecule drugs. FPRX's lead clinical candidate is FP-1039/GSK3052230 (partnered with GSK), which is entering Ph.Ib testing for FGFR1+ NSCLC. Other potential indications for this ligand trap inhibitor of FGFR1 signaling include breast, head and neck, renal cell, and liver cancer. FPRX is developing FPA008, an anti-CSF1R antibody, for inflammatory diseases, and the company intends to begin Ph.I testing of the drug by YE'13. In FRPX's preclinical pipeline, FPA144, an anti-FGFR2 antibody, should enter the clinic in 2014 for gastric cancer. FPRX has three active discovery collaborations with GSK, another with UCB, and has successfully completed collaborations with Boehringer Ingelheim, Centocor, and PFE.

Projected FP-1039 Squamous NSCLC Sales/Royalties ($M)



Source: Guggenheim Securities, LLC

PRODUCT PORTFOLIO

Product	Pre Clin	I	II	Mkt	Indication
FP-1039				2017E	NSCLC
FP-1039				2018-2019E	Breast cancer & other solid tumors (RCC/HNC)
FPA008					Inflammatory diseases
FPA144					Stomach cancer
Undisclosed Candidates					Cancer, immunotherapy,

Source: Company Documents and Guggenheim Securities, LLC

KEY FINANCIALS

Market Cap	$212.2M
52 week high-low	$12.80-$16.00
Inst. own	NA
Enterprise Value	$182.8M
Total Cash	$28M
Total Cash Per Share	$1.76

KEY QUESTIONS

- What is the likelihood of positive initial results in 2H14 from the recently initiated Ph.Ib trial of FP-1039 in FGFR1+ squamous NSCLC?
- What is the potential for additional cancer indications for FP-1039?
- Even though in early stage development, is there potential for a rapid path to approval of FP-1039 (2H17)?
- What is the market opportunity for FP-1039 in FGFR1+ squamous NSCLC?
- With FPA008 about to enter the clinic for inflammatory diseases, what is this candidate's potential differentiation vs. established therapies?
- Assuming Ph.I interim data by YE'15, what is the LT potential of FPA144 in FGFR2+ gastric cancer?
- How unique and potentially productive is FPRX's protein therapeutic discovery platform?

FPRX – Investment Thesis

Initiating with BUY, $20 Price Target

• **We believe FPRX is attractive, based on a promising early stage oncology/inflammatory disease pipeline and a productive platform.** FPRX's lead compound, FP-1039/GSK3052230, a FGF ligand trap partnered with GSK, is in Ph.Ib testing for FGFR1-amplified NSCLC, and we believe this drug has a good probability of success in this indication and potentially other FGFR1-amplified solid tumors, including mBC, HNC, and RCC. The company's late preclinical pipeline includes FPA008, an anti-CSF1R mAb for RA/inflammatory diseases, which should enter Ph.I testing by YE'13, and FPA144, an anti-FGFR2b mAb for gastric cancer, likely entering the clinic in '14. Importantly, all three of these candidates were discovered or identified by FPRX using the company's unique protein therapeutic discovery technology. FPRX is led by CEO Dr. Rusty Williams, a distinguished scientist with years of biotechnology industry experience, and we believe the company's other key leaders are established industry veterans. Although we expect limited NT clinical newsflow for FPRX, we believe the stock is attractive for longer-term investors, based primarily on FP-1039's potential.

• **We believe FP-1039 has potential in NSCLC and additional solid tumors.** FP-1039 targets the FGFR1 signaling pathway, which is known to be a primary driver in a subset of lung, breast, and head and neck cancers when dysregulated. In particular, we believe there is good evidence that FGFR1 amplification leads to poorer prognosis in squamous NSCLC. Based on this, several other biotechnology/pharmaceutical companies have attempted to target the FGFR1 pathway, with promising efficacy but concerning on-target side effects, such as severe weight loss and hyperphosphatemia. In contrast, in a recently completed Ph.I trial, FP-1039 was reasonably well tolerated, with no evidence of typical FGFR1 side effects. Although the best response demonstrated in the Ph.I trial was stable disease, we do not view this as surprising, as pts were not screened for amplification of FGFR1. Based on a favorable side effect profile and a clear molecular rationale for targeting the FGFR1 pathway in cancers driven by FGFR1 amplification, we see a good probability of FP-1039's success in subpopulations of NSCLC and other solid tumors.

• **GSK has very recently initiated a Ph.Ib trial of FP-1039 in FGFR1-amplified cancers, with first results expected in 2H14.** The trial will consist of three arms: 1) 1st-line squamous NSCLC, FP-1039 combined with paclitaxel/carboplatin, 2) 2nd-line squamous NSCLC, FP-1039 combined with docetaxel, and 3) 3rd/later-line solid tumors (NSCLC, mBC, HNC, and RCC), FP-1039 monotherapy. Notably, in contrast to the Ph.I solid tumor trial, this trial will only include pts with molecularly confirmed FGFR1 amplification. Given the relatively low efficacy bar set by chemotherapeutics and newer targeted agents in FGFR1 amplified squamous NSCLC, we see a good probability that this trial will yield positive efficacy results in 2H14.

FPRX– Investment Thesis (cont'd)

Initiating with BUY, $20 Price Target

• **Possible accelerated approval of FP-1039 in FGFR1+ squamous NSCLC.** We believe there is a significant unmet need for efficacious and, notably, relatively well tolerated FGFR1 inhibitors in FGFR1+ squamous NSCLC and other solid tumors where FGFR1 is amplified. Based on this, we see the potential for accelerated approval of FP-1039 on positive Ph.II/III results in NSCLC as early as 2017. We believe the FDA is currently open to early approval of targeted cancer therapies, provided there are very well-defined pt populations that experience a substantial clinical benefit from these drugs. We believe FP-1039 is likely to fit this description in FGFR1+ squamous NSCLC. If the Ph.Ib results for the drug are positive, consistent with our expectation, we expect GSK will move rapidly forward with a Ph.II/III trial in this setting, which could be completed in late '16/early '17. On this timeline, we see a reasonable probability (50%) of the first U.S. approval of FP-1039 in mid-'17/2H17.

• **We estimate FP-1039 will exceed $1B in worldwide sales in FGFR1+ squamous NSCLC.** NSCLC is currently the leading cause of cancer death in the world, with ~750,000 new cases expected worldwide in '13. Squamous NSCLC represents ~30% of these cases, or ~225,000 pts. There is fairly conclusive scientific evidence that FGFR1 amplification occurs in 20–25% of squamous NSCLCs, defining a large, ~50,000 pt candidate pool for FG-1039. FPRX is entitled to a tiered, low-double digit to high-teen royalty on U.S/E.U. FP-1039 sales, and the company currently retains Japanese rights to the drug. Assuming ~50% penetration and $90,000/year U.S. pricing, consistent with marketed targeted therapies, we estimate worldwide FP-1039 sales exceeding $1B by '22. We note that our estimates are focused only on squamous NSCLC, despite FP-1039' s meaningful LT potential in additional FGFR1+ solid tumors.

• **FPRX's next candidate to enter the clinic, FPA008, has the potential to improve upon the efficacy of established anti-inflammatory disease drugs.** FPA008's mechanism of action should incorporate the effective attributes of existing RA/psoriasis drugs, like AMGN's Enbrel and ABT's Humira, while adding additional efficacy from the downregulation of monocytes and macrophages that these drugs do not impact. FPRX plans to begin Ph.I testing of FPA008 in healthy volunteers by YE'13, with first clinical results likely in '14. We believe FPA008's potentially immunosuppressive effects, while likely to be associated with high efficacy in RA, could lead to side effects, such as opportunistic infections. For this reason, we view the first Ph.I safety results for FPA008 as an important milestone for FPRX.

FPRX– Investment Thesis (cont'd)

Initiating with BUY, $20 Price Target

• **FPA144, entering late preclinical development, could be a significant advance for FGFR2+ gastric cancer.** The FGFR2 gene is amplified in a small subset of gastric cancers (~5%) and is associated with significantly poorer prognosis. FPA144 is an anti-FGFR2 mAb that is effective in preclinical models of FGFR2+ gastric cancer, both through inhibition of FGFR2 signaling and direct cell killing. Although FPA144 is not scheduled to enter the clinic until '14, we believe it could be a LT value driver for FPRX.

• **FPRX has a unique protein therapeutics technology platform.** FPRX has the capability to target >5,600 extracellular proteins with antibodies and/or protein therapeutics, which we believe gives the company a significant advantage vs. competitors. Although there has been an assumption that the "low hanging fruit" has been picked among antibody/protein therapeutics, we believe this is likely an artifact of the limitations of drug discovery that is driven by non-systematic approaches. Based on progress with FP-1039, FPA008, and FP144, along with additional preclinical programs in cancer immunotherapy and other disease settings, we believe FPRX's technology has demonstrated the potential to overcome certain of these limitations.

• **FPRX's unique technology has led to several major collaboration deals, and we believe this deal flow is likely to continue.** The company has three major collaborations with GSK, including, notably, development of FP-1039, and has successfully completed several other collaborations with large pharmaceutical companies. To date, FPRX has generated more than $220M of non-dilutive funding from collaborations, which has enabled the company to operate without significant outside financing since 2005. FPRX has stated that it intends to form at least one new deal a year, going forward, and we expect the company to announce a new collaboration in the 4Q13–2Q14 time frame, which would likely lead the stock higher.

• **D.C. Scorecard: Neutral, with a 3.2 average score.** FPRX currently has no meaningful Washington, D.C. presence, which we believe is appropriate for a developmental-stage company. Importantly, we believe FP-1039, which is focused on a well defined subpopulation of NSCLC, and potentially other cancers, should have favorable positioning with the FDA's ODAC, given this division's recent positive stance on targeted cancer therapies.

FPRX– Valuation, Key Metrics, and Risks

Initiating with Buy, $20 Price Target

Our $20 price target is based on a forward, 10-year DCF of probability-adjusted sales estimates for FP-1039 in NSCLC. We assign a 50% probability of clinical/commercial success for FP-1039 in FGFR1+ stage III/IV squamous NSCLC. Given positive Ph.I results for the biologic in this setting, we believe this is an appropriate, if not conservative, probability adjustment. Our valuation applies a 15.5% discount rate to reflect the relatively early stage of FPRX's lead program and a 2% terminal growth rate to reflect the company's strong collaboration profile and its proprietary drug discovery platform to continue to identify new protein therapeutic targets.

Risks to Our Price Target

Key risks to our price target include, but are not limited to, negative clinical trial results, either related to safety or efficacy, for FPRX's drug candidates; failure to gain U.S./E.U. regulatory approval for FP-1039, FPA008, or FPA144; emerging clinical results for competitive therapies to these therapies in NSCLC/RA/GC; failure of FPRX's collaborative partners, most importantly GSK, to adequately advance development of clinical candidates; failure of FPRX to generate adequate financing; challenges to FPRX's intellectual property positions; and lower-than-expected U.S./ROW sales of FP-1039.

Terminal growth rate

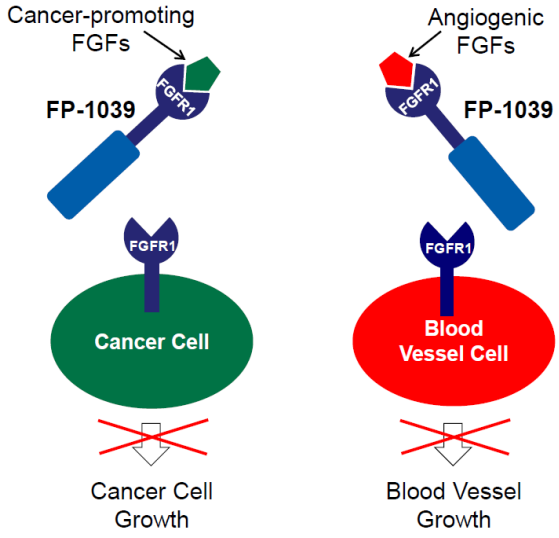
Discount rate	1.00%	1.25%	1.50%	1.75%	**2.00%**	2.25%	2.50%	2.75%	3.00%
14.50%	$ 21.51	$ 21.91	$ 22.32	$ 22.75	**$ 23.19**	$ 23.65	$ 24.13	$ 24.64	$ 25.16
14.75%	$ 20.66	$ 21.04	$ 21.42	$ 21.83	**$ 22.24**	$ 22.68	$ 23.13	$ 23.61	$ 24.10
15.00%	$ 19.85	$ 20.20	$ 20.57	$ 20.95	**$ 21.34**	$ 21.75	$ 22.18	$ 22.63	$ 23.09
15.25%	$ 19.07	$ 19.41	$ 19.75	$ 20.11	**$ 20.49**	$ 20.87	$ 21.28	$ 21.69	$ 22.13
15.50%	**$ 18.33**	**$ 18.64**	**$ 18.97**	**$ 19.31**	**$ 19.67**	**$ 20.03**	**$ 20.41**	**$ 20.81**	**$ 21.22**
15.75%	$ 17.62	$ 17.92	$ 18.23	$ 18.55	**$ 18.88**	$ 19.23	$ 19.59	$ 19.96	$ 20.35
16.00%	$ 16.93	$ 17.22	$ 17.51	$ 17.82	**$ 18.13**	$ 18.46	$ 18.80	$ 19.15	$ 19.52
16.25%	$ 16.28	$ 16.55	$ 16.83	$ 17.12	**$ 17.42**	$ 17.73	$ 18.05	$ 18.38	$ 18.73
16.50%	$ 15.66	$ 15.91	$ 16.18	$ 16.45	**$ 16.73**	$ 17.03	$ 17.33	$ 17.65	$ 17.97
16.75%	$ 15.06	$ 15.30	$ 15.55	$ 15.81	**$ 16.08**	$ 16.36	$ 16.64	$ 16.94	$ 17.25

Source:Guggenheim Securities, LLC

FPRX – FGF-FGFR1 Pathway Background

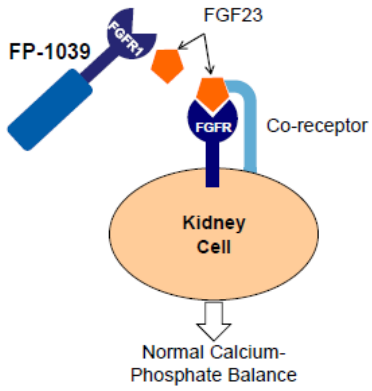
FGF ligands and their receptors, the FGFRs, regulate tumor cell proliferation and angiogenesis. There are 22 known proteins in the FGF family, and the FGFs have diverse, and sometimes overlapping, biological roles, including both mitogenic and hormonal signaling. FGF/FGFR pathway dysregulation, most frequently through amplification of the FGFR1 gene leading to FGF-independent receptor activation, has been implicated as a key driver of tumor progression in several cancers, including squamous NSCLC, mBC, and HNC. In addition, we believe it is well established that FGF/FGFR signaling has a key role in promoting angiogenesis in highly vascular tumors, as RCC.

The development of drugs targeting the FGFR1 pathway has been slowed by on-target side effect concerns. Based on the fairly extensive clinical/preclinical results with 1st-generation TKIs targeting FGFR1, we believe non-selective blockade of the receptor generally leads to unacceptable side effects, substantially limiting the therapeutic window for these drugs. In rats, treatment of solid tumors with PD176067, a small-molecule inhibitor of FGFR1, resulted in hyperphosphatemia and calcium-phosphorus deposition in organs. More notably, in Ph.I/II clinical trials, NVS's BGJ398 and AZN's AZD4547 were both shown to cause increases in serum phosphate levels and retinal side effects. We believe these effects on phosphate metabolism are caused by the blockade of hormonal FGF signaling, specifically FGF-23, which regulates serum phosphate and calcium levels. FPRX's solution to this problem is FP-1039, an FGF ligand trap that blocks mitogenic FGFs but does not inhibit hormonal FGFs like FGF-23. Based on this specificity, we believe that FP-1039 will likely be better tolerated than alternative FGFR1-targeted therapies.



Source: Five Prime

- FGF23 regulates phosphate metabolism
- FGFR tyrosine kinase inhibitors (TKIs)
  - » block FGF23 action
    - ↳raise blood phosphate
      - ↳may cause tissue calcification
- FP-1039
  - » does not block FGF23
    - ↳does not raise blood phosphate



Source: Five Prime

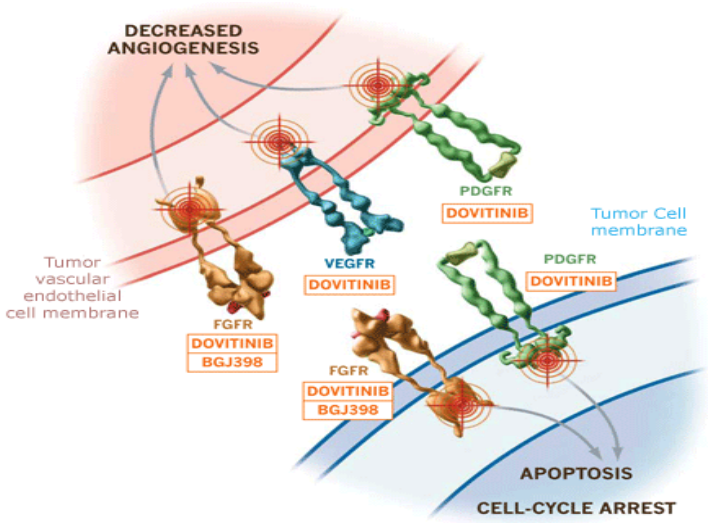
FPRX – FGF-FGFR1 Pathway Background (cont'd)

We believe the clinical track record for FGFR1 inhibitors establishes a clear rationale for FP-1039 in NSCLC and other solid tumors. Despite questionable side effect profiles, 1st-/2nd- generation FGFR1 inhibitors have shown anti-cancer activity in several settings. NVS's 1st-generation FGFR1/VEGFR inhibitor, dovitinib, demonstrated evidence of anti-cancer activity in FGFR1+ mRCC pts in a Ph.II trial (16 mos. OS in evaluable pts vs. 10.2 mos. OS in pts who previously failed VEGFR and mTOR inhibitor treatment). Further, recently reported Ph.III dovitinib results in mRCC showed comparable, albeit not superior, efficacy versus Bayer's sorafenib. 2nd-generation small molecule FGFR1 inhibitors, like AZD4547 and BGJ398, have also shown early signs of efficacy.

Ph.I results for AZD4547 in NSCLC and other FGFR1+ cancers show encouraging evidence of anti-tumor activity. In the Ph.I trial, 21 pts with FGFR1 or FGFR2 amplified tumors received AZD4547 80 mg bid. Importantly, one squamous NSCLC pt with high-level FGFR1 amplification had a PR lasting 12 weeks following progression on two prior lines of therapy. Four additional pts (one mBC, two squamous NSCLC, two transitional cell carcinomas) had durable SD (>24 wks.) as the best response. Notably, however, AZD4547 was associated with typical FGFR1-associated side effects, including hyperphosphatemia, dry skin, and retinal detachment. There is an ongoing Ph.II proof-of-concept trial assessing the efficacy of AZD4547 in 48 pts with advanced FGFR1 or 2 amplified breast, squamous NSCLC, or stomach cancer, with results likely in 2H14.

Similar to '4547, NVS's BGJ398 has shown activity in Ph.I testing. The Ph.I dose escalation trial of this 2nd-generation FGFR1 inhibitor enrolled 10 pts with FGFR1-amplified mBC and three pts with FGFR1-amplified squamous NSCLC. One NSCLC pt responded to 100mg of BGJ398, with a 33% reduction in target lesions by eight weeks. Although no additional response rate data has been reported from this trial, we believe the drug's activity in squamous NSCLC is consistent with the activity of other 1st- and 2nd-generation FGFR1 inhibitors.

We do not view the 2nd-generation FGFR1 inhibitors as significant competitive threats to FP-1039. We believe AZD4547 and BGJ398 share some of the side effect limitations of older small molecule FGFR1 inhibitors. However, we believe the early clinical results for these drugs suggest FGFR1 inhibition has notable activity in squamous NSCLC, providing an important conceptual groundwork for FP-1039 in this indication.

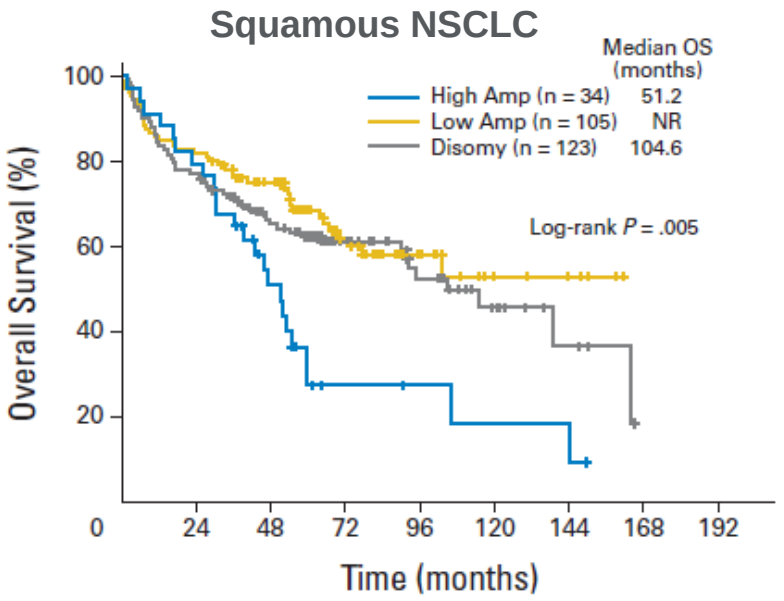


Source: Novartis Oncology

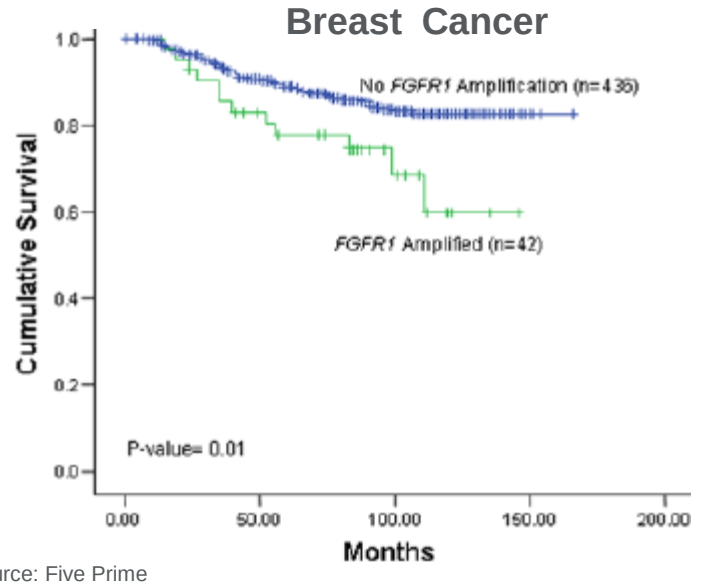
FPRX – FGFR1 Amplification Is a Negative Prognostic Factor

Published literature suggests FGFR1 amplification is a negative prognostic indicator in squamous NSCLC. In a retrospective analysis, Kim et al. showed that FGFR1 amplification was found to be an independent negative prognostic factor in surgically resected squamous NSCLC. 262 East Asian pts with squamous NSCLC were divided into groups based on FGFR1 amplification frequency, including high FGFR1 amplification (>9 gene copies; 13% of pts vs. 22% in a recent study in Caucasians) and low amplification pts with between 2 and 9 copies (40%). Although ethnicity was not deemed to be a differentiating factor in FGFR1 amplification frequency, smokers showed the highest frequency. Median OS was also significantly shorter for the high amplification group: 51.2 mos. vs. 115 mos. for the disomy group (2 copies). Additionally, median DFS was significantly shorter for the high amplification group: 26.9 months vs. 103.1 months for the low group. We believe these findings illustrate the unmet need for effective FGFR1-targeted therapies in squamous NSCLC.

We believe there is also evidence that FGFR1 amplification is associated with poor prognosis in mBC. In a cohort of 880 unselected mBC pts, a study by Elsheikh et al. showed that 8.7% had FGFR1 amplification, as defined as >5 gene copies. Notably, FGFR1 amplification was significantly more prevalent in tumors that lacked HER2 amplification and in pts >50 years of age. In the analysis, there was a significant association with FGFR1 amplification and shorter OS, indicating FGFR1 amplification as an independent negative prognostic in BC pts with estrogen-receptor-positive tumors. Based on this evidence, we believe FGFR1 amplified mBC may be an important future FP-1039 indication. Notably, NVS is already conducting a Ph.II trial of dovitinib in this setting.



Source: Five Prime



Source: Five Prime

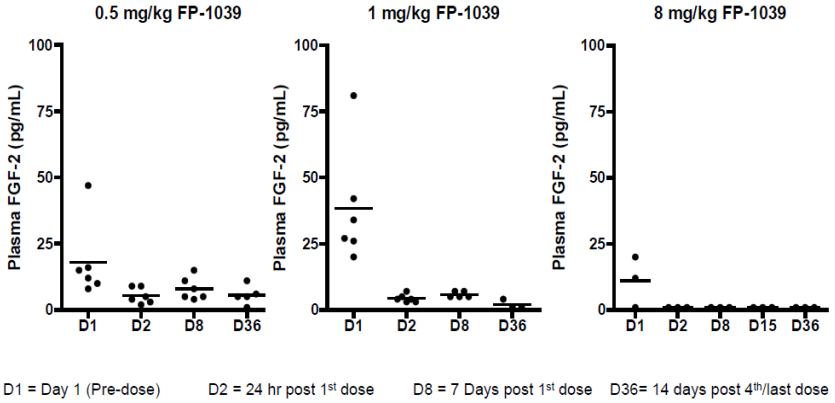
FPRX – Solid Ph.I Efficacy/Safety Results for FP-1039

Compelling FP-1039 side effect profile, with early hints of activity, in Ph.I solid tumor results. The Ph.I ascending dose trial of FP-1039 primarily assessed the drug's safety in IV administration 1x/wk over four weeks. The trial enrolled 39 unselected pts with advanced solid tumors, including BC, NSCLC, colorectal cancer, prostate cancer, HNC, and EC. Critically, FP-1039 was well tolerated and no MTD was defined. The AEs were primarily asymptomatic lab abnormalities, with no evidence of hyperphosphatemia/retinal toxicity, consistent with little/no inhibition of hormonal FGF signaling. Two DLTs were observed at 1mg/kg, including bowel perforation and neutropenia, but these side effects were deemed unrelated to the drug, as no additional DLTs were observed at doses as high as 16mg/kg.

Plasma levels of the key mitogenic FGF, FGF2, decreased significantly in all pts treated with FP-1039. FP-1039 sequesters FGF2, which in turn should decrease free FGF2 levels in the blood. All pts in the Ph.I trial had elevated FGF2 plasma levels at entry versus normal subjects, and FGF2 levels were measured at several time points over 36 days of FP-1039 dosing. Notably, the drug showed substantial dose-related reductions in FGF2, which we believe should be associated with clinical activity in future trials.

Early efficacy signals, despite unselected pt population. 17/39 pts (44%) achieved SD of varying durations, with one prostate cancer pt experiencing 20% tumor reduction, decreased PET positivity, and a ~seven months SD. Importantly, we do not view the absence of clinical responses in the Ph.I results as surprising, given the very gradual dose escalation, the broad range of tumor types, and lack of selection for FGFR1 amplification in the trial.

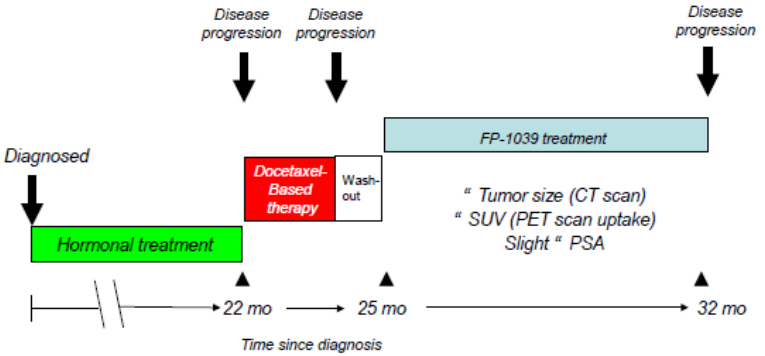
FGF2 Plasma Levels Post FP-1039 Therapy



D1 = Day 1 (Pre-dose) D2 = 24 hr post 1st dose D8 = 7 Days post 1st dose D36= 14 days post 4th/last dose

Source: Five Prime

Evidence of Clinical Activity of FP-1039



- Tumor shrinkage, 7 months of stable disease in prostate cancer patient on FP-1039 who had rapidly progressed on chemotherapy
- 16 additional patients had stable disease

Source: Five Prime

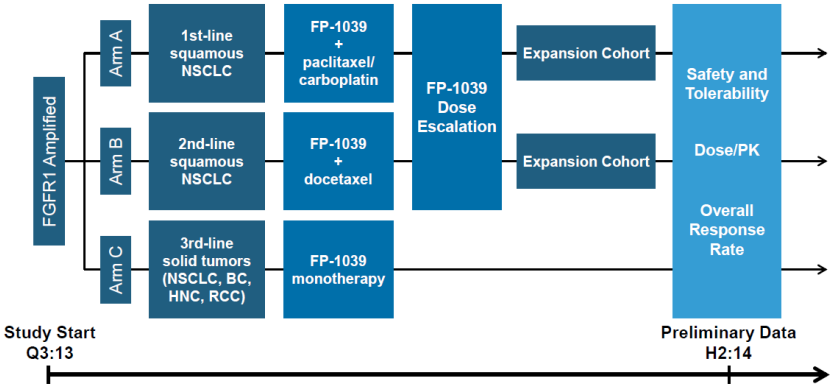
FPRX – Potential Fast Track to FP-1039 Approval in Squamous NSCLC and Multiple Other Solid Tumors Over Time

Ph.Ib trial focused on squamous NSCLC, but includes an exploratory arm in other FGFR1 amplified tumors. The recently initiated Ph.Ib trial consists of three arms testing FP-1039 in combination with carbotax/docetaxel in 1st- and 2nd-line squamous NSCLC and FP1039 monotherapy in 3rd-line solid tumors (NSCLC, mBC, HNC, and RCC). The trial is evaluating 70-104 pts over six months on safety, tolerability, dosing and ORR, with preliminary data expected 2H14. Importantly, this trial will only include pts with histologically or cytologically confirmed FGFR1+ amplification. Given the strong rationale for FP-1039 in these settings, and the relatively low efficacy bar set by established therapeutic approaches, we see a good probability that this trial will be successful.

FP-1039 has the potential for broad clinical activity. Although we do not include projections for FP-1039 in FGFR1+ tumors outside of squamous NSCLC in our FPRX valuation, we believe there is strong potential for the drug in mBC, HNC, and RCC, given the measurable prevalence of FGFR1 amplification in these cancers. FPRX and GSK have not finalized the future clinical plan for FP-1039 in these indications, but we believe initial results from the Ph.Ib trial will allow the companies to select one or more additional indications to pursue.

Ph.Ib Trial Design

Designed to assess preliminary efficacy in patients with *FGFR1* amplification (N=70-104)



Source: Five Prime

FP-1039 Potential for Broad Clinical Activity

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	

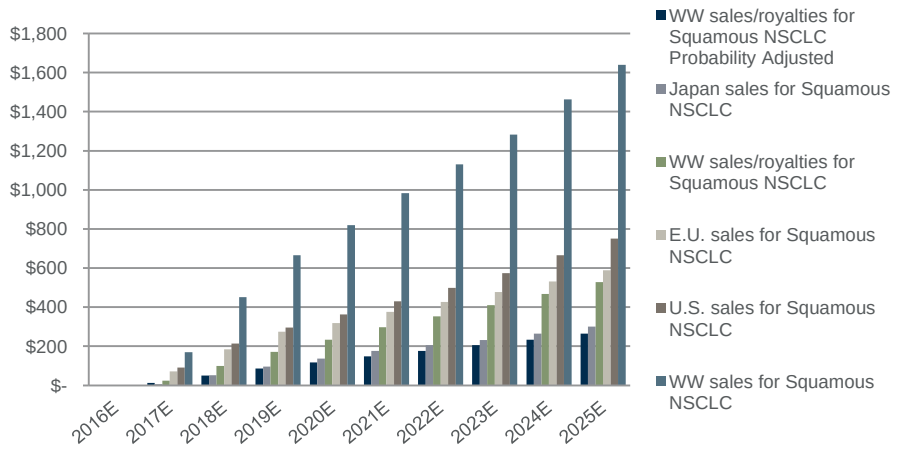
Source: Five Prime

FPRX – We Project >$1B Peak WW Sales of FP-1039 in FGFR1+ Squamous NSCLC

We believe FP-1039 can substantially exceed $1B in worldwide sales in FGFR1+ squamous NSCLC. NSCLC is currently the leading cause of cancer death in the world, with ~750,000 new cases expected in the United States, European Union, and Japan, combined, in '13. Squamous NSCLC represents ~30% of these cases, or ~225,000 pts. There is fairly conclusive scientific evidence that FGFR1 amplification occurs in 20–25% of squamous NSCLCs, defining a large, ~50,000 pt target pool for FG-1039 in the U.S./E.U./Japan.

Expecting strong penetration and pricing. We project ~50% penetration of FP-1039 in FGFR1 amplified squamous NSCLC at peak. We believe this level of penetration is consistent with established targeted oncology therapies, and we expect strong, ~$90,000/year pricing in the United States. We note that FPRX is entitled to a tiered, low-double digit to high-teen royalty on sales of FP-1039 in the U.S/E.U. from GSK and the company currently holds all Japanese rights to the drug. As FP-1039 is still relatively early stage, we believe applying a probability adjustment of 50% is the most appropriate method of estimating the drug's current value to FPRX. We note that our FP-1039 estimates are focused solely on NSCLC, which we view as conservative.

Projected FP-1039 Squamous NSCLC Sales/Royalties ($M)



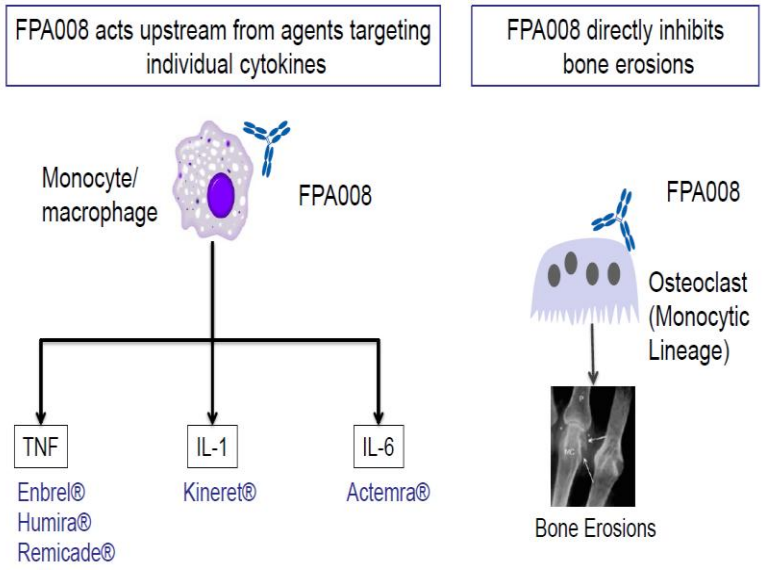
Source: Guggenheim Securities, LLC

FPRX – FPA008 Entering Ph.I Testing by YE'13; Differentiated Mechanism for Inflammatory Diseases

We believe FPA008 has the potential to improve upon the high efficacy of current treatments for inflammatory diseases, such as RA. FPRX's next candidate to enter the clinic, FPA008, is a mAb that blocks CSF1R and inhibits activation by CSF1 and IL-34, preventing activation of inflammatory monocytes and macrophages. Prior to FPRX discovering IL-34, it was thought that CSF1 was the key ligand for CSF1R, but the company's published work strongly suggests IL-34 is critical for CSF1R's pro-inflammatory activity. A number of current therapies for RA/psoriasis target individual inflammatory cytokines, such as TNF (Enbrel, Humira, and Remicade) and IL-6 (Actemra). In contrast, by targeting CSF1R, FPA008 should reduce the production of multiple inflammatory cytokines by monocytes/macrophages, potentially leading to greater efficacy.

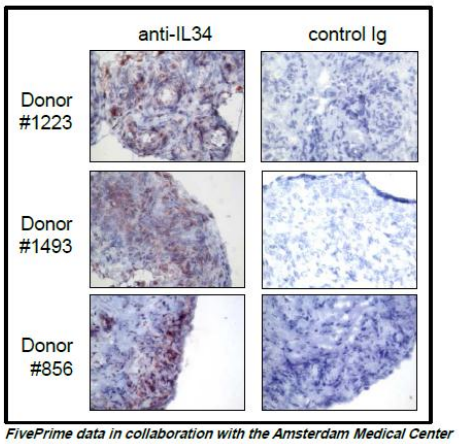
FPA008 has shown to be effective in preventing inflammation and bone destruction in preclinical models, with no concerning safety issues. Biopsy samples from inflamed joints incubated with FPA008 ex vivo showed reduced levels of TNF alpha, IL-6, and IL-1beta, compared with samples incubated with a control antibody. In addition, FPA008 was superior in reducing joint swelling, inflammation, and bone damage versus Enbrel in rodent RA models. Importantly, although FPA008 has mechanistic similarities to the anti-TNFs, the drug showed no evidence of immunosuppression or opportunistic infections in primates treated for extended periods.

By YE'13, FPRX will begin Ph.I testing of FPA008 in healthy volunteers, with initial results expected late '14. Based on the current lack of clinical safety data on FPA008, we view the first human safety results for FPA008 as a critical event for FPRX, prior to transitioning specifically to RA pts in a subsequent Ph.II trial.

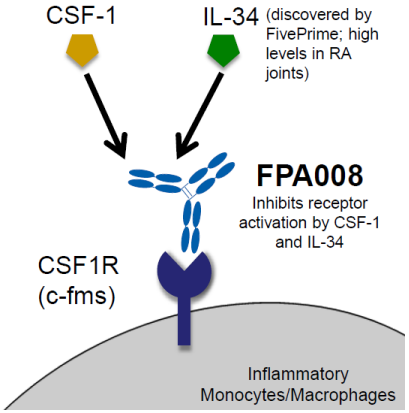


Source: Five Prime

IL-34 is Expressed in RA Patient Synovium



Source: Five Prime



Source: Five Prime

FPRX – FPA008 Positioned to Tap into Large Market for RA Therapies

FPA008 has considerable market potential in RA, even if the drug is reserved for anti-TNF failures. The anti-TNFs, notably Enbrel and Humira, are very entrenched in RA, and we believe it will remain difficult for new therapies to supplant use of these drugs for many years to come. Appropriately, FPRX will initially focus development of FPA008 in RA pts that have failed anti-TNF treatment. However, we note that this is still a very large, ~50,000–200,000 U.S./E.U. pt pool, and we would expect sales of FPA008 to approach or exceed blockbuster status in this setting. The RA market size is over $25B based on worldwide sales of the top three RA biologics—Humira, Remicade, and Enbrel.

FPA008 may be competitive vs. other CSF1R-targeted approaches. Currently, there are no approved agents that inhibit monocytic-lineage cells by blocking IL-34 and/or CSF-1. Although large-cap companies like AMGN and Roche are conducting Ph.I trials in anti-CSF1R mAbs, these companies are focusing on oncology as the initial indication for these candidates, not on inflammatory diseases. Further, in preclinical testing, FPA008 has shown limited overall monocyte depleting effects, which would be a liability for the anti-CSF1R mAbs in development for cancer. In addition, FPA008 is administered subcutaneously versus intravenously in other anti-CSF1R antibodies. Furthermore, as a mAb, FPA008 is more selective than CSF1R small molecules in development, including J&J's candidate for RA and Hodgkin's disease. Of course, these large pharmaceutical companies could eventually decide to pursue inflammatory diseases with their candidates, but we believe FPA008 is likely to remain ahead of these candidates in development.

Competition Around Macrophage Inhibition

Approach	Stage	Disease Setting	FPA008 Advantages
Anti-CSF1R Antibodies			
AMG820 (Amgen)	Phase 1	Cancer	FPA008 antibody and formulations designed for chronic autoimmune diseases: non depleting, subcutaneous formulation
IMC-CS4 (Imclone)	Phase 1	Cancer	
RG-7155 (Roche)	Phase 1	Cancer	
CSF1R Small Molecules			
PLX5622 (Daiichi)	Phase 1	Cancer, RA	FPA008 more selective than small molecules
ARRY-382 (Array)	Phase 1	Cancer	
JNJ-40346527(J&J)	Phase 1	Hodgkin's, RA	
Anti-GMCSF(R) Antibodies			
Mavrilimumab (Medimmune)	Phase 2	RA	GM-CSF but not CSF1 deficiency associated with alveolar proteinosis and cryptococcal meningitis FPA008 does not target neutrophils
MOR-103 (GSK)	Phase 2	RA, MS	
KB003 (Kalobios)	Phase 2	Asthma	

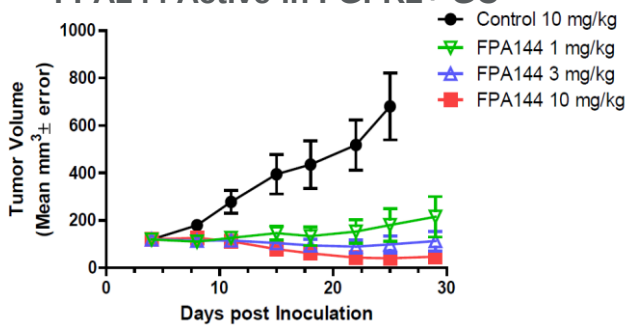
Source: Five Prime

FPRX – FPA144 Could Be a Significant Advance for FGFR2+ Gastric Cancer

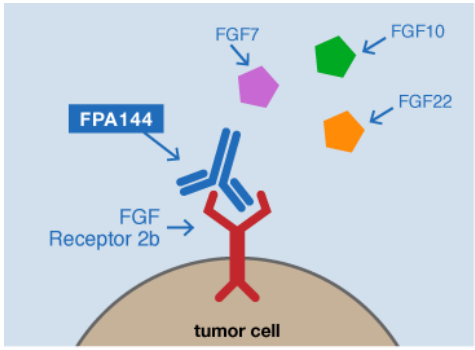
FPA144 is a differentiated, wholly owned, early stage mAb for FGFR2+ gastric cancer (GC). FPA144 binds to FGFR2b proteins on the surface of tumor cells and engages the immune system in a process called antibody-dependent cell-mediated cytotoxicity, or ADCC. FPA144 has been found to be effective in preclinical models of FGFR2+ GC, both through inhibition of FGFR2 signaling and direct cell killing. It is currently differentiated from other approaches in that FPA144 engages ADCC and has not been shown to cause hyperphosphatemia or toxicities related to VEGFR inhibition.

FPA144 targets an ultra-orphan indication with the potential for fast track development timelines. The FGFR2 gene is amplified in a small subset of gastric cancers (~5%) and is associated with significantly poorer prognosis, which may justify an ultraorphan and fast track designation. FPRX plans to initiate a Ph.I FPA144 trial in 2H14 in the United States and Asia, with interim data expected by YE'15. We currently do not include FPA144 upside in our valuation. However, based on the drug's strong biological rationale in FGFR2+ gastric cancer and potentially in esophageal, breast, and ovarian cancers, we believe FPA144 could be a LT value driver for FPRX. Notably, FPRX maintains worldwide rights to the program.

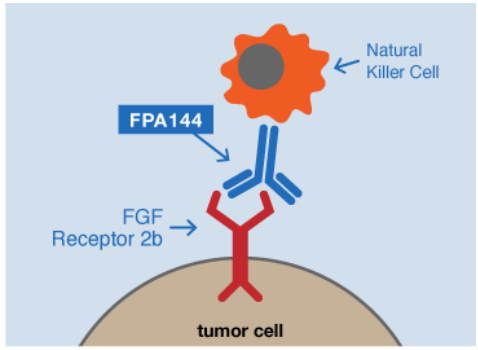
FPA144 Active in FGFR2+ GC



Source: Five Prime



Blocks ligand binding



Induces cell killing by ADCC

Source: Five Prime

Competition Around FGFR2 Inhibition

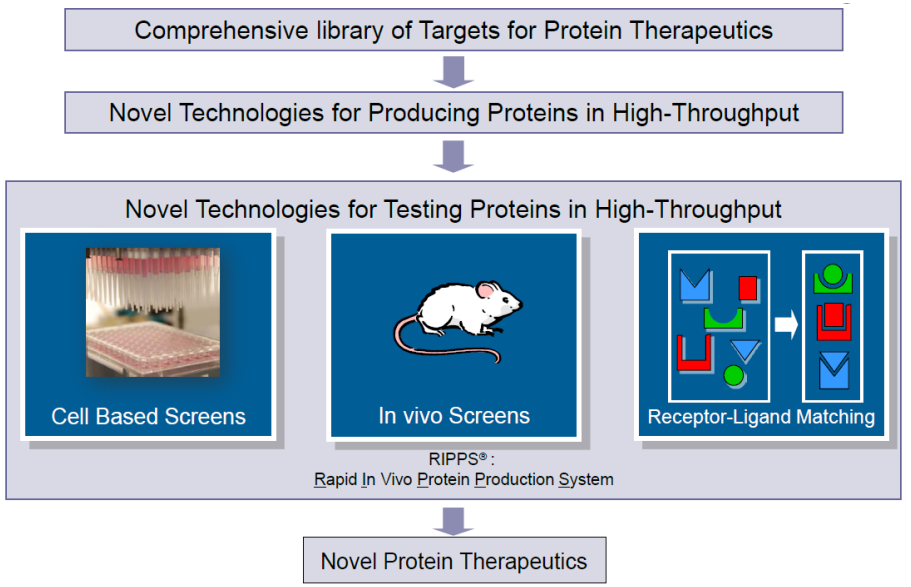
Approach	Stage	Comments	FPA144 Advantages
Selective FGFR TKI AZD-4547, (Astra Zeneca)	Phase 2	Monotherapy vs paclitaxel in Patients with Gastric Cancer with *FGFR2* Gene Amplification	No hyperphosphatemia
BGJ-398 (Novartis); LY-2874455 (Eli Lilly); JNJ42756793 (Astex/J&J)	Phase 1	No development in Gastric Cancer reported	No hyperphosphatemia
Non-selective TKI Dovitinib /TKI258 (Novartis)	Broad program	Single arm Ph 2 study in Gastric Cancer in Japan; no patient selection	No toxicities related to VEGFR inhibition
Anti-FGFR2b mAb GP369 (AVEO)	Preclinical		•Direct tumor killing (ADCC) •More advanced program

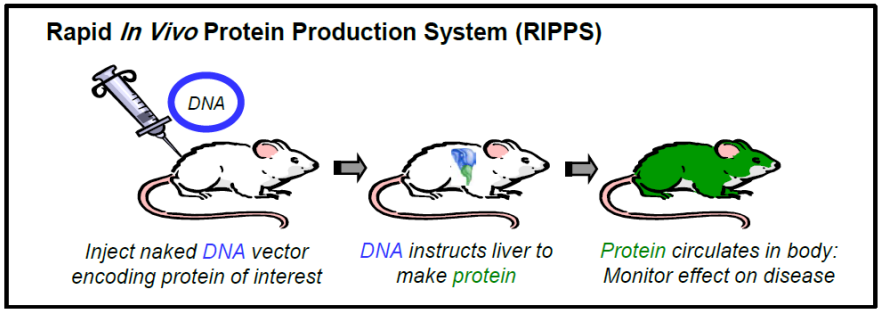
Source: Five Prime

FPRX – Protein Therapeutic Discovery Platform

FPRX's core technology has the potential to substantially accelerate the discovery of novel protein therapeutics. Of the >5,600 potential targets for protein therapeutics, only ~30 account for all marketed protein drugs in cancer and inflammation. The company's discovery platform consists of two key components: 1) a proprietary library of >5,600 secreted proteins representing a significant source of novel drug targets; and 2) proprietary technologies for high-throughput production and testing of proteins in animal disease models (RIPPS). We believe FPRX's platform is differentiated based on both the comprehensiveness of the library and the speed at which the company can conduct protein drug discovery. We believe the company's progress with FP-1039, FPA008, and FPA144, along with additional preclinical programs in cancer immunotherapy and other disease settings, provides strong evidence of the productivity of this platform.

FPRX's platform has been the foundation for several major collaboration deals, and we believe this deal flow is likely to continue. The company has generated more than $220M of non-dilutive funding through three major collaborations with GSK, including, notably, development of FP-1039. Additionally, FPRX has successfully completed several other collaborations with large pharmaceutical companies, including UCB, Inc., which have enabled the company to operate without significant outside financing since 2005. We expect the company to announce a new collaboration in the 4Q13–2Q14 timeframe.





Source: Five Prime

FPRX – D.C. Scorecard

Overall Score: Neutral (3.2 Average)

SUBCATEGORY & SCORE	COMMENTS	
FDA Standing: **Neutral- 3**	• As a pre-commercial company, we believe FPRX has yet to build a solid track record with the FDA. With currently only one clinical-stage program, we believe it is likely the company has yet to build a reputation with the Agency. • **Notably, we believe the FDA is continuing to devote meaningful time and attention on first-time sponsors, which we believe has the potential to benefit an emerging oncology company like FPRX.**	• **We believe the FDA is open to accelerated approval of targeted cancer therapies, provided there are well-defined pt populations that benefit from these drugs. As FP-1039 is targeting molecularly defined subpopulations in squamous NSCLC and other tumors, we believe the drug would likely have favorable positioning with the FDA's ODAC.**
Medicare/Medicaid Reimbursement: **Neutral- 3**	• We expect that FPRX's current clinical candidates would be reimbursed under Part B, which we believe is somewhat favorable for oncology drugs compared to Part D.	
ACA Impact: **Net Positive- 4**	• **We believe the ACA will likely bring a number of new pts under coverage who are not yet Medicare eligible, which should benefit FPRX from a commercial perspective.**	
Biosimilars: **Neutral- 3**	• We do not believe biosimilar competition is a NT risk for FPRX, given the company's early stage of development. In the very LT, however, given the company's sole focus on protein therapeutics, the company may face biosimilar competition.	• **In the United States, FP-1039 is covered by a composition of matter patent expiring in 2026 and a method of use patent expiring in 2031. In Europe, FP-1039 is also covered by composition of matter and method of use patents, both expiring in 2026.**
D.C. Presence: **Neutral- 3**	• To our knowledge, FPRX does not have any significant D.C. presence, which we believe is appropriate for an early stage company.	

FPRX – Income Statement 2012–2020E

Five Prime Therapeutics Inc.

Amounts in thousands, except per-share figures	2012A	2013E	2014E	2015E	2016E	2017E	2018E	2019E	2020E
Income Statement									
Revenues:									
Collaboration Revenue	9,983	20,104	22,300	40,640	28,454	55,200	51,400	64,250	73,888
FP-1039 Revenue (2)	-	-	-	-	-	12,206	50,047	85,627	116,795
Total operating revenue	**9,983**	**20,104**	**22,300**	**40,640**	**28,454**	**67,406**	**101,447**	**149,877**	**190,683**
Operating expenses:									
Cost of goods	-	-	-	-	-	-	-	-	-
Research & development	28,778	31,125	34,238	37,148	41,234	43,914	46,110	48,415	50,836
Selling, general & administrative	9,009	13,818	14,509	15,597	17,937	19,551	20,724	21,967	23,285
Total operating expenses	**37,787**	**44,943**	**48,746**	**52,745**	**59,171**	**63,465**	**66,834**	**70,383**	**74,122**
Income (Loss) from operations	**(27,804)**	**(24,839)**	**(26,446)**	**(12,105)**	**(30,717)**	**3,941**	**34,613**	**79,494**	**116,561**
Other income (expense)	209	868	802	715	755	503	596	1,470	3,163
Pretax income (loss)	**(27,595)**	**(23,971)**	**(25,644)**	**(11,390)**	**(29,962)**	**4,443**	**35,209**	**80,964**	**119,725**
Income tax provision (benefit)	-	-	-	-	-	-	-	-	-
Net income (loss)	**(27,595)**	**(23,971)**	**(25,644)**	**(11,390)**	**(29,962)**	**4,443**	**35,209**	**80,964**	**119,725**
Basic & diluted net loss per share	**($1.87)**	**($1.34)**	**($1.00)**	**($0.40)**	**($0.98)**	**$0.14**	**$1.10**	**$2.44**	**$3.45**
Basic & diluted common shares outstanding (1)	14,724	17,895	25,600	28,450	30,618	31,268	32,058	33,178	34,728

(1) Reflects conversion of preferred stock to common stock

(2) Probability adjusted estimates

Source: Company documents and Guggenheim Securities, LLC

FPRX – Balance Sheet 2012–2020E

Five Prime Therapeutics Inc.

Amounts in thousands, except per-share figures	2012A	2013E	2014E	2015E	2016E	2017E	2018E	2019E	2020E
Balance Sheet									
Current assets:									
Cash and cash equivalents	11,391	73,880	45,383	53,221	38,789	25,688	49,164	121,941	235,554
Short-term investments	27,021	-	-	-	-	-	-	-	-
Prepaid expenses and other current assets	689	689	689	689	689	689	689	689	689
Total current assets	**39,101**	**74,569**	**46,072**	**53,910**	**39,478**	**26,377**	**49,853**	**122,630**	**236,243**
Property and equipment, net	4,631	27,920	18,014	58,414	91,500	117,690	138,108	155,012	169,873
Investments, net of current portion	-	-	-	-	-	-	-	-	-
Accounts receivable	-	-	-	-	1,423	3,370	5,072	7,494	9,534
Other assets	359	-	-	-	-	-	-	-	-
Restricted cash	-	86	86	86	86	86	86	86	86
Total assets	**44,091**	**102,575**	**64,172**	**112,411**	**132,486**	**147,524**	**193,119**	**285,221**	**415,736**
Current liabilities:									
Accounts payable	2,470	2,488	2,211	1,865	1,501	975	359	(326)	(1,085)
Accrued expenses	2,250	2,250	2,250	2,250	2,250	2,250	2,250	2,250	2,250
Payable to collaborative partner	-								
Other accrued liabilities	303								
Preferred stock warrant liability	563								
Deferred revenue, current portion	7,498								
Total current liabilities	**13,084**	**4,738**	**4,461**	**4,115**	**3,751**	**3,225**	**2,609**	**1,924**	**1,165**
Deferred revenue, long-term portion	7,258								
Deferred rent, long-term portion	2,448	45	39	38	38	40	38	38	39
Other long-term liabilities	897								
Liability for shares subject to repurchase	-	22	20	20	20	21	20	20	20
Obligation to issue warrant	-	-	-	-	-	-	-	-	-
Commitments:									
Series A convertible preferred stock, $0.001 par value	84,600								
Series A1 convertible preferred stock, $0.001 par value	11,000								
Series A2 convertible preferred stock, $0.001 par value	33,863								
Series A3 convertible preferred stock, $0.001 par value	6,819								
Stockholders' equity (deficit)									
Preferred stock, $0.0001 par value; 5,000 shares authorized; none issued									
Common stock, $0.0001 par value; 1.28M shares at 6/30/13	1	2	3	3	3	3	3	3	3
Additional paid-in capital	6,816	90,787	116,431	167,821	237,783	233,340	198,131	117,167	(2,557)
Accumulated other comprehensive income (loss)	7								
Deficit accumulated during the development stage	(122,702)								
Other	-	6,981	(56,782)	(59,587)	(109,108)	(89,105)	(7,683)	166,068	417,066
Total stockholders' equity (deficit)	**(115,878)**	**97,770**	**59,652**	**108,238**	**128,678**	**144,238**	**190,452**	**283,239**	**414,512**
Total liabilities, redeemable convertible preferred stock and stockholders' equity (deficit)	**44,091**	**102,575**	**64,172**	**112,411**	**132,486**	**147,524**	**193,119**	**285,221**	**415,736**

Source: Company documents and Guggenheim Securities, LLC

FPRX – Cash Flows 2012–2020E

Five Prime Therapeutics Inc.

Amounts in thousands, except per-share figures	2012A	2013E	2014E	2015E	2016E	2017E	2018E	2019E	2020E
Statement of Cash Flows									
Operating activities									
Net (loss) income	(27,595)	(23,971)	(25,644)	(11,390)	(29,962)	4,443	35,209	80,964	119,725
Adjustments to reconcile net loss to net cash used in operating activities:									
Depreciation and amortization	1,643	1,643	9,906	6,391	20,724	32,463	41,754	48,998	54,995
(Gain) loss on disposal of property and equipment	(5)								
Stock-based compensation expense	1,721	1,134	1,348	1,462	1,582	1,775	1,904	2,005	2,111
Amortization of premium on marketable securities	538	-	-	-	-	-	-	-	-
Revaluation of preferred stock warrant liability	(119)	-	-	-	-	-	-	-	-
Receivable from collaborative partners	449	-	-	-	-	-	-	-	-
Other long-term assets	372								
Accounts payable	18	18	18	18	18	18	18	18	18
Accrued personel-related expenses	(19)	-	-	-	-	-	-	-	-
Payable to collaborative partner	(3,000)	18	(277)	(346)	(364)	(526)	(617)	(685)	(759)
Deferred revenue	7,379	7,379	7,379	7,379	7,379	7,379	7,379	7,379	7,379
Deferred rent	457								
Other accrued liabilities	(236)								
Net cash used in operating activities	(18,397)	(13,779)	(7,271)	3,515	(622)	45,552	85,647	138,679	183,470
Investing activities									
Purchases of marketable securities	(45,419)	(24,932)	(43,527)	(46,791)	(53,810)	(58,653)	(62,172)	(65,902)	(69,856)
Maturities of marketable securities	64,636	41,200	22,300	11,115	-	-	-	-	-
Purchases of property and equipment	(737)								
Restricted cash	38								
Proceeds from lease incentives	-	-	-	-	-	-	-	-	-
Proceeds from disposal of property and equipment	-	-	-	-	-	-	-	-	-
Net cash used in investing activities	18,518	16,268	(21,227)	(35,676)	(53,810)	(58,653)	(62,172)	(65,902)	(69,856)
Financing activities									
Proceeds from issuance of redeemable convertible preferred stock	6,819	-	-	-	-	-	-	-	-
Proceeds from issuance of common stock	105	-	-	-	-	-	-	-	-
Payments under capital lease obligation	(15)								
Net proceeds from issuance of common stock/restricted common stock	-	60,000		40,000	40,000		-	-	-
Net cash provided by financing activities	6,909	60,000	-	40,000	40,000	-	-	-	-
(Decrease) increase in cash and cash equivalents	7,030	62,489	(28,497)	7,838	(14,432)	(13,100)	23,476	72,777	113,614
Cash and cash equivalents at beginning of period	4,361	11,391	73,880	45,383	53,221	38,789	25,688	49,164	121,941
Cash and cash equivalents at end of period	**11,391**	**73,880**	**45,383**	**53,221**	**38,789**	**25,688**	**49,164**	**121,941**	**235,554**

Source: Company documents and Guggenheim Securities, LLC

Public Companies Mentioned in this Report

- Amgen (AMGN, NEUTRAL, $110.89)
- Array Biopharma (ARRY, NC, $5.53)
- Astex Pharmaceuticals- (ASTX, NC, $8.49)
- AstraZeneca (AZN, NC, $50.96)
- Aveo Pharmaceuticals (AVEO, NC, $2.12)
- Bayer (BAYZF, NC, $117.71)
- Daiichi Sankyo (DSNKY, NC, $18.22)
- Eli Lilly (LLY, NC, $48.88)
- GlaxoSmithKline (GSK, NC, $49.80)
- Johnson & Johnson (JNJ, NC, $89.45)
- KaloBios (KBIO, NC, $4.49)
- Novartis (NVS, NC, $74.95)
- Roche (RHHBY, NC, $65.74)

ANALYST CERTIFICATION

By issuing this research report, each Guggenheim Securities, LLC ("Guggenheim Securities") research analyst whose name appears in this report hereby certifies that (i) all of the views expressed in this report accurately reflect the research analyst's personal views about any and all of the subject securities or issuers discussed herein and (ii) no part of the research analyst's compensation was, is, or will be directly or indirectly related to the specific recommendations or views expressed by the research analyst.

IMPORTANT DISCLOSURES

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Guggenheim Securities, LLC managed or co-managed a public offering of securities for Five Prime Therapeutics, Inc. during the past 12 months.

Five Prime Therapeutics, Inc. is or was during the 12-month period preceding the date of the distribution of this report a client of Guggenheim Securities. Guggenheim Securities, LLC Provided Five Prime Therapeutics, Inc.investment banking services.

Guggenheim Securities, LLC or its affiliates expect(s) to receive or intend(s) to seek compensation for investment banking services from Five Prime Therapeutics, Inc. and Amgen Inc. in the next 3 months.

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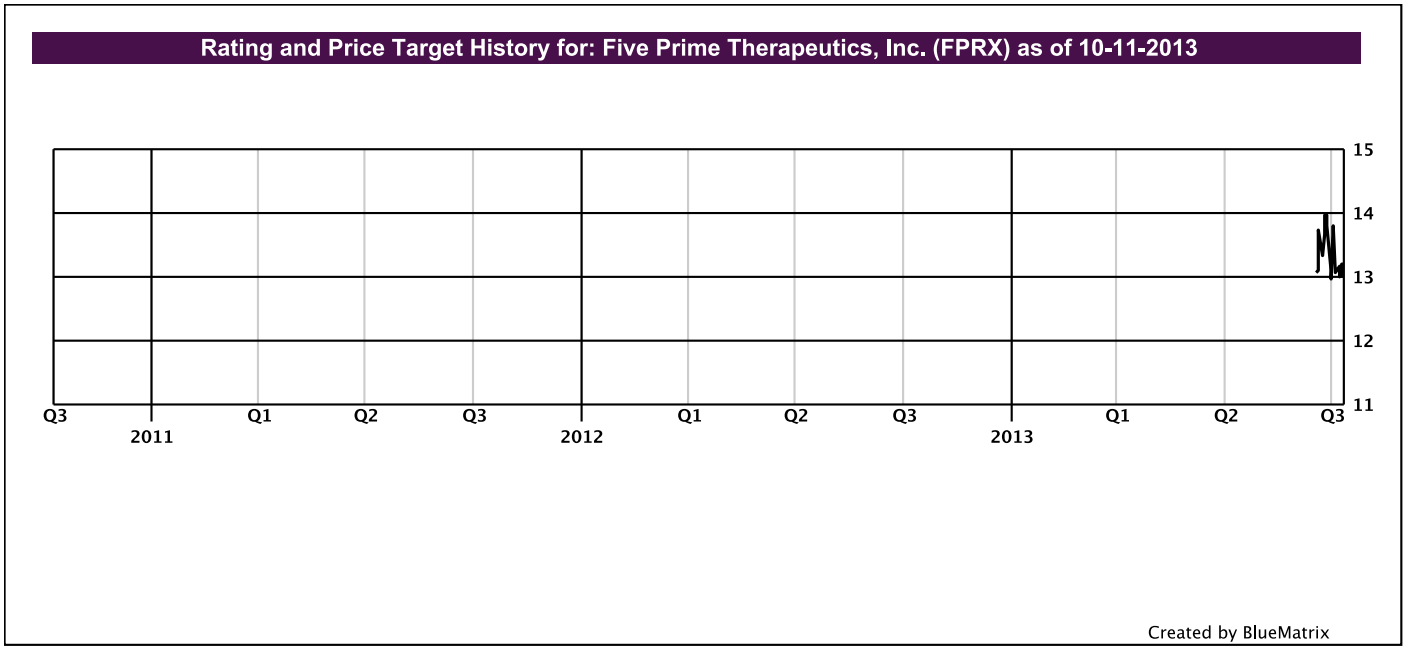

RATING DEFINITIONS

BUY (B) - Describes stocks that we expect to provide a total return (price appreciation plus yield) of 15% or more within a 12-month period.
NEUTRAL (N) - Describes stocks that we expect to provide a total return (price appreciation plus yield) of plus 15% or minus 15% within a 12-month period.
SELL (S) - Describes stocks that we expect to provide a total negative return (price appreciation plus yield) of 15% or more within a 12-month period.
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			IB Serv./ Past 12Mos.	
Rating Category	Count	Percent	Count	Percent
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Neutral	81	45.00%	6	7.41%
Sell	0	0.00%	0	0.00%

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