November 5, 2012

Intercept Pharmaceuticals

(ICPT-NASDAQ)

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

Biotechnology

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Rare Orphan Drug Opportunity in Liver Disease; Initiating Coverage at OUTPERFORM

Investment Thesis

We are initiating coverage of Intercept Pharmaceuticals (ICPT) with an **OUTPERFORM** rating and $31 price target. Our positive outlook is supported by a proprietary platform of bile acid analogs, a deep pipeline of clinical candidates, and a lead opportunity in Primary Biliary Cirrhosis (PBC), a rare orphan disease supporting a $500 million–$1 billion opportunity. With phase 3 data for lead bile acid analog OCA (INT-747) expected by mid-2013, phase 2 data in PBC is compelling, in our opinion, and should support a clear benefit in patients failing current standard bile acid therapy with ursodiol (URSO). We estimate a probability adjusted NPV for OCA in PBC alone at roughly $29/share assuming 60% likelihood of success. While the orphan disease opportunity in PBC alone should support upside from current levels, we believe that expansion opportunities for OCA into broader liver diseases like portal hypertension and NASH represents an incremental opportunity that should support a premium valuation.

Forecasts

We estimate a loss per share in 2012 of ($1.16), remaining stable in 2013 at ($1.17) and expanding to ($1.70) in 2014 on OCA pre-launch expense. We expect a narrowed loss to ($0.04) in 2015 on OCA commercialization and first profitability in 2016 at $2.01. We estimate 2017 and 2018 EPS of $3.27 and $4.58, with a 22% three-year EPS CAGR estimated beyond 2018.

Valuation

Our $31 price target is based on 20x our 2018E EPS of $4.58 discounted 30%. We believe that the 20x multiple is in line with current biotech growth companies and supported by a 20%-plus EPS CAGR beyond 2018. We believe that the 30% discount rate adequately reflects risk to our estimates.

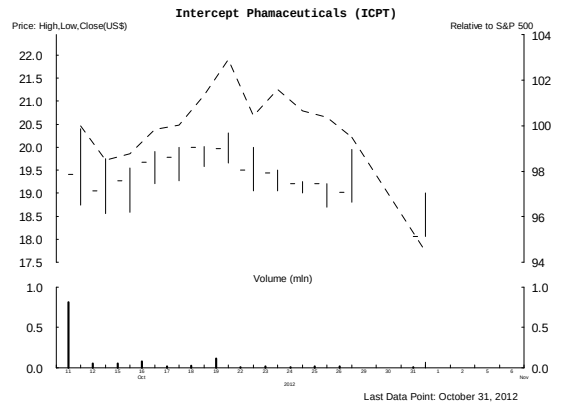
Recommendation

We rate Intercept Pharmaceuticals **OUTPERFORM**.

Securities Info

Price (2-Nov)	**$18.75**	**Target Price**	**$31**
52-Wk High/Low	$20/$18	Dividend	--
Mkt Cap (mm)	$360	Yield	--
Shs O/S (mm, BASIC)	19.2	Float O/S (mm)	6.0
Options O/S (mm)	na	ADVol (30-day, 000s)	82

Price Performance



Valuation/Financial Data

(FY-Dec.)	2010A	2011A	2012E	2013E
EPS GAAP	-$1.33	-$1.17	-$1.16	-$1.17
P/E			nm	nm
First Call Cons.				
FCF	NA	NA	NA	-$1.14
P/FCF			na	nm
EBITDA ($mm)	-$16	-$14	-$16	-$23
EV/EBITDA			nm	nm
Rev. ($mm)	$0	$2	$8	$13
EV/Rev			34.7x	21.3x

Quarterly EPS	**1Q**	**2Q**	**3Q**	**4Q**
2011A	NA	NA	NA	NA
2012E	NA	NA	-$0.26	-$0.21

Balance Sheet Data (na**)**

Net Debt ($mm)	-$79	TotalDebt/EBITDA	nm
Total Debt ($mm)	$0	EBITDA/IntExp	na
Net Debt/Cap.	nm	Price/Book	4.1x

Notes: All values in US$.

Source: BMO Capital Markets estimates, Bloomberg, Thomson Reuters, and IHS Global Insight.

Please refer to pages 39 to 44 for Important Disclosures, including the Analyst's Certification.

Investment Thesis

We believe that Intercept (ICPT) will emerge over the next 12 months as the next orphan drug stock success, with positive phase 3 data anticipated in mid-2013 for its bile acid analogue obeticholic acid (OCA) in patients with primary biliary cirrhosis (PBC). With orphan drug companies, focused on rare disease, commanding premium valuations relative to peers, and with multiples of forward revenues approaching 12x we believe that upside potential for ICPT is substantial, with wholly owned rights to what could be a $500 million–$1 billion opportunity in treatment refractory PBC.

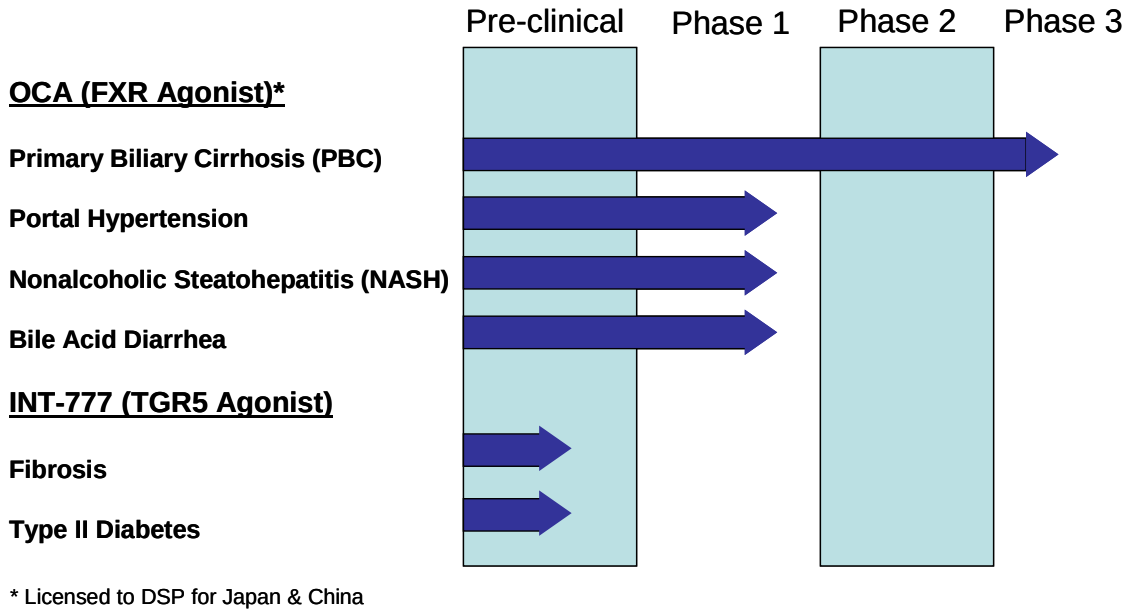
Review of two separate phase 2 studies for OCA in patients with PBC suggests a statistically significant and clinically relevant effect on several key surrogates of clinical benefit including Alkaline Phosphatase (ALP), and importantly on a composite responder index being used in phase 3 and predictive of reduced disease progression to cirrhosis, transplant, and death. Formal scientific advice from European regulators suggests that the surrogate endpoint in phase 3 will be accepted as a basis of approval and while this will be an item for FDA panel review, we believe that scientific evidence and thought leader support for the phase 3 endpoint is compelling.

While ICPT will inevitably be valued on its lead orphan disease opportunity in PBC, we would highlight development of OCA in broader chronic liver diseases like portal hypertension, non-alcoholic steatohepatitis (NASH) and bile acid induced diarrhea as providing substantial option value for ICPT shareholders. In addition, with a potential critical role for bile acid receptor modulation at the intersection of fatty liver disease and the epidemic of metabolic syndrome in patients with obesity and diabetes we believe that ICPT's leading position in bile acid chemistry and bile acid receptor targeting could support multiple blockbuster opportunities ahead.

Overview

Intercept is a development-stage biotechnology company focused on development of novel therapeutics to treat chronic liver disease. With proprietary expertise in bile acid chemistry, Intercept is developing several bile acid analogs for both rare orphan liver disease as well as more prevalent liver diseases. Intercept's current pipeline is summarized in Exhibit 1.

Exhibit 1: Intercept Pharmaceuticals Pipeline

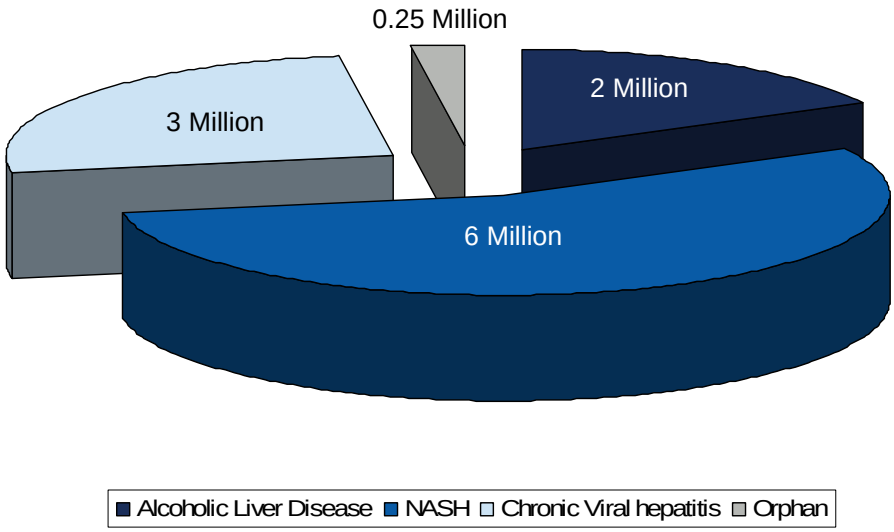


Source: Intercept Pharmaceuticals and BMO Capital Markets.

The lead bile acid analog for Intercept obeticholic acid (OCA) is in phase 3 development for patients with primary biliary cirrhosis, a rare orphan disease affecting roughly 60,000 patients in developed markets. Intercept is also developing OCA in phase 2 for more prevalent chronic liver diseases, including portal hypertension, non-alcoholic steatohepatitis (NASH), and bile acid induced diarrhea.

The prevalence of liver disease in the US is summarized in Exhibit 2. NASH is the most common liver disease in the US, affecting ~ 6 million individuals. Chronic viral hepatitis B or C affects a further 3 million individuals and alcoholic-related liver disease another 2 million. Around one quarter of a million individuals are affected by a cluster of autoimmune and other orphan diseases including primary biliary cirrhosis.

Exhibit 2: Prevalence of Liver Disease in the US



Orphan
Autoimmune – Primary Biliary Cirrhosis, Autoimmune Hepatitis, Primary Sclerosing Cholangitis
Other orphan liver disease – Hemochromatosis. Wilson's Disease, Sarcoidosis etc.

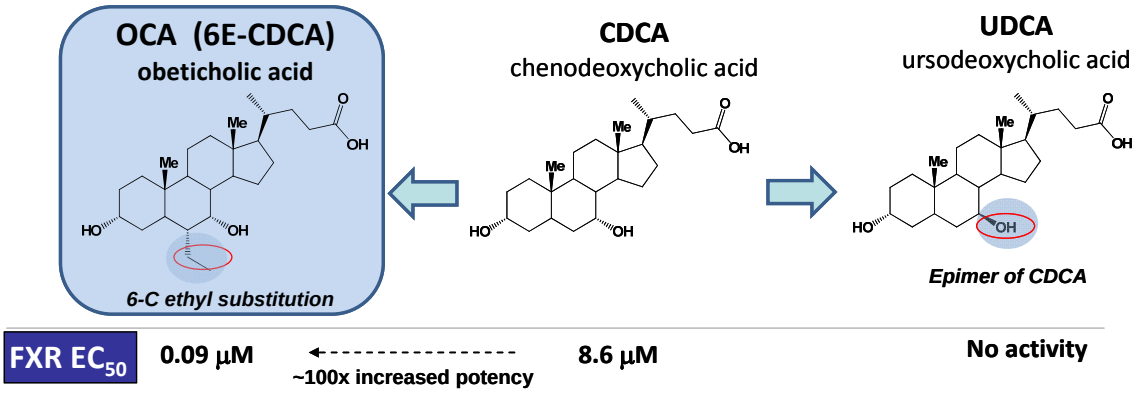
Source: Intercept Pharmaceuticals and BMO Capital Markets.

Intercept's primary discovery underlying its product pipeline and therapeutic platform is that bile acid analogs stimulate the farnesoid receptor (FXR) in both the liver as well as other organs. As a result Intercept is leveraging its expertise in bile acid chemistry and enhanced understanding of the FXR to develop next generation bile acid analogs for broader diseases, including type 2 diabetes and metabolic syndrome.

Primer on OCA and Bile Acid Analogs

Obeticholic acid (OCA) is a bile acid analog that selectively binds to the farnesoid X receptor (FXR) in the liver and induces several biologic effects that appear liver-protective. Obeticholic acid (OCA), or 6-alpha Chenodeoxycholic acid, is an analog of the natural occurring bile acid chenodeoxyxcholic acid (CDCA) and is specifically designed to potently stimulate the FXR. With an FXR EC50 of 0.099uM, OCA is ~100x more potent than naturally occurring CDCA, which has an EC50 of only 8.66uM, and much more potent than ursodeoxycholic, which is used therapeutically for primary biliary cirrhosis (PBC) but which has no activity on FXR. Preclinical studies have demonstrated biologic effects consistent with FXR stimulation, including induction of FXR genes. Importantly, OCA has no documented effect on other nuclear receptors and does stimulate other bile acid receptors to the same degree, including TGR5 where it has an EC50 of 20uM.

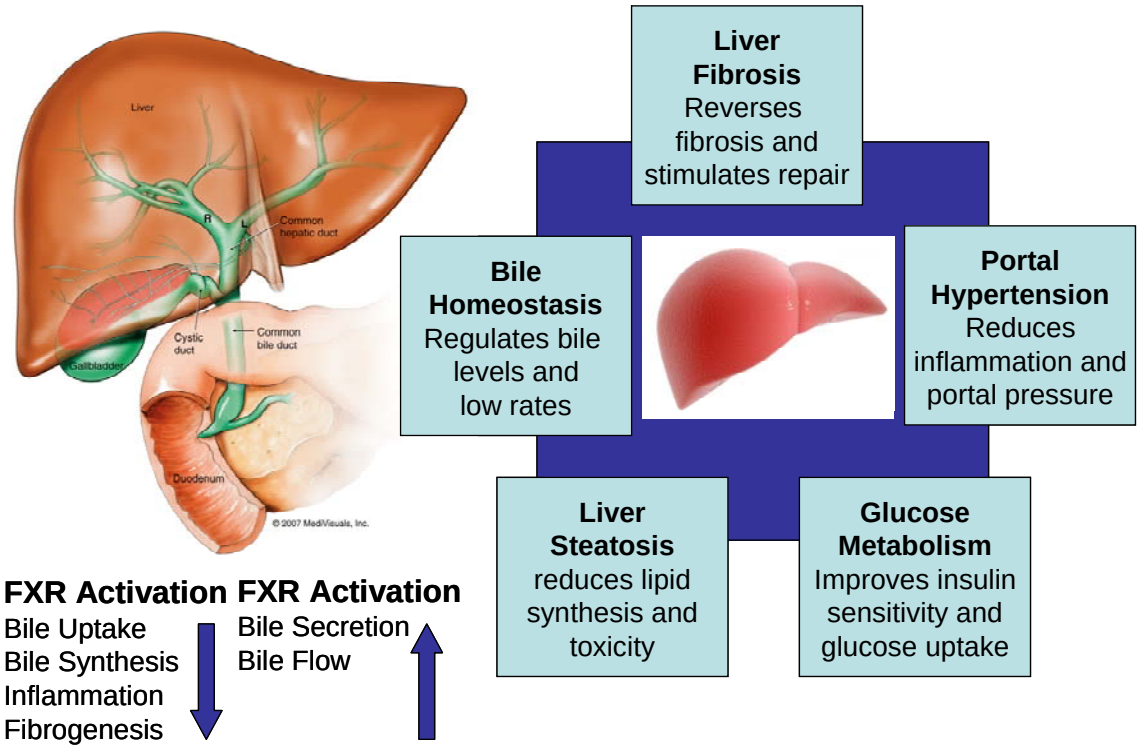
Exhibit 3- Chemical Structure of OCA and Related Compounds



Source: Intercept Pharmaceuticals and BMO Capital Markets.

Bile acids are regularly produced in the liver and act as natural detergents to emulsify and enhance absorption of fats from the small intestine. Bile acid production and clearance from the liver is regulated by dedicated receptors like FXR. Bile acid activation of FXR is also thought to induce anti-fibrotic and anti-inflammatory effects necessary for normal liver function. Bile acids also stimulate FXR in other organs beyond the liver, with potential therapeutic effects in intestinal disease and kidney disease.

Exhibit 4: Consequences of Farnesoid X Receptor (FXR) activation

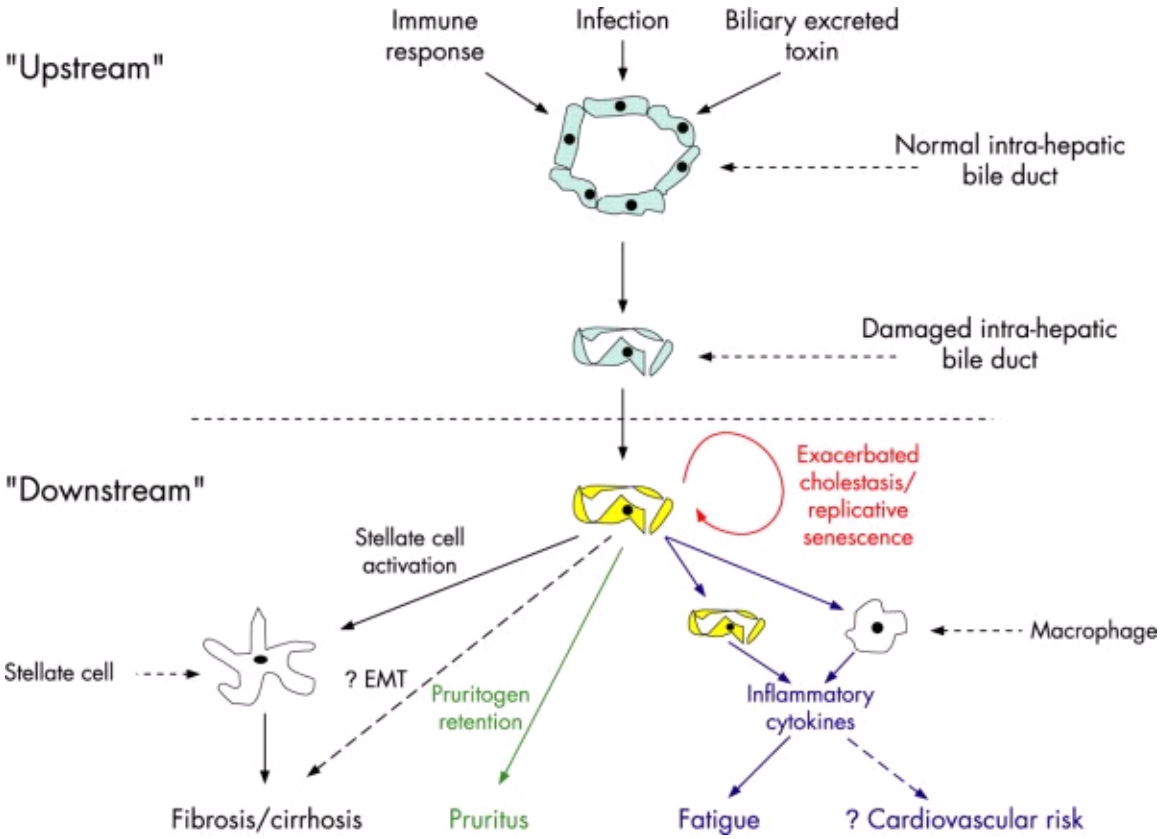


Source: Intercept Pharmaceuticals and BMO Capital Markets.

Primary biliary cirrhosis (PBC) is a rare liver disease involving the autoimmune destruction of bile ducts responsible for transporting bile acids out of the liver. Progressive destruction of bile ducts results in a build-up of bile acids in the liver, with natural detergent-like activity of the bile acids then turned against healthy liver cells. Bile acid mediated toxicity in the liver results in chronic inflammation and progressive fibrosis whereby damaged liver cells release alkaline phosphatase (ALP), a key marker of PBC detectable in blood. As PBC progresses, increased levels of ALP are observed along with impaired liver function and increases in bilirubin levels.

At present the only approved agent for the treatment of patients with PBC is ursodeoxycholic acid, which is available under the brand name URSO and as generic ursodiol. Ursodiol is a naturally occurring bile acid that represents a small fraction of the bile pool and is the least detergent of the bile acids in humans. Treatment with ursodiol is designed to dilute more detergent bile acids in the liver and thereby blunt the toxic effects on healthy liver cells. Ursodiol has no known pharmacologic effect, does not stimulate FXR and published data suggest that ~50% of patients have inadequate response to treatment. Compliance with ursodiol has also been described as challenging given the requirement of a gram of drug in divided daily doses.

Exhibit 5: Consequences of Bile Acid mediated Toxicity

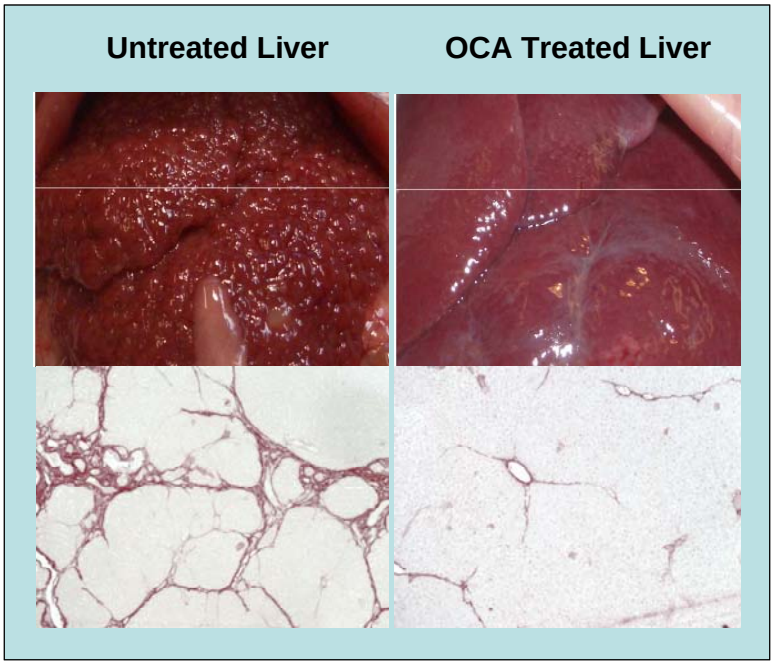


Source: Google Images and BMO Capital Markets.

It should be noted that primary biliary cirrhosis (PBC) is associated with a number of other autoimmune disorders including CRST syndrome of calcinosis, Raynaud's, sclerodactyly and telangiectasia, the sicca syndrome of dry eyes and dry mouth, autoimmune thyroiditis, and renal tubular acidosis. An underlying autoimmune cause is also supported by the fact that 95% of patients have elevated IgG anti-mitochondrial antibodies that aren't observed in other forms of chronic liver disease and cirrhosis.

Preclinical data for OCA suggest a profound benefit at reversing histopathologic features of PBC and reversing inflammatory and fibrotic changes associated with progression of the disease and development of cirrhosis. In particular, across multiple animal models, changes have been seen in alpha-1 collagen, alpha-SMA, TGF-B, MMP-2, TIMP-1 and TIMP-2. These anti-fibrotic changes have been associated with reduced fibrosis, reversal of cirrhosis, and reductions in portal vein pressure.

Exhibit 6: The Effects of OCA Treatment on Gross Liver Pathology Associated with PBC



Source: Intercept Pharmaceuticals and BMO Capital Markets.

OCA for Primary Biliary Cirrhosis

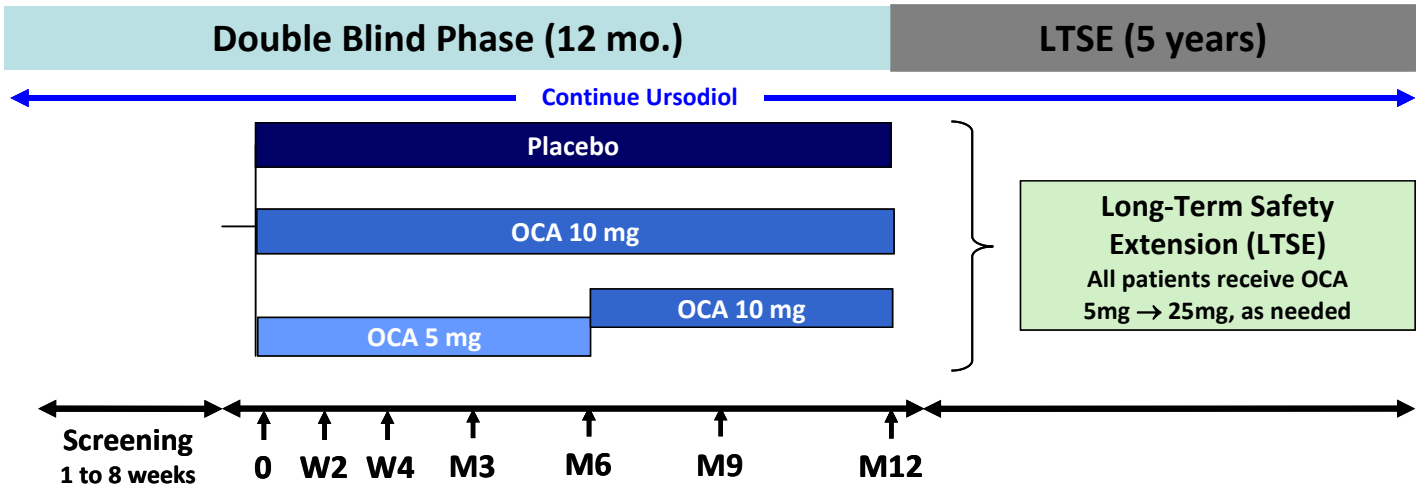
Intercept is currently conducting a phase 3 trial of OCA in patients with primary biliary cirrhosis (PBC) that have inadequate response to urso or who are urso intolerant. The trial is expected to report data by mid-2014 and is supported by two prior phase 2 studies suggesting statistically significant reductions in alkaline phosphatase (ALP) and other secondary endpoints. The primary endpoint of the phase 3 POISE study involves a responder index involving ALP

and bilirubin levels and is considered a surrogate endpoint with regulators. The use of a surrogate endpoint has been supported by the European Medicines Agency (EMA) and Intercept is in the process of confirming the value of the surrogate for purposes of predicting risk of liver failure and death for FDA. If successful in gaining approval for OCA in major markets, Intercept has suggested that an addressable population of ~30,000 may be eligible for treatment owing to inadequate response or intolerance to urso.

Considering POISE Phase 3 Trial Design

The phase 3 POISE study is evaluating OCA in combination with ursodiol in patients with PBC that have inadequate therapeutic response to ursodiol or who are unable to tolerate ursodiol. Patients enrolled in the POISE study have to have been on ursodiol for at least 12 months or in the case of patients that are ursodiol intolerant, stopped taking the medication at least 3 months prior to study entry. The primary endpoint of the POISE study is the proportion of patients meeting pre-defined response criteria at 12 months, including achievement of ALP <1.67x ULN, at least a 15% reduction in ALP from baseline together with a normal bilirubin.

Exhibit 7: POISE Phase 3 Study Schema



Source: Intercept Pharmaceuticals and BMO Capital Markets.

The POISE study was initiated in January 2012 and is designed to enroll 180 patients across ~60 centers in North America, Europe, and Australia. The study has 90% power to detect a difference in PBC response between treatment arm at 40% and control arm of 15%. Note that the control arm response rate would be predicted at 9% based on prior phase 2 experience. Key secondary endpoints include:

- proportion of patients achieving 10%, 20%, and 40% reductions in ALP;
- proportion of patients with ALP <3xULN, AST<2xULN and normal bilirubin;

- proportion of patients achieving ALP <1.5xULN, AST<1.5xULN and normal bilirubin;
- separate measures of albumin, GGT, ALT, AST, PTT and INR;
- quality of life (QOL), measures of pruritus through the 5-D pruritus questionnaire and visual analog scale (VAS);
- enhanced liver fibrosis (ELF) test;
- transient elastography (TE); and
- biomarkers of liver fibrosis including TNF-alpha, TGF-beta, IL-6, CK-18, and lysophosphatidic acid.

Considering Detail of Phase 2 Studies in PBC

Intercept has previously conducted two phase 3 trials of OCA in patients with PBC. Phase 2 studies included one study of OCA in combination with ursodiol in patients with prior inadequate treatment response or intolerance to ursodiol as well as a second monotherapy study in patients naïve to ursodiol or who had discontinued ursodiol previously. Both studies met primary endpoints related to reductions in ALP as well as numerous secondary endpoints related to other key measures of liver health.

Phase 2 Combination Study Provides Insight Into POISE

The phase 2 combination study of OCA + ursodiol randomized 165 patients with ALP levels >1.5xULN and inadequate response to ursodiol to the addition of OCA 10mg, OCA 25mg, OCA 50mg or placebo for 12 weeks of treatment and 2 weeks of follow-up.

The phase 2 combination study of OCA + ursodiol (study 747-202) enrolled patients from 8 countries and 33 centers, with 56% of patients enrolled in the US, 26% enrolled in Canada, and 18% enrolled in Europe.

Patients enrolled in the study were between the ages of 18 and 70 years and had proven or likely PBC by virtue of two of three of the following criteria:

- A history of elevated ALP for >6 months.
- A positive anti-mitochondrial antibody (AMA) titer or PBC-specific anti-nuclear antibody.
- A liver biopsy result consistent with features of PBC.

Overall 65% of patients enrolled in the study met all three criteria for PBC diagnosis and 35% met two criteria for PBC diagnosis. Patients enrolled in the phase 2 combination study were excluded if they had prior exposure to colchicine, methotrexate, azathioprine or steroids over the prior three months, if they had any history of decompensated liver disease or esophageal

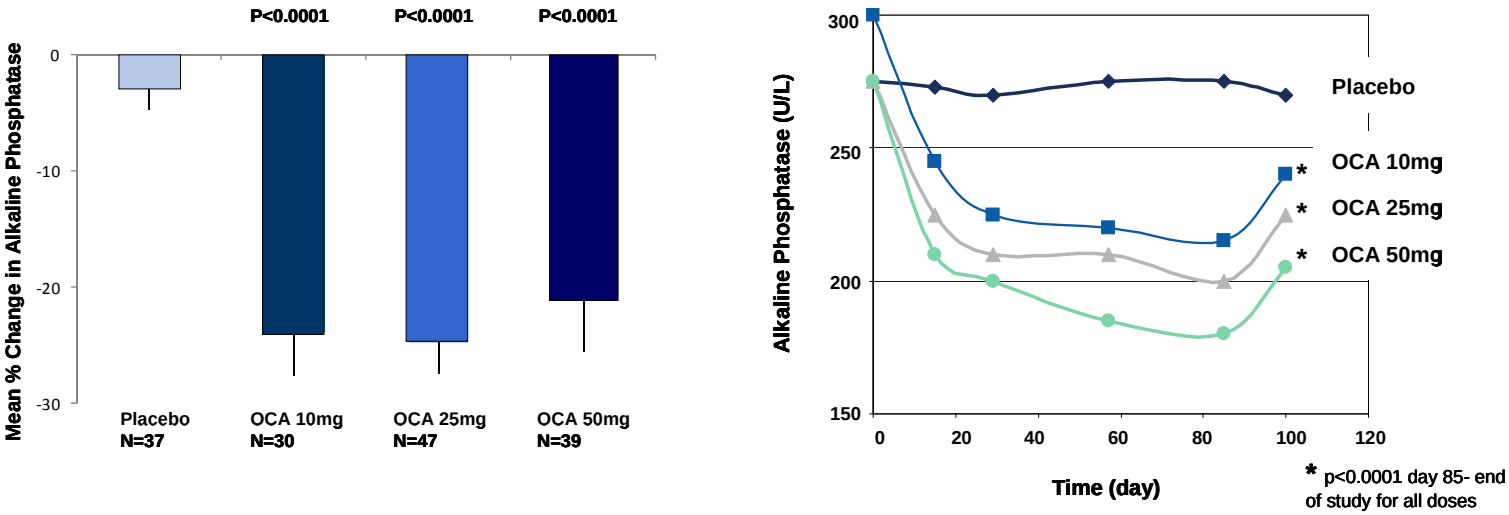
varices and if they had bilirubin >2xULN, ALT/AST >5xULN, serum creatinine >1.5mg/dl or if they had other concomitant liver diseases.

Overall, 221 patients were screened for inclusion in the phase 2 combination study, 165 were randomized to the study, 136 completed the study with 27 discontinuing therapy and with 2 patients lost to follow-up and with a total of 161 patients included in the modified intent to treat (mITT) analysis. The four patients excluded from the mITT were those that had no study visit beyond the baseline visit.

A total of 95% of patients enrolled in the phase 2 combination study were female, 96% were Caucasian, average age was 55 years, and average dose of ursodiol was 15.9mg/kg. Baseline ALP levels for patients enrolled were 286.6 IU/L and 2.4x ULN overall and fairly consistent across groups in the study.

Patients enrolled in the phase 2 combination study had baseline ALP levels of ~2.4xULN. At the end of the 12-week treatment period all three doses of OCA produced statistically significant reductions in ALP levels, with mean reductions of 21%–25% vs. 3% with placebo treatment. Addition of OCA to ursodiol also produced similar significant reductions in other liver markers including GGT, AST, ALT, and bilirubin as well as in markers of inflammation, including CRP and IgM levels.

Exhibit 8: Mean % Change and Absolute Changes in Alkaline Phosphatase Levels in OCA Phase 2 PBC Trial

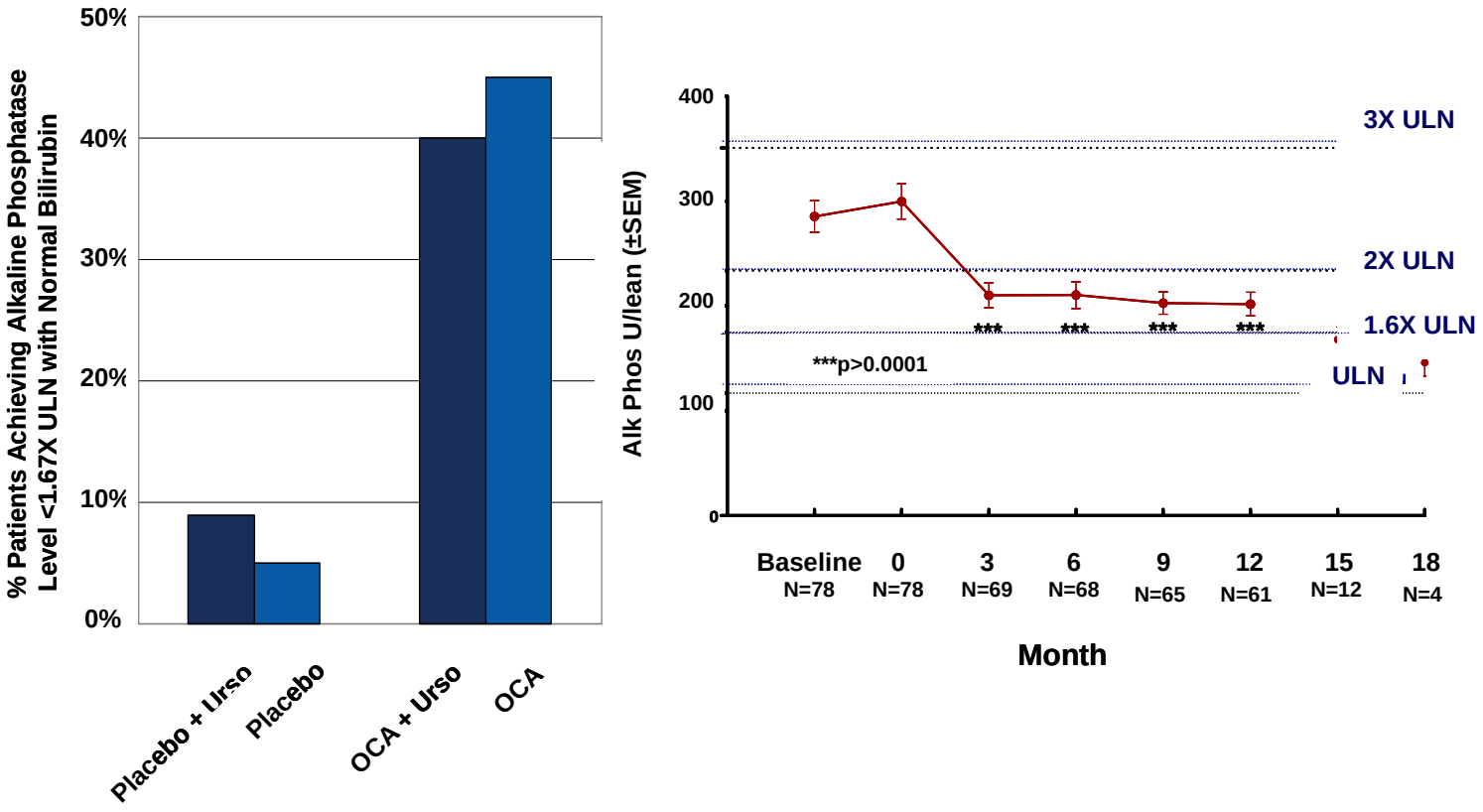


Source: Intercept Pharmaceuticals and BMO Capital Markets.

Specific reductions in other liver biomarkers in the phase 2 combination therapy study included reductions of 48%–63% in GGT, reductions of 21%–35% in ALT, and reductions of 9%–17% in AST. Importantly, no dose response was seen for OCA between 10mg and 50mg when combined with ursodiol, and in fact the reduction in AST was largest with the OCA 10mg dose at 17%.

With the phase 3 POISE study designed around a different composite endpoint at 12 months, as opposed to 12 weeks, longer-term data have been reported from a long-term open label extension study in 78 of 165 patients enrolled in the phase 2 combination study. In the phase 2 combination study the addition of OCA reduced baseline ALP levels from 2.4xULN to roughly 1.67xULN after 3 months of treatment and maintained that level to 12 months of follow-up. Importantly, in the long-term extension of the phase 2 study >50% of patients treated with OCA achieved the POISE primary endpoint of ALP reduction <1.67x ULN, 15% ALP reduction and normal bilirubin level at 12 months of follow-up.

Exhibit 9: OCA Increases Proportion of Patients Achieving Normalization of Alkaline Phosphatase



Source: Intercept Pharmaceuticals and BMO Capital Markets.

The phase 2 combination study also highlights potential adverse events (AEs) associated with the combination of OCA and ursodiol, with pruritus (itching) as the primary side effect emerging as the dose of OCA was escalated from 10mg to 50mg. Overall rates of pruritus in the phase 2 combination study were 47% for OCA 10mg, 85% for OCA 25mg, 80% for OCA 50mg and 50% for placebo. Severe pruritus and study discontinuation due to pruritus were higher than placebo at all OCA dose levels and also demonstrated a clear dose response. Importantly, the severity of pruritus with OCA 10mg was described as mild in roughly 50% of patients whereas the pruritus was described as severe in the majority of patients receiving OCA

50mg. In general other adverse events were evenly distributed between OCA-treated patients and placebo-treated patients, with the exception of mild nausea. There were no severe adverse events (SAEs) reported with OCA 10mg dose in the phase 2 combination study and only four SAEs deemed possibly related to OCA 50mg, including a GI bleed, a case of jaundice, a PBC flare, and an episode of chest pain.

Phase 2 Monotherapy Study Confirms OCA Benefit in PBC

In addition to the phase 2 combination study Intercept has also completed at phase 2 monotherapy study in PBC patients naïve to ursodiol or discontinued from ursodiol for at least three months prior to study entry. The phase 2 monotherapy study enrolled 59 patients and was designed to compare OCA 10mg or OCA 50mg vs. placebo over a 12-week treatment period and 2-week follow up period. The phase 2 monotherapy study for OCA in PBC, or study 747-201, enrolled patients aged 18-70 years, with likely or proven PBC by virtue of at least two of the following criteria:

- History of elevated ALP levels for >6 months
- Positive antimitochondrial antibody (AMA) titer
- Liver biopsy consistent with PBC

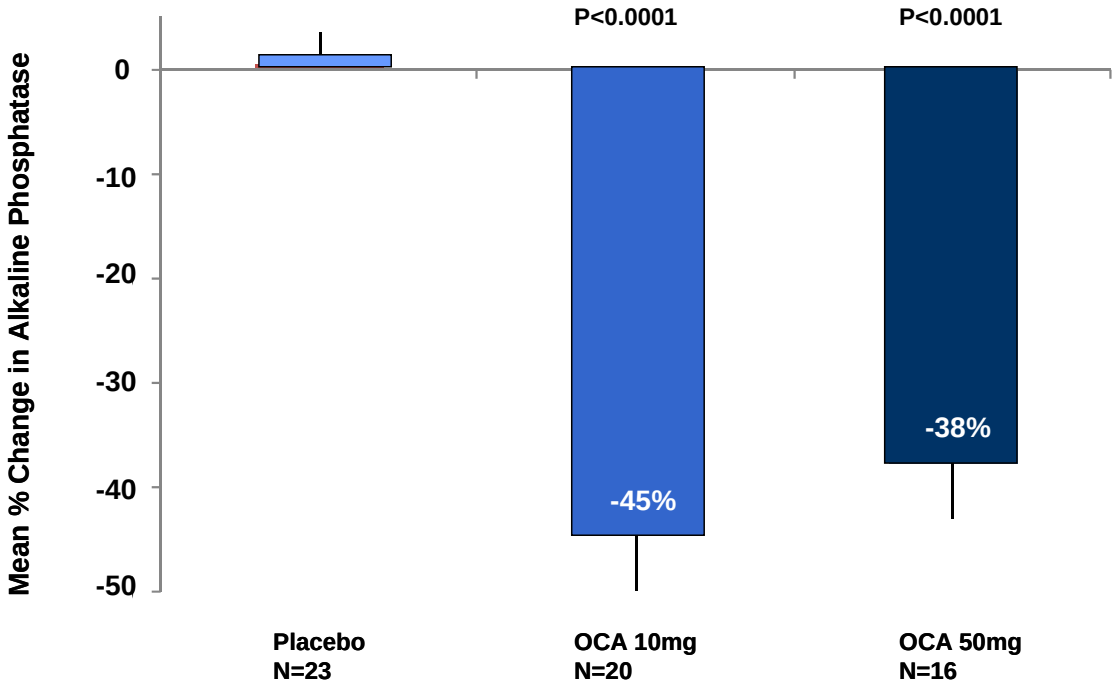
Patients enrolled in the phase 2 monotherapy study had baseline ALP levels of 1.5x to 10xULN and patients were excluded if there was any history of liver decompensation, baseline bilirubin >2xULN, baseline ALT or AST >5xULN, serum creatinine >1.5mg/dl or other concomitant liver disease like hepatitis B (HBV) or hepatitis C (HCV). Patients in the study were enrolled from 7 countries and 20 centers, with 28% of patients from the US, 15% of patients from Canada, and 57% of patients from Europe.

Overall, 71 patients were screened for the phase 2 monotherapy study; 60 were randomized to treatment; 12 discontinued, were lost to follow-up, or withdrew consent; 48 completed the study; and 59 patients were included in the intent to treat (ITT) analysis, with only 1 patient excluded who withdrew consent prior to the first study dose.

With primary focus on the OCA 10mg dose group reductions in ALP from a mean level of 3.9xULN to 1.9xULN were reported. Efficacy, in terms of ALP reduction, was roughly comparable between the OCA 10mg dose and OCA 50mg dose with 45% reduction ($p<0.001$) and 38% reduction ($p<0.0001$), respectively. Reductions in other liver markers were also observed with OCA including reductions in GGT of 63%–75%, and reductions in ALT, bilirubin, and IgM levels.

Reductions in other liver markers provide strong support for the benefit of OCA monotherapy in patients with PBC with ALT reductions of 37% ($p=0.08$) for OCA 10mg and 35% for OCA 50mg (p=NS) and with bilirubin reductions of -0.7 uMol/L for OCA 10mg ($p=0.018$) and -0.30 uMol/L for OCA 50mg ($p=0.02$). Importantly, given the suspected underlying immune pathogenesis, treatment with OCA also reduces signs of autoimmune activation with IgM reductions of -0.7 g/L with OCA 10mg and with reduction of -1.2 g/L with OCA 50mg.

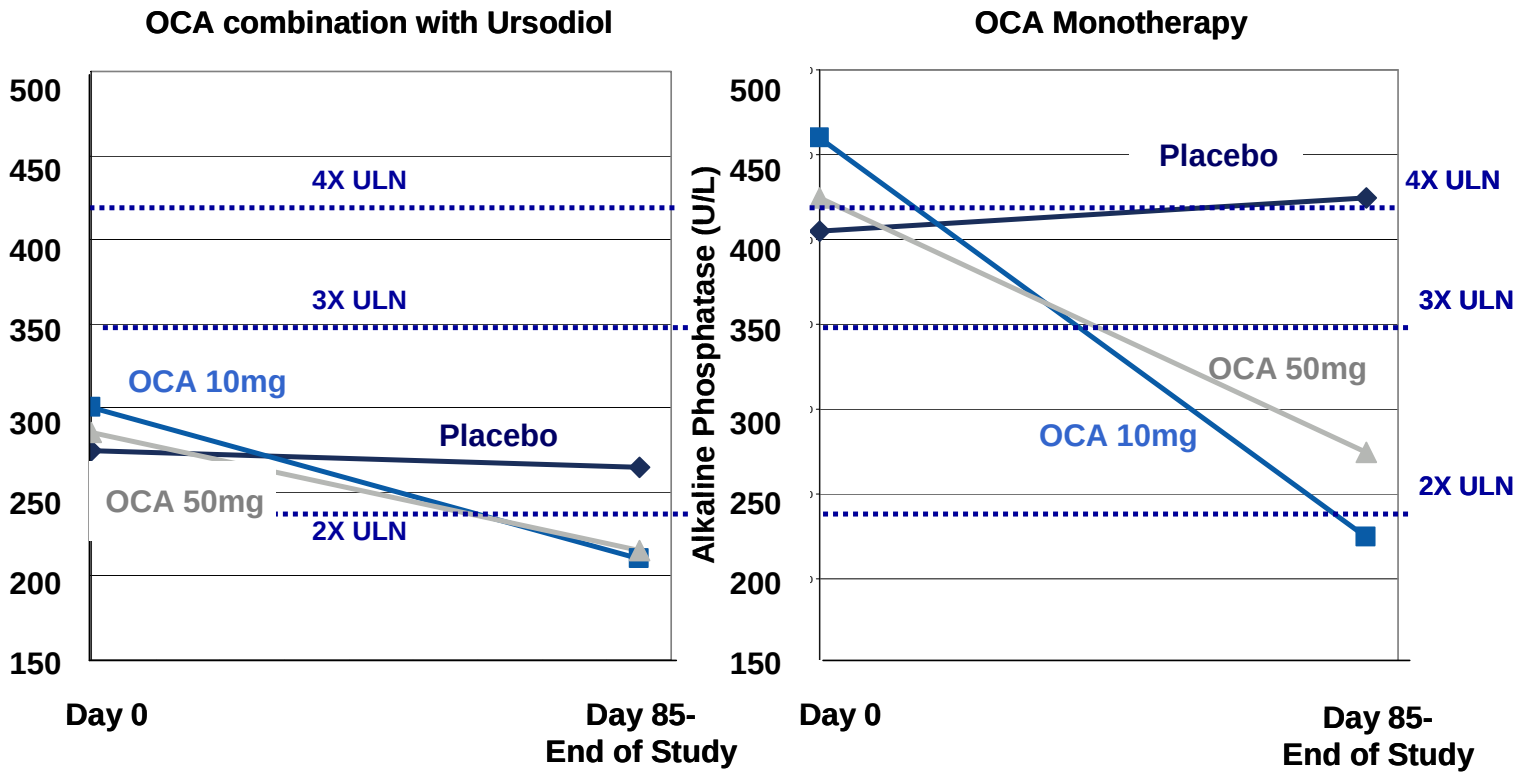
Exhibit 10: Mean Change in Alkaline Phosphatase in Patients Receiving OCA Monotherapy



Sources: Intercept Pharmaceuticals and BMO Capital Markets.

As with the OCA + ursodiol combination study the phase 2 monotherapy study had an open label long-term extension phase that is still ongoing. With primary focus on the POISE phase 3 composite endpoint ~45% of OCA treated patients experienced the combination of ALP <1.67xULN, absolute reduction in ALP of at least 15% and normal bilirubin level after 12 months of treatment. The composite response rate of ~45% compared favorably to the placebo composite response rate of <5% and achieved statistical significance (p=0.012). Additional responder analysis from the phase 2 monotherapy study suggests that 70% of patients receiving OCA 10mg met Barcelona response criteria, only 44% of patients receiving OCA 50mg met Barcelona response criteria and no patients receiving placebo met Barcelona response criteria.

Exhibit 11: Summary of Alkaline Phosphatase Changes in Patients Receiving OCA



Source: Intercept Pharmaceuticals and BMO Capital Markets.

Long-term safety and tolerability data from the OCA monotherapy study was consistent with that reported from the combination study and included an 86% rate of pruritus and rates of headache, arthralgia, fatigue, and nausea of 32%, 25%, 21%, and 21%, respectively. Looking at adverse events (AE) more closely by dose level suggests lower rates with OCA 10mg vs OCA 50mg, and with the exception of pruritus, little difference between OCA 10mg and placebo. Specifically pruritus was reported in 94% of patients treated with OCA 50mg, 70% of patients treated with OCA 10mg, and 30% of patients treated with placebo. Roughly 40% of patients receiving OCA 50mg discontinued due to pruritus but only 15% of patients receiving OCA 10mg discontinued due to pruritus. Dizziness, on the other hand, was experienced by 17% of placebo patients and with neither dose of OCA, fatigue was experienced by 13% of placebo patients, 6% of OCA 50mg patients, and no patients with OCA 10mg. Finally, nausea was experienced by 25% of patients receiving OCA 50mg, by 17% of placebo patients, and by no patients receiving OCA 10mg.

Considering the Value of Surrogate Endpoints in PBC

Assuming success in the POISE phase 3 study, Intercept intends on submitting a New Drug Application (NDA) with FDA and Marketing Authorization Application (MAA) with European Medicines Agency (EMA). Intercept has previously been provided with written scientific advice from EMA suggesting that the surrogate endpoints in POISE will be acceptable as a basis for considering approval in PBC. Intercept has not yet reached full agreement with FDA on the acceptability of ALP and bilirubin as endpoints but intends to request accelerated approval under new regulations that enable use of a surrogate endpoint reasonably likely to predict clinical benefit.

In considering the acceptability of the POISE surrogate endpoints it is worthwhile to review published literature on the value of ALP and bilirubin levels in terms of predicting clinical outcomes in patients with PBC. Several published clinical studies have demonstrated that reductions in ALP, on its own or in combination with normal bilirubin levels, correlate with a significant reduction in liver transplant and death.

In considering the value of the surrogate endpoint chosen for the POISE study and supported by scientific advice from EMA it is worthwhile reviewing the evolution of various treatment response criteria that have emerged from leading PBC centers and that have informed Intercept in its selection of the POISE phase 3 composite endpoint.

In reviewing published data designed to correlate biochemical markers and clinical outcomes in PBC we would note different response criteria from several groups, which we believe in aggregate support the POISE phase 3 surrogate endpoint. These biochemical response criteria include:

- Barcelona Criteria (Pares) - 40% decrease in ALP or normalization of ALP.
- Rotterdam (Kuiper) - normalization of bilirubin and/or albumin.
- Paris I (Corpechot) - ALP <3xULN and AST <2xULN and bilirubin <1mg/dl.
- Paris II (Corpechot) - ALP <1.5xULN and AST <1.5xULN and bilirubin <1mg/dl
- Toronto I (Kumagai) - ALP <1.76xULN
- Toronto II (Kumagai) - ALP <1.67xULN
- Toronto III (Kumagai) - ALP <1.67xULN and bilirubin <1mg/dl
- Modified Toronto III (Intercept) - ALP <1.67xULN and 15% decrease in ALP and bilirubin <1mg/dl

The Barcelona response criteria refers to a study conducted by Dr. Albert Pares from the Liver Unit of the Digestive Disease Institute in Barcelona, Spain. In this study, designed to assess long-term survival in patients with ALP response, Pares and his group in Barcelona evaluated 192 patients with PBC treated with ursodiol for 1.5 to 14 years. The Barcelona response criteria

was defined as a >40% decrease in ALP from baseline or achievement of normal ALP after one year of therapy.

Long-term survival in the study was compared to that predicted by a PBC survival model from the Mayo Clinic and against a standardized matched control group in the general Spanish population. With 17 of 192 (8.9%) patients progressing to death or liver transplant eligibility, data suggest that 61% of patients meeting Barcelona response criteria had overall survival better than that predicted by the Mayo Clinic model and similar to that for the normal control population, while the 39% of patients failing to meet response criteria was significantly lower than that predicted for the control population, although also higher than that predicted by the Mayo Clinic model.

The Toronto response criteria published by Kumagai and colleagues has evolved over time to include multiple biochemical endpoints as predictors of histologic progression of disease. Central to the various response criteria identified by the Toronto group has been an ALP threshold of roughly 1.67–1.76xULN. In a publication from the *American Journal of Gastroenterology* in 2010 Kumagai and colleagues in Toronto reviewed results from 69 patients with PBC and follow-up liver biopsy ~ 10 years after initial histologic diagnosis. Results suggest that histologic progression in the stage of fibrosis by one stage was associated with ALP >1.67xULN, and with ALP >1.76xULN in patients with progression of fibrosis through two stages. Overall, patients with Toronto response criteria of ALP <1.67xULN have no histologic progression of disease over 10 years.

More recently Dr. Corpechot and colleagues from Paris have published a review in the *Journal of Hepatology* evaluating the efficiency of various response criteria to predict outcomes in patients with early-stage PBC. In their review of 165 patients at their own center the Paris group compared biochemical response criteria after one year of treatment with ursodiol in terms of ability to discriminate between patients with favorable outcome and those with unfavorable outcome in terms of liver-related death, liver transplant, complications of cirrhosis, and histological evidence of cirrhosis. Response criteria evaluated by Corpechot included the following:

- Barcelona criteria - decrease in ALP >40% from baseline or reaching normal levels.
- Paris criteria - ALP <3xULN, AST <2xULN and normal bilirubin, and modified criteria of ALP/AST <2xULN and normal bilirubin (Paris IIa), ALP/AST <1.5XULN (Paris IIb) and normal bilirubin and normal ALP-AST-bilirubin.
- Rotterdam criteria - normal bilirubin and albumin levels when one, or both, were abnormal at baseline.
- Toronto criteria - ALP <1.76xULN.

Across the Corpechot study the rate of biochemical response ranged from 63% to 87% over one year of treatment with ursodiol and rate of adverse outcome in non-responders ranged from 4% to 27%. In the study both the Paris criteria and Toronto criteria significantly discriminated patients between patients with favorable and unfavorable longer-term liver outcomes, while no significant association with long-term outcomes was observed with the Barcelona and

Rotterdam criteria. In evaluating variations of the Paris response criteria with lower ALP and AST thresholds the Corpechot study suggests that the best predictive value came from a definition of ALP and AST <1.5x ULN with normal bilirubin. The Toronto group has also further refined its response criteria to include ALP <1.67xULN, 15% reduction in ALP and normal bilirubin, with strong support from its own database to inform the POISE phase 3 composite responder endpoint.

Given the lack of clear consensus on the optimal response criteria in PBC additional analyses are being conducted in a large independent "Supergroup" study of pooled patient data from >10 leading PBC centers in North America and Europe and from ~4,000 patients. Results from this pooled analysis are expected in 2013 and Intercept expects that the data will help substantiate the modified Toronto response criteria and POISE phase 3 surrogate endpoint. We expect the initial data analysis to focus specifically on the POISE endpoint of ALP <1.67xULN, 15% reduction in ALP and normal bilirubin, but do expect subsequent analysis to include assessments of Paris, Rotterdam, and Barcelona criteria and likely modifications of each of these. To the extent that other composite response criteria emerge from the "Supergroup" study as more optimal than the Intercept criteria we believe that POISE will capture these other metrics of response.

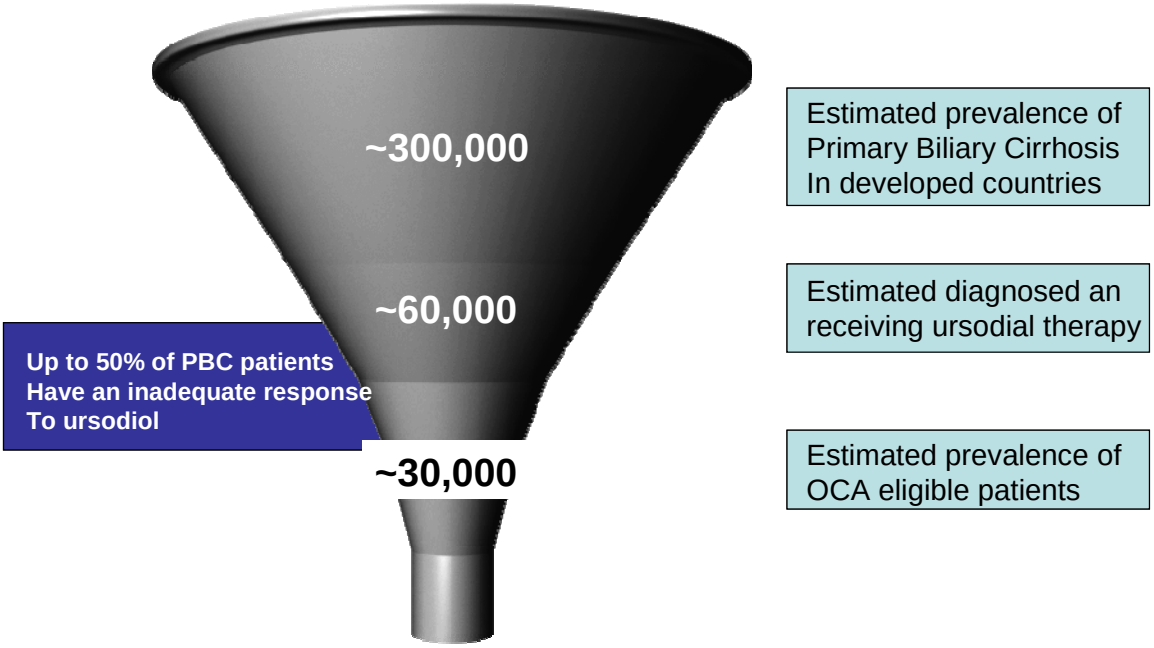
Ultimately the question on clinical relevance of any surrogate endpoint comes down to a review issue for FDA and one to be vetted through expert panel. To that end we believe that the evolution of FDA thinking toward use of surrogate endpoints for rare orphan diseases is important to consider. We would note that on July 9, 2012 the Food and Drug Administration Safety and Innovation Act (FDASIA) was signed into law with the reauthorization of the Prescription Drug User Fee Act (PDUFA) with PDUFA V performance goals established for the period from 2013 to 2017. Within PDUFA V are specific performance goals to advance development of drugs for rare disease including increasing staffing for the CDER Rare Disease Program, development of guidance for facilitating development of drugs for rare disease and improving understanding among FDA reviewers regarding non-traditional development programs, recognition of challenges for post-marketing studies and encouraging flexibility and scientific judgment, as appropriate, when reviewing applications for rare disease.

Since the passage of FDASIA and PDUFA V reauthorization in July we have seen a number of positive FDA and/or FDA advisory panel recommendations for drugs that treat rare diseases. Included in this list are KYPROLIS (carfilzomib) as salvage therapy for relapsed/refractory myeloma, GATTEX (teduglutide) for short-bowel syndrome (SBS) and both lomitapide and KYNAMRO (mipomersen) for homozygous familial hypercholesterolemia (HoFH).

Considering the Commercial Opportunity

As suggested earlier there are an estimated 300,000 patients in developed countries with primary biliary cirrhosis (PBC), of which 60,000 patients are estimated to be receiving treatment with ursodiol and where 50% of patients, or 30,000 patients, are estimated to have inadequate treatment response to ursodiol alone.

Exhibit 12: Defining the OCA Market Opportunity in PBC



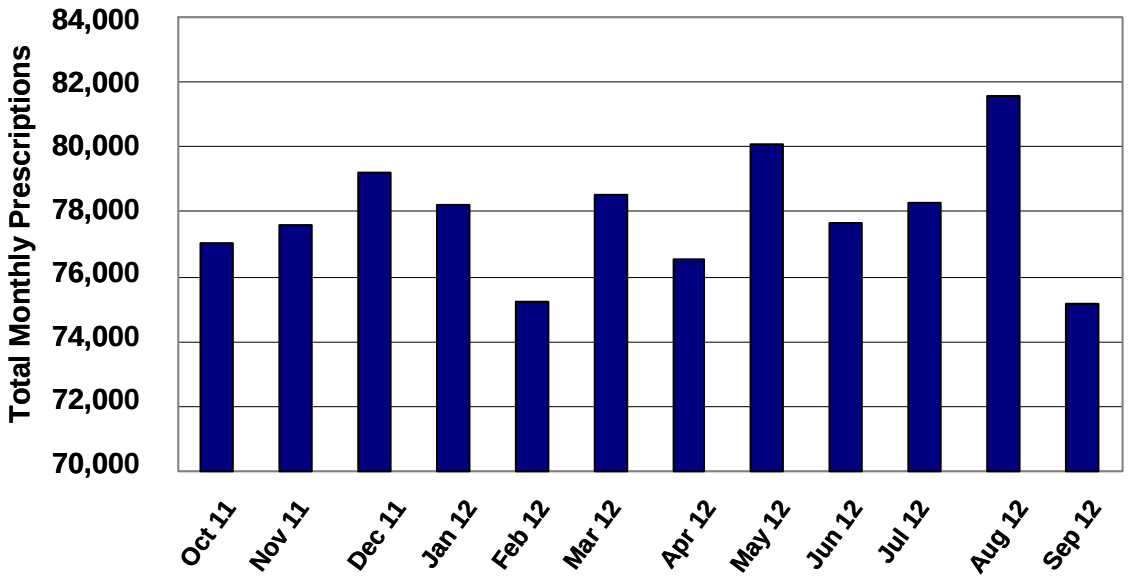
Source: Intercept Pharmaceuticals and BMO Capital Markets.

Over the last 15–20 years a number of drugs have been evaluated in patients with PBC and results have been uniformly disappointing by virtue of lack of efficacy or unacceptable toxicity. Examples include Rifampicin, which improves pruritus but induces hepatitis; azathioprine, which has negligible benefit but is associated with neutropenia; chlorambucil, which has some biochemical benefits but causes bone marrow suppression; and steroids, which show some histologic improvements but are associated with osteoporosis, diabetes, and portal vein thrombosis; and methotrexate, which has no effect and also causes bone marrow suppression.

Historical experience with rare orphan diseases and new therapeutic options to treat these diseases suggests that the key to commercial success is finding the patients. To that end, Intercept may benefit from a well defined patient group being treated with ursodiol, although defining the size of this market in dollar terms is somewhat challenging owing to the use of generics. In reviewing aggregate prescription data across available ursodiol generics as well as brand Urso we a highlight monthly prescription total of ~80,000. The average prescription size

is not known but in taking a conservative assumption that one prescription equals a 30-day supply, on a brand basis, assuming $12,000 per year or $1,000 per month, this prescription level would support sales of $80 million per month or $1 billion per year. Assuming 50% of this branded opportunity were inadequate responders, this would suggest an addressable opportunity of ~$500 million for OCA on very conservative pricing assumptions.

Exhibit 13: Ursodiol Monthly TRx



Source: IMS Health and BMO Capital Markets.

Expanded Opportunities for OCA in Liver Disease

Intercept is currently conducting an open-label phase 2a study of OCA in patients with portal hypertension, another phase 2a study in patients with bile acid diarrhea, and a phase 2b study of OCA for the treatment of non-alcoholic steatohepatitis (NASH).

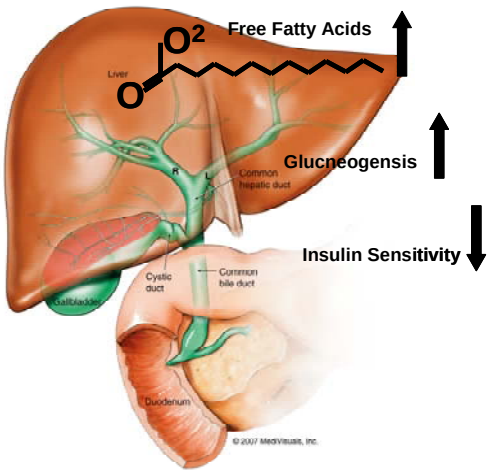
Exhibit 14: FXR Knockout Model Identifies Indications for OCA Beyond PBC

FXR Knockout Phenotype

Liver
Steatosis
Fibrosis
Hepatocellular Carcinoma
Dysregulated Repair

Gastrointestinal
GI Colitis
Nephropathy

Metabolic
Glucose Intolerance
Peripheral Insulin Resistance



Sources: Intercept Pharmaceuticals and BMO Capital Markets.

Data flow from these expanded liver disease indications is expected to be significant over the next few years, with portal hypertension data by YE12 and with data in NASH expected by YE14.

OCA for Portal Hypertension

Intercept is currently conducting an open-label phase 2a trial of OCA in patients with portal hypertension. The phase 2a study is called PESTO and is designed to evaluate the effect of OCA on hepatic portal venous pressure in patients with end-stage liver disease. Patients in the PESTO study are treated initially with OCA at a dose of 10mg for seven days with a primary endpoint of lowering hepatic venous pressure gradient by 15% or more or to a level of 12 mm Hg or less. Currently 12 patients have been enrolled at the 10mg dose level, and initial data on 8 evaluable patients have been reported in abstract form as a late breaker for the American Academy for the Study of Liver Disease (AASLD) annual Liver meeting in Boston.

Results from the PESTO study in 12 patients with established alcoholic cirrhosis and portal hypertension suggested that short-term low-dose OCA therapy was safe, well tolerated, and lowered hepatic venous gradient pressure (HVPG) in 5 of 6 evaluable patients. Of the 12 patients enrolled, 4 were assessed for drug safety and tolerance and 8 for changes in HVPG

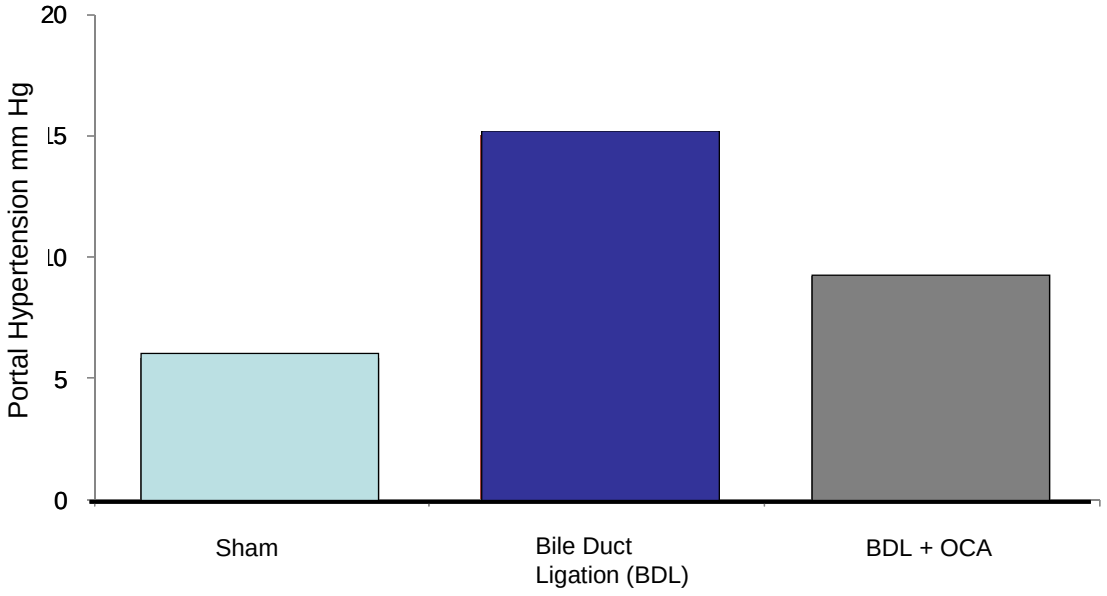
before and after seven days of OCA (10mg/d). Response to OCA was defined as a HVPG reduction to <15 mmHg or ≥15% from baseline. Of the eight efficacy evaluable patients, five were considered responders with an average change in HVPG of 4 mmHg from 16 mmHg at baseline to 12 mmHg at the end of study. This equated to a mean percent change in PVPG of 24% (range 15% to 38%). Of the three non-responders, one showed an HVPG decrease from 14 mmHg to 12 mmHg, one non-change in HVPG, and one an increase in HVPG. In addition to changes in HVPG, all responders showed an increase in mean arterial pressure from baseline.

All patients tolerated OCA with adverse events reported being mild and self limiting including nausea (n=2), headache (n=2) and one each with localized pruritus, flushing, and dyspnea.

Portal hypertension is a consequence of liver cirrhosis and occurs in patients with end-stage liver disease. The cirrhotic liver provides increased resistance to portal vein circulation and pressure builds in the portal vein with potential disastrous consequences. One of the early consequences of portal hypertension is the development of esophageal varices, whereby veins in the lower part of the esophagus become distended and rupture. Treatment of portal hypertension, to avoid esophageal varices development and other complications, typically involves use of beta blockers, although results are mixed with only 25%–30% of patients responding.

OCA has been studied in an animal model of liver cirrhosis with treatment resulting in acute reversal of portal hypertension. Reductions in portal vein pressure of roughly 5 mmHg were statistically significant when compared to a positive control ($p<0.001$). The mechanism of action of OCA in this model of portal hypertension was localized vasodilation without broader reductions in systemic blood pressure.

Exhibit 15: OCA is Effective in a Model of Portal Hypertension



Source: Intercept Pharmaceuticals and BMO Capital Markets.

OCA for Non-Alcoholic Steatohepatitis (NASH)

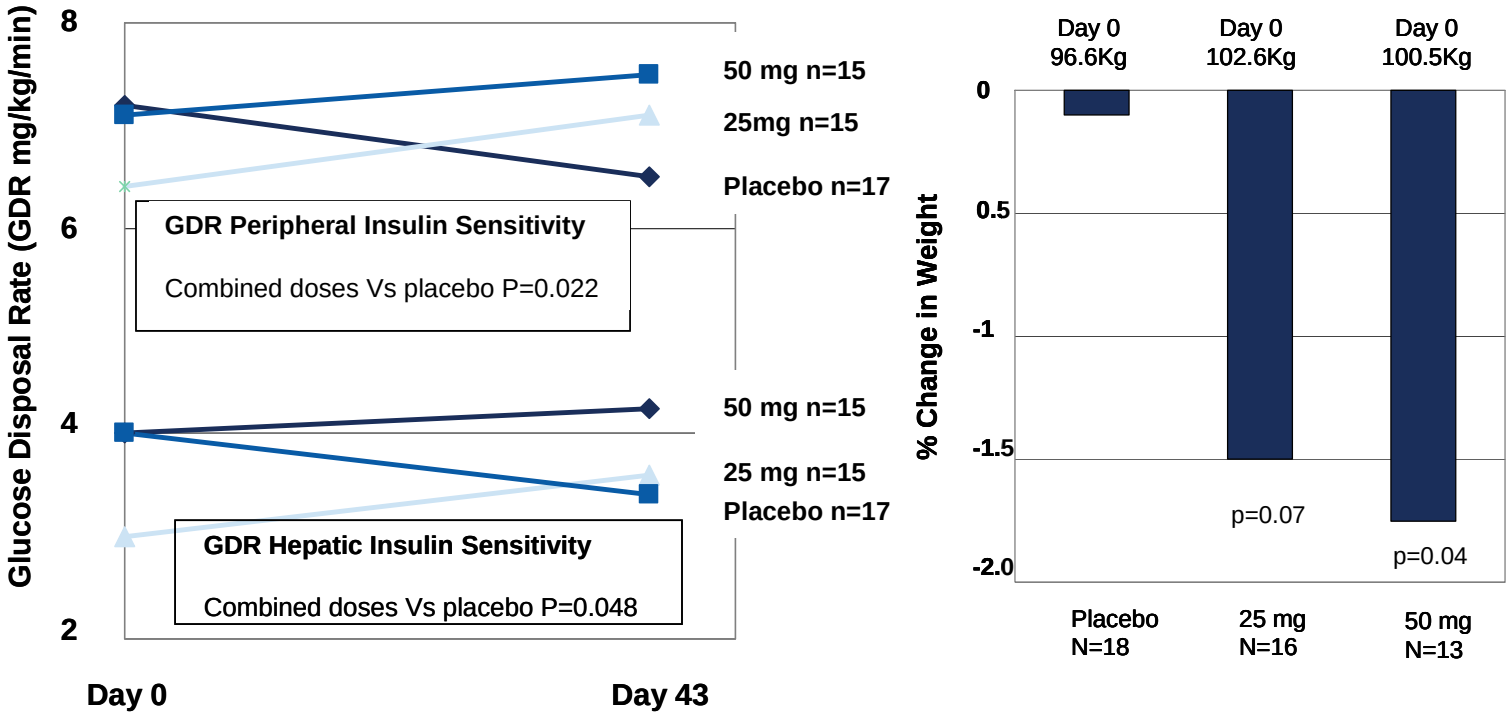
Activation of FXR has been shown to play a key role in regulation of metabolic pathways involved in non-alcoholic steatohepatitis or NASH, a condition thought to affect >12% of the US population. NASH develops in ~30% of patients with non-alcoholic fatty liver disease (NAFLD), a condition of excess fat accumulation in the liver. The transition from NAFLD to NASH is not well understood but involves induction of chronic inflammation in response to liver fat accumulation. It is estimated that ~2.7% of the US population, or roughly 6 million patients, have advanced liver fibrosis or cirrhosis due to NASH.

Intercept is currently sponsoring, in collaboration with the US National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK), a phase 2b study of OCA in patients with NASH called the FLINT study. The FLINT study is a multi-center, randomized, placebo-controlled trial designed to randomize 280 patients with NASH to either OCA 25mg or placebo over a 72-week time period. The primary endpoint is measured by liver biopsy and is defined as an improvement of two or more points in the NAFLD histopathologic activity score with no worsening of liver fibrosis. The study is funded primarily by NIDDK, which overseas clinical research at eight leading NASH centers in the US, and data are expected by YE14.

The FLINT trial is being overseen by a data safety monitoring board (DSMB) and in June 2012 the DSMB performed an interim analysis supporting continuation of the study. This first interim analysis was based on data from 101 patients completing at least 24 weeks of the FLINT study and up to 15 months of dosing. The analysis assessed ALT changes from baseline as well as all available safety data. With ALT change from baseline as the efficacy criterion and with consideration of all safety data the DSMB determined that it was appropriate for the FLINT study to continue.

Prior to FLINT, Intercept conducted a phase 2 trial of OCA in patients with type 2 diabetes and nonalcoholic fatty liver disease (Study 203). This multi-center, double-blind, randomized, placebo controlled study enrolled 64 patients in the US. Patients were randomized to OCA at 25 mg/d, 50 mg/d or placebo for six weeks of treatment. The primary endpoint was improvement in insulin sensitivity, with secondary endpoints including liver and metabolic function and weight change.

Exhibit 16: Summary of Alkaline Phosphatase Changes in Patients Receiving OCA



Source: Intercept Pharmaceuticals and BMO Capital Markets.

As noted in Exhibit 16, OCA improved glucose disposal rate in patients with type II diabetes and NASH in both the liver and the periphery. These metabolic changes were accompanied by reductions in weight, which at the 50 mg dose of OCA amounted to a ~4 pound decrease over 43 days.

Phase 2 results for OCA in patients with diabetes and non-alcoholic fatty liver disease (NAFLD) are consistent with preclinical data published in 2009 (Sanyal A., Hepatology 2009; 50 (S4): 398A) that demonstrated improved insulin sensitivity and dose related decrease in weight.

OCA for Bile Acid Induced Diarrhea

Bile acid diarrhea is a common, but under-recognized, subtype of chronic diarrhea associated with irritable bowel syndrome (IBS) and inflammatory bowel disease (IBD). Indeed, roughly 32% of patients with IBS-D have evidence of primary bile acid diarrhea and 80% of these patients respond to the bile acid sequestrant cholestyramine.

The disorder is thought to be due to over-production of bile acids by the liver due to loss of negative feedback from fibroblast growth factor 19 (FGF-19), a regulatory hormone of bile acid production produced in the small intestine. FGF-19 synthesis in the small intestine is under

direct regulation of FXR and OCA has been shown to increase FGF-19 in all three completed phase 2 studies.

An open label phase 2a study of OCA in bile acid diarrhea was initiated by Imperial College London in July 2012 and is expected to report results by mid-2013. The OBADIAH trial will assess changes in FGF-19 level over a two-week treatment period in 10 patients with bile acid diarrhea treated with OCA.

Bile Acid Analogue Platform for Targeting FXR

Intercept has dominant positioning in the area of bile acid chemistry and rational design of bile acid analogs against FXR and other bile acid receptors. Its leadership position is supported by key patents, its collaboration with leading scientists at the University of Perugia, and proprietary knowledge and expertise of its employees. OCA was discovered by Intercept co-founder Professor Roberto Pellicciari at the University of Perugia and underlying patents have been assigned to Intercept. The ongoing collaboration with Dr. Pellicciari and chemists at the University of Perugia has resulted in a pipeline of other bile acid analogs that target FXR, and another dedicated bile acid receptor called TGR5.

Expanded Opportunities for FXR/Bile Acid Receptor Targeted Drugs

While primary focus of research and development is on OCA as an FXR agonist for PBC and broader chronic liver diseases, Intercept is developing other novel bile acid analogs targeting FXR as well as a second dedicated bile acid receptor called TGR5. Intercept currently has two separate follow-on bile acid analogs in its pipeline including INT-767, a dual agonist of FXR and TGR5, and INT-777, a potent TGR5 agonist. Both drug candidates are orally bioavailable with the dual FXR/TGR5 agonist INT-767 derived from the primary human bile acid CDCA and with the potent TGR5 agonist INT-777 derived from the primary human bile acid cholic acid.

As a dual agonist of FXR and TGR5 INT-767 is ~5x more potent than OCA as an FXR agonist and has demonstrated greater anti-fibrotic and anti-inflammatory effects than OCA across several animal models of chronic liver disease, chronic intestinal disease, and chronic kidney disease. Intercept is currently targeting INT-767 development for chronic kidney diseases that involve progressive fibrosis and ultimate kidney failure. With initial focus potentially on diabetic nephropathy (DN), Intercept is planning to initiate preclinical development in support of an IND filing, although timelines have not yet been fully established.

Development of INT-777 is further advanced than INT-767 and Intercept has completed preclinical studies necessary to support an IND filing. As a selective and potent TGR5 agonist INT-777 is expected to have effects at increasing endogenous production of GLP-1 and incretin hormone responsible for glucose dependent insulin release from the pancreas. In preclinical models of diabetes, treatment with INT-777 induced GLP-1 secretion with resultant increase in insulin sensitivity and reduction in blood sugar. Notably TGR5 is also implicated in the

regulation of metabolism in fat and muscle and treatment with INT-777 in animal models has been associated with increased basal energy use, prevention of weight gain, reduction in lipids, and reduction in fatty liver and liver fibrosis. Given the broad metabolic effects of INT-777, Intercept is targeting potential development in patients with type 2 diabetes and metabolic syndrome.

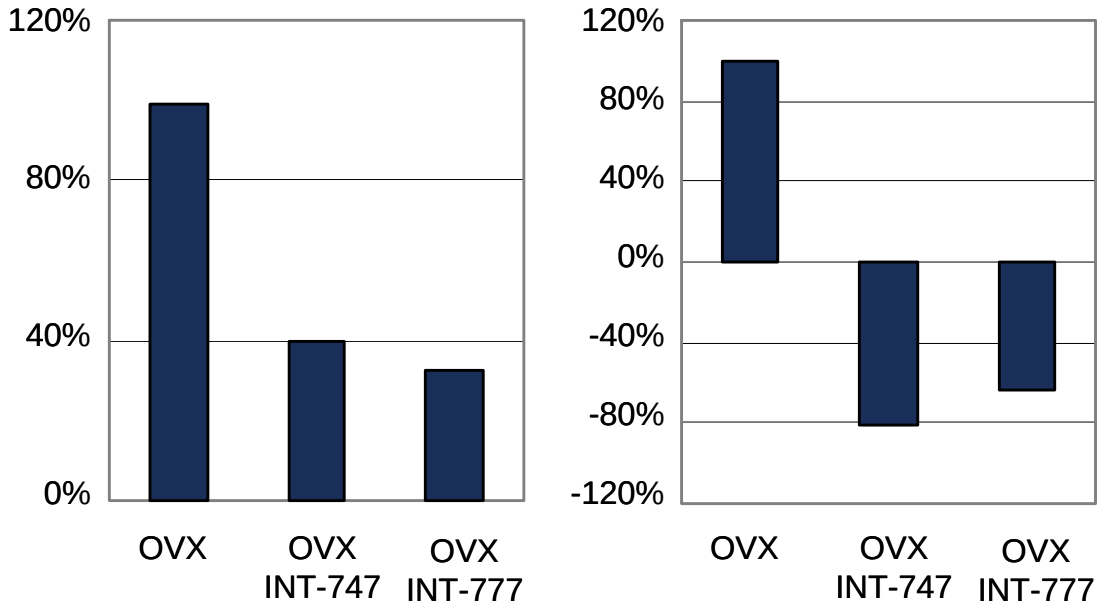
At the 2012 AASLD meeting Dutch and Brazilian researchers will present preclinical data evaluating the potential role of FXR or TGR5 agonism for menopausal-related obesity and hepatic steatosis. The researchers used Intercept's FXR agonist INT-747 and TGR5 agonist INT-777 in a murine model of surgically induced menopause.

Specifically, the researchers compared weight gain and metabolic changes in female mice fed a high fat diet for five weeks following surgical removal of ovaries (ovarectomy - OVX) to induce menopause compared to sham operated mice whose ovaries remained intact. Following surgery the OVX mice were split into four cohorts:

- INT-747 30mg/kg/d
- INT-777 45mg/kg/d
- Estradiol 15 µg/kg/d
- No treatment

The estradiol cohort was included as a positive hormone replacement control and all treatments were administered for the last four weeks of the five-week experiment. At week five all mice were sacrificed. Panel A of Exhibit 17 shows the changes in body weight of OVX mice compared to sham treated controls. OVX mice doubled their weight over five weeks, but this was attenuated by INT-747 or INT-777 treatment. INT-747 treated OVX mice gained 40% less body weight over five weeks than control OVX mice while those treated with INT-747 gained 33% less weight. To rule out non-drug related reasons for the differences in weight gain, the authors noted that no differences in food intake between the different cohorts were observed. Further, despite no differences in physical activity between the groups, indirect calorimetry showed that OVX mice expended less energy than sham mice, with INT-747/777 treated mice intermediate.

Exhibit 17: A/B – Weight Change and Liver Triglyceride Content Compared to Sham Treated Controls



Source: AASLD and BMO Capital Markets.

Panel B of Exhibit 17, shows the differences in liver triglyceride content compared to sham treated animals. Over the five-week experiment, OVX mice receiving a high fat diet exhibited a doubling of liver triglyceride content. In comparison, OVX mice receiving either INT-747 or INT-777 showed an decrease in liver triglyceride content compared to sham controls. Not shown in panel B, but reported in the AASLD abstract, OVX mice receiving hormone replacement therapy showed a 67% decrease in liver triglyceride content. The authors also noted that while no significant changes in the expression of metabolic genes in the livers of mice from different cohorts were found, significant changes were observed in muscle, brown adipose tissue, and white adipose tissue.

The authors conclude that activation of FXR and or TGR5 prevents weight gain and hepatic steatosis caused by ovarectomy. In our opinion, FXR and or TGR5 agonism could represent a novel therapeutic strategy to prevent unwanted weight gain in high-risk individuals following menopause. Mechanistically, activation of FXR and or TGR5 is proposed to increase energy expenditure as evidenced by changes in metabolic genes from numerous tissues.

Strategic Collaborations for Bile Acid Analogues

Intercept has licensed rights to OCA for PBC and NASH to Dainippon Sumitomo Pharma (DSP) in Japan and China (excluding Taiwan), and DSP has an option to obtain an exclusive license to commercialize OCA for other indications beyond PBC and NASH in these same territories. Under terms of the agreement DSP is obligated to use commercially reasonable efforts to commercialize OCA in Japan and China for PBC and NASH. The license agreement with DSP was signed in late March 2011 and involved a $15 million upfront payment, $30 million in development milestone payments, $70 million in regulatory approval milestones and $200 million in sales milestones, all payable by DSP to Intercept. DSP also has a royalty obligation to Intercept escalating from the low tens to the twenties percent level of net sales in Japan, China, and other Asian territories covered in the agreement.

Intercept has also entered into a strategic alliance with French pharmaceutical company Servier for research, development, and commercialization of TGR5 agonists, other than INT-767 and INT-777, for use in diabetes, obesity, atherosclerosis, and reperfusion injury in all countries other than the US and Japan. In exchange Servier has granted Intercept an exclusive royalty-free license to develop these same TGR5 agonists in the US and Japan. Under terms of the agreement Intercept is required to identify and optimize potential TGR5 agonist candidates for further development by Servier.

The agreement with Servier was signed in August, 2011 with upfront payment of $1.4 million by Servier to Intercept and with an initial one-year research term, subject to extension by mutual agreement. Since entering into the original agreement the research term has been extended to July 2012 and then more recently to January 31, 2013. Servier has agreed to pay preclinical and clinical development costs for any TGR5 agonist that meets prespecified criteria. Intercept is required to reimburse Servier for a mid-double-digit percentage of costs related to development in support of a TGR5 agonist product in the US or Europe.

Intellectual Property

Intercept is the owner of 45 issued or granted US and non-US patents relating to OCA as of July 31, 2012. These patents cover pharmaceutical compounds, pharmaceutical compositions, methods of making and methods of using these compounds. In addition, Intercept is the owner of record for 12 pending US and non-US patents relating to OCA. Patents covering the composition of matter for OCA expire in 2022 with other patent and patent applications relating to OCA expiring between 2022 and 2028. With respect to INT-767 and INT-777, composition of matter patents are expected to expire in 2029 and 2030, respectively, although other patents for both compounds begin to expire in 2027 and 2028, respectively.

Outlook and Future Considerations

We have a positive outlook for Intercept based on its strong platform, deep pipeline of bile acid analogs, broad opportunities for OCA in chronic liver disease and attractive lead opportunity in

primary biliary cirrhosis (PBC). Relative to other late-stage orphan drug companies we believe that Intercept is attractively valued with significant upside potential on future revenue opportunities in PBC and with significant option value to other OCA indications and earlier stage programs.

Exhibit 18: ICPT Orphan Drug Comparables

ORPHAN DRUG COMPANIES					
Company	**EV (M)**	**2013E Revenue (M)**	**2013E EV/Rev**	**Stage of Development**	**Therapeutic Focus**
Alexion Pharmaceuticals	$17,374.3	$1,482.6	11.7x	Market	PNH
BioMarin Pharmaceutical	4,509.0	566.7	8.0x	Market	PKU
Amarin	1,591.5	173.4	9.2x	Market	↑ Trg
Synageva BioPharma	898.1	N/A	N/A	Phase 2/3	LAL Def.
NPS Pharmaceuticals	897.1	159.2	5.6x	NDA	SBS
Avanir Pharmaceuticals	330.0	86.5	3.8x	Market	PBA
Aegerion	477.3	30.7	15.6x	NDA	HoFH
Corcept Therapeutics	192.4	40.2	4.8x	Market	Cushing's
Amicus Therapeutics	161.1	41.7	3.9x	Phase 3	Fabry's
Dyax	335.3	78.9	4.2x	Market	HAE
Gentium	159.4	61.4	2.6x	Phase 3	VOD
Mean	**$2,447.8**	**$272.1**	**6.9x**		
Median	**477.3**	**82.7**	**5.2x**		
Intercept Pharmaceuticals	285.3	N/A	NA	Phase 3	Liver Disease

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

We believe that phase 2 data for OCA in PBC is compelling and should bode well for phase 3 success in POISE. Statistically significant lowering of ALP across monotherapy and combination therapy studies goes well beyond the 10% level deemed clinically relevant by PBC key opinion leaders (KOLs) and responder analysis by POISE criteria suggests a significant cushion between what has been demonstrated and what needs to be achieved in phase 3. Internal consistency of data across liver biomarkers, markers of liver function like bilirubin and across markers of autoimmune effects suggests a real biologic benefit that should be replicated in phase 3.

While absence of a special protocol assessment (SPA) and specific agreement with FDA on the primary composite endpoint in POISE presents some risk to OCA approval, we believe that "Supergroup" data will likely validate the surrogate endpoint and reduce this regulatory risk. Irrespective of whether FDA formally accepts the surrogate responder endpoint defined in POISE, this will ultimately be a review issue for a future FDA Gastrointestinal Disease Advisory Committee (GIDAC) panel. Given the strong biologic rationale and preclinical data for OCA in PBC, consistent benefit across endpoints in phase 2, multiple endpoints to confirm benefit in phase 3, prior approval of a less effective bile acid product, and very strong thought leader support, we believe, that OCA approval in PBC is very likely.

Assuming positive phase 3 POISE data and favorable FDA panel review we expect OCA launch for PBC in 4Q14 with EMA approval and launch in Europe on a one quarter lag to 1Q15. We estimate global sales of OCA from 2015 through 2018 of $59 million, $128 million,

$181 million, and peak sales of ~$500 million in 2025, assuming ~60% penetration of patients with inadequate ursodiol response. Our OCA sales estimates support profitability within one year of launch, with 2016 to 2018 EPS estimates of $2.01, $3.27, and $4.58 on a fully taxed basis, and with an estimated three-year EPS CAGR beyond 2018 of 22%.

Exhibit 19: Probability-Adjusted NPV Scenarios in PBC

ICPT NPV SCENARIO ANALYSIS							
Likelihood of EU Approval	100%	$37.22	$39.60	$41.98	$44.35	$46.73	$49.11
	90%	$34.69	$37.07	$39.44	$41.82	$44.20	$46.57
	80%	$32.15	$34.53	$36.91	$39.29	$41.66	$44.04
	70%	$29.62	$32.00	$34.37	$36.75	$39.13	$41.50
	60%	$27.09	$29.46	$31.84	$34.22	$36.59	$38.97
	50%	$24.55	$26.93	$29.31	$31.68	$34.06	$36.44
		50%	60%	70%	80%	90%	100%
		Likelihood of FDA Approval					

Source: BMO Capital Markets.

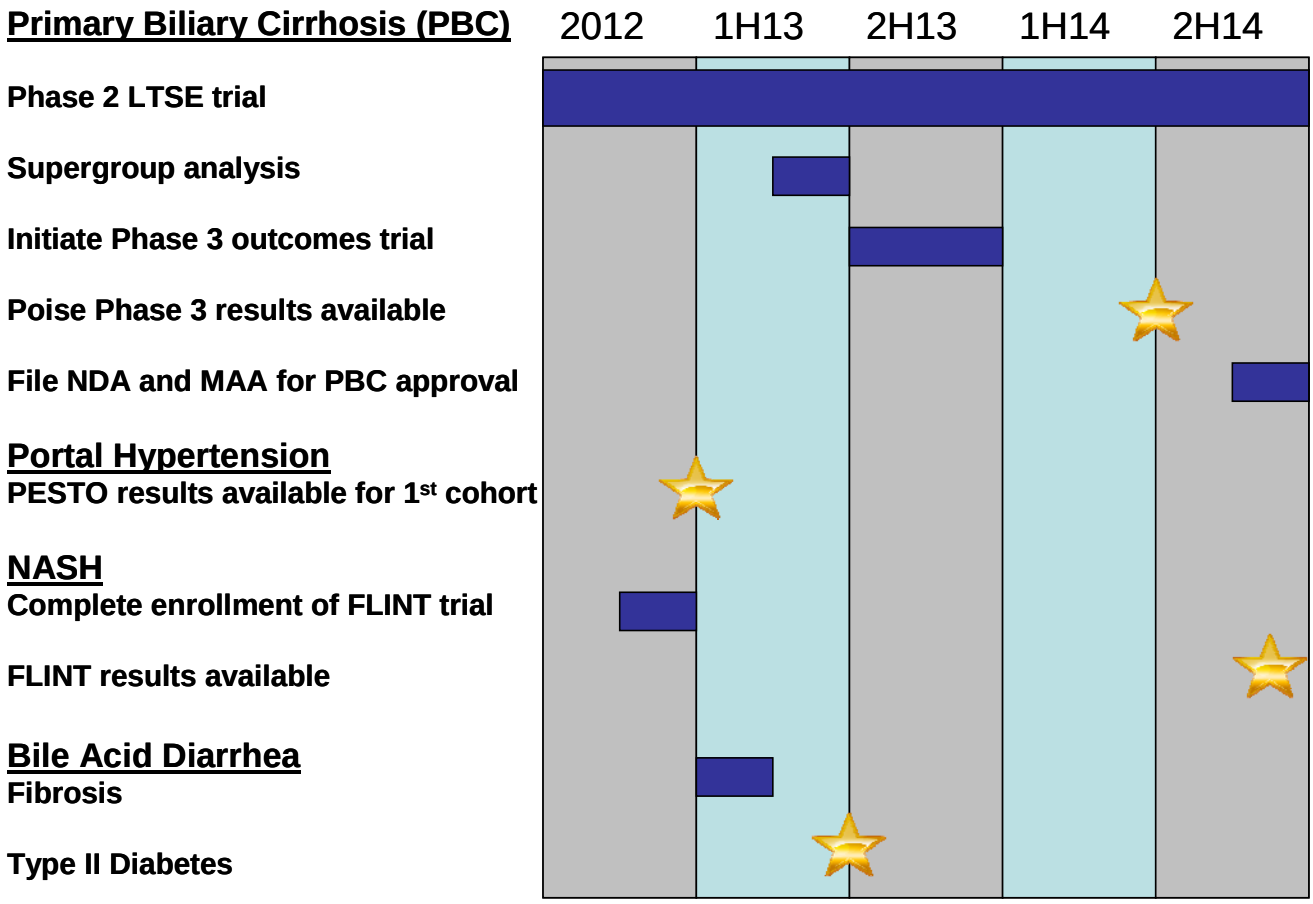
In assessing the potential value associated with our estimates for OCA in PBC we have looked at a probability-adjusted NPV as one metric for assessing fair value. With our estimates, and assuming a 10% discount rate for a commercial stage asset, we have looked at a scenario analysis of varying likelihoods of successfully reaching that commercial stage. Based on our review of preclinical, phase 1 and phase 2 data and with input from key opinion leaders (KOLs) and regulatory experts we believe that the likelihood of successful commercialization in the US and EU is >50%. At an average likelihood of success of 60% for both the US and EU we would estimate a probability-adjusted NPV of $29-$30 per share, and in the range of our $31 per share target price. On successful approval and launch of OCA for PBC we could justify a $49 per share value for ICPT on NPV basis, using our estimates. We would note that experience with other companies transitioning from development stage to commercialization stage is that an inflection in value beyond that predicted by discounted cash flow (DCF) analysis does occur, and that on successful launch stocks do tend to trade higher on relative value metrics related to multiples of earnings and multiples of revenues. Thus, with other orphan disease companies such as Alexion trading at 11-12x forward revenues and with our BMO Capital estimate of 2016 revenues of $140 million one could justify a valuation of $1.5 billion in 2015, assuming a strong launch in 2014.

We believe that programs for OCA in portal hypertension, NAFLD, and NASH, as well as primary bile acid diarrhea represent significant option value beyond PBC and could support

significant incremental upside potential on proof-of-concept data. As with OCA in PBC, the biologic rationale for OCA in other chronic liver diseases and bile acid disorders is strong, preclinical data is compelling and early data for OCA in portal hypertension at this year's AASLD Liver Meeting is encouraging. In the absence of greater visibility on development timelines, specific funding requirements, and strategic positioning of OCA in these other indications, we have not yet developed specific estimates in our model for these indications, but would highlight the fact that each opportunity is potentially several fold larger than the PBC indication.

While Intercept may not garner significant value for its proprietary bile acid analog chemistry and bile acid receptor targeting platform, we believe that this platform provides downside protection against any individual product and will represent a future driver of value as the pipeline matures and advances. Metabolic effects of FXR and TGR5 are incredibly leveragable, in our opinion, and given the intersection of fatty liver, insulin resistance, and lipid abnormalities in patients with metabolic syndrome, we believe that Intercept may be well positioned to target multiple aspects of what is becoming a significant health burden in the US.

Exhibit 20: Dataflow for OCA Clinical Studies



Source: Intercept Pharmaceuticals and BMO Capital Markets.

As with any development stage biotechnology company we believe that data flow will drive share price performance and would highlight a number of important catalysts over the next 12 months, including phase 3 POISE data for OCA in PBC, "Supergroup" data on surrogate endpoints in PBC, initiation of a PBC outcomes study, potential MAA and NDA filing for OCA in PBC, initial bile acid diarrhea data, and updates on progress in portal hypertension and NASH as well as earlier stage programs.

Risks to Intercept Outlook

There are a number of potential risks to our outlook for Intercept, including preclinical risks, clinical risks, regulatory risks, manufacturing risks, commercialization risks, and other risks. These risks are not unique to Intercept but are common to development-stage biotechnology companies and have been considered in our discount rates and probability-adjusted valuation methodology.

In terms of preclinical toxicity risk we would note that two-year carcinogenicity studies are ongoing in rodents and are not yet available. While we understand that OCA is not genotoxic or mutagenic we cannot rule out with certainty that an FXR agonist will not have some cancer signal associated with it preclinically. The natural role of bile acids in normal liver function and the stimulation of FXR by CDCA in humans would seem to argue against any risk to carcinogenicity studies, however, and no excess cancers have been observed in clinical studies as far as we know.

Safety of OCA has been relatively thoroughly vetted across phase 1 and phase 2 studies, with no major adverse events (AEs) noted in excess of placebo with exception of pruritus and mild nausea. That being said, QTc studies and drug interaction studies have yet to report data, and could represent an unanticipated risk to OCA development. We are unaware of any interaction of OCA or other bile acid analogs with hERG channels and as such risk to QTc prolongation would appear to be low, and no risk of arrhythmia or other CV effects have been noted in OCA clinical development to date.

In terms of greater risk to clinical development, we believe that prospective evaluation of a novel composite endpoint in the phase 3 POISE study, over a longer period of time than in phase 2 and in a different group of patients does carry risk, although we would note a relatively significant cushion between composite response rates assessed retrospectively in phase 2 and that required for phase 3 success. These phase 2 data are retrospective and may suffer from a selection bias in terms of evaluating only patients that were able to go on to long-term extension. It should also be considered that other studies of OCA are ongoing in NASH and portal hypertension and visibility on this data is relatively poor relative to that for OCA in PBC.

Regulatory risk is a prominent risk for any development stage biotechnology product and predicting FDA and EMA action is always difficult. Formal scientific advice from EMA and agreement on key aspects of phase 3 trial design for OCA in PBC certainly reduce risk for European approval, but absence of such agreement with FDA does suggest greater risk to approval in the US. There is no guarantee that "Supergroup" data will support the phase 3 composite endpoint of ALP<1.67, 15% reduction in ALP and normal bilirubin as an optimal surrogate endpoint predictive of harder clinical endpoints. Certainly supportive data from large treatment centers contributing to the "Supergroup" analysis strongly suggests that the POISE phase 3 endpoint will predict clinical benefit on harder endpoints like time to transplant and death; however, risk could exist to inclusion of other treatment centers in the analysis. Ultimately this will be a review issue for some future FDA GIDAC panel and will be contemplated in the context of broader safety data.

Finally, in terms of commercialization risk, there is always risk to "finding" appropriate patients for treatment in cases of rare orphan diseases, to gaining adequate reimbursement in terms of what is typically a premium price for patients with rare orphan disease, and for maintaining patients on therapy. Pruritus has been documented as a prominent adverse event with OCA and in patients with PBC in general, and while this is reported as manageable by PBC experts, maintaining patients on OCA will be one of the keys to commercial success. We would note that enrollment of phase 2 studies of OCA took considerable time and this may be an indicator of the difficulty in finding these patients, although once again PBC experts suggest a significant

market opportunity. Patent expiry, potential brand and generic competitor entry, and reliance on third-party manufacturers to supply commercial drug all represent potential risks to our positive outlook for Intercept.

Overview of Intercept Management

CEO, President, and Co-Founder - Mark Pruzanski, M.D.

Dr. Pruzanski co-founded Intercept Pharmaceuticals in 2002 and since that time has served in the role of CEO and president. Dr. Pruzanski also serves on the company's board of directors. Dr. Pruzanski received his M.D. from McMaster University, an M.A. degree in International Affairs from the Johns Hopkins University School of Advanced International Studies, and a Bachelor's degree from McGill University. Dr. Pruzanski has more than 15 years of experience in life sciences company management, venture capital, and strategic consulting, and was previously a venture partner at Apple Tree Partners, an early-stage life sciences venture capital firm he co-founded in 1999. He currently also serves on the boards of the Emerging Company Section of the Biotechnology Industry Association (BIO), and the Foundation for the Defense of Democracies, a think tank in Washington, D.C. Dr. Pruzanski is a co-author of a number of scientific publications and an inventor of several patents related to Intercept Pharmaceuticals product candidates and scientific discoveries.

Chief Medical Officer, Executive Vice President - David Shapiro, M.D.

Dr. Shapiro has served as Intercept Pharmaceuticals' chief medical officer and executive vice president, development since 2008. He has more than 25 years of clinical development experience in the pharmaceutical industry. Dr. Shapiro founded a consulting company, Integrated Quality Resources, that focused on development-stage biopharmaceutical companies and was active in this role from 2005 to 2008. From 2000 to 2005, Dr. Shapiro was executive vice president, medical affairs and chief medical officer of Idun Pharmaceuticals, prior to its acquisition by Pfizer. From 1995 to 1998, he was president of the Scripps Medical Research Center at Scripps Clinic. He also served as vice president, clinical research at Gensia and as director and group leader, hypertension clinical research at Merck Research Laboratories from 1985 to 1990. Dr. Shapiro has authored more than 20 peer-reviewed publications and organized and chaired several conferences aimed at improving product development. He received his medical degree from Dundee University and Medical School, and undertook his postgraduate medical training in the university-affiliated hospitals in Oxford, United Kingdom, and the University of Vermont. Dr. Shapiro served on the board of directors of Altair Therapeutics and served for two terms on the Executive Committee of the Board of the American Academy of Pharmaceutical Physicians. He is an elected fellow of both the Royal College of Physicians of London and the Faculty of Pharmaceutical Physicians of the United Kingdom.

CFO, Treasurer, and Secretary - Barbara Duncan

Ms. Duncan has served as Intercept Pharmaceuticals' chief financial officer and secretary since May 2009 and as treasurer since 2010. Ms. Duncan received her B.S. from Louisiana State University and her M.B.A. from the Wharton School, University of Pennsylvania. She has more than 14 years experience in the life sciences industry. From 2001 through April 2009, Ms. Duncan served as chief financial officer and then chief executive officer at DOV Pharmaceutical, which was sold to Euthymics Bioscience in 2010. Prior to joining DOV, Ms. Duncan served as a vice president of Lehman Brothers in its corporate finance division from August 1998 to August 2001, where she provided financial advisory services primarily to companies in the life sciences and general industrial industries. From September 1994 to August 1998, Ms. Duncan was an associate and director at SBC Warburg Dillon Read, Inc. in its corporate finance group, where she focused primarily on structuring mergers, divestitures, and financings for companies in the life sciences and general industrial industries. She also worked for PepsiCo, Inc. from 1989 to 1992 in its international audit division, and was a certified public accountant in the audit division of Deloitte & Touche LLP from 1986 to 1989. She previously served as a director of DOV and currently serves on the board of directors of Edgemont Pharmaceuticals, LLC, a privately held, specialty pharmaceutical company with a primary focus in the field of neuroscience.

Chief Scientific Officer - Luciano Adorini, M.D.

Dr. Adorini has served as Intercept Pharmaceuticals' chief scientific officer since 2008. Dr. Adorini received his M.D. degree from the Medical School of Padova University and conducted postdoctoral studies at the University of California at Los Angeles. Dr. Adorini has more than 20 years of industry experience. From January 2002 through December 2007, Dr. Adorini served as chief scientific officer at BioXell S.p.A. From January 1993 to December 2001, he served as associate director of Roche Milano Richerche, where he contributed to the development of several drugs. Prior to that, Dr. Adorini was research director of a unit at the Preclinical Research Center, Sandoz Pharma, Ltd., in Basel, Switzerland. Dr. Adorini has authored more than 280 journal articles and other scientific publications, becoming a highly cited researcher in immunology, with a focus on immunosuppressive and immunoregulatory mechanisms in the treatment of inflammatory and autoimmune diseases. He is a board member of a number of peer-reviewed publications and has served as president of the Italian Society of Immunology, Clinical Immunology, and Allergology.

Other companies mentioned (priced as of the close on November 2, 2012):

Alexion (ALXN, $89.09, Not Rated)
Dainippon Pharmaceutical (DNPUF-5, $10.63, Not Rated)
Merck (MRK, $46.00, **MARKET PERFORM**, by Alex Arfaei)
Novartis (NVS, $60.66, Not Rated)
Pepsico (PEP, $69.05, Not Rated)
Pfizer (PFE, $24.55, **OUTPERFORM**, by Alex Arfaei)
Roche (RHHBY, $48.30, Not Rated)

ICPT Income Statement 2011A-2020E

INCOME STATEMENT ($M)	2011A	2012E	2013E	2014E	2015E	2016E	2017E	2018E	2019E	2020E
REVENUES										
Product Revenues	$ -	$ -	$ -	$ 5.8	$ 58.9	$ 128.2	$ 180.7	$ 231.1	$ 282.3	$ 334.5
Licensing Revenue	1.8	8.1	13.2	13.2	13.2	13.2	13.2	13.2	13.2	13.2
Grant revenue and other	-	-	-	-	-	-	-	-	-	-
TOTAL REVENUES	**$ 1.8**	**$ 8.1**	**$ 13.2**	**$ 19.0**	**$ 72.1**	**$ 141.4**	**$ 193.9**	**$ 244.3**	**$ 295.5**	**$ 347.7**
EXPENSES (GAAP)										
Cost of Goods Sold (COGS)	$ -	$ -	$ -	$ 0.3	$ 3.0	$ 6.4	$ 9.0	$ 11.6	$ 14.1	$ 16.7
R&D Expense	11.4	17.8	21.0	29.4	26.8	30.0	33.2	36.4	39.6	42.8
SG&A Expense	4.2	6.5	15.0	28.0	41.0	46.0	49.2	52.4	55.6	58.8
Other	-	-	-	-	-	-	-	-	-	-
TOTAL EXPENSES	15.6	24.3	36.0	51.9	70.8	82.4	91.4	100.4	109.3	118.3
Operating Income	**(13.8)**	**(16.2)**	**(22.8)**	**(32.9)**	**1.3**	**59.0**	**102.5**	**143.9**	**186.2**	**229.4**
Depreciation and amortization	-	-	-	-	-	-	-	-	-	-
EBIT	(13.8)	(16.2)	(22.8)	(32.9)	1.3	59.0	102.5	143.9	186.2	229.4
Interest and other income	0.1	0.1	0.1	0.1	0.0	0.0	0.2	0.3	0.6	0.9
Interest and other expense	(0.0)	(0.0)	-	-	-	-	-	-	-	-
Other Income (Expense)	(2.0)	(0.7)	-	-	-	-	-	-	-	-
Interest and Other Income (Expense)	(1.9)	(0.7)	0.1	0.1	0.0	0.0	0.2	0.3	0.6	0.9
Pre-Tax Income	(15.7)	(16.8)	(22.7)	(32.9)	1.3	59.1	102.7	144.3	186.8	230.3
Income Taxes	-	-	-	-	2.2	16.5	28.7	40.4	52.3	64.5
Net Income (GAAP)	**$ (15.7)**	**$ (16.8)**	**$ (22.7)**	**$ (32.9)**	**$ (0.8)**	**$ 42.5**	**$ 73.9**	**$ 103.9**	**$ 134.5**	**$ 165.8**
EPS (GAAP) (basic)	$ (1.17)	$ (1.16)	$ (1.17)	$ (1.70)	$ (0.04)	$ 2.01	$ 3.27	$ 4.58	$ 5.91	$ 7.26
EPS (GAAP) (diluted)	**$ (1.17)**	**$ (1.16)**	**$ (1.17)**	**$ (1.70)**	**$ (0.04)**	**$ 2.01**	**$ 3.27**	**$ 4.58**	**$ 5.91**	**$ 7.26**
Total of Reconciliation Items	-	4.0	8.0	8.0	8.0	8.0	8.0	8.0	8.0	8.0
Net Income (Non-GAAP)	$ (7.8)	$ (12.8)	$ (14.7)	$ (24.9)	$ 7.2	$ 50.5	$ 81.9	$ 111.9	$ 142.5	$ 173.8
Impact of Adjustments to EPS	-	0.25	0.41	0.41	0.41	0.38	0.35	0.35	0.35	0.35
EPS (Non-GAAP) (basic)	$ (0.57)	$ (0.90)	$ (0.76)	$ (1.28)	$ 0.37	$ 2.39	$ 3.63	$ 4.94	$ 6.26	$ 7.61
EPS (Non-GAAP) (diluted)	$ (0.57)	$ (0.90)	$ (0.76)	$ (1.28)	$ 0.37	$ 2.39	$ 3.63	$ 4.94	$ 6.26	$ 7.61
Weighted average shares outstanding (basic)	13.5	14.9	19.3	19.4	19.4	21.0	22.6	22.7	22.8	22.8
Weighted average shares outstanding (diluted)	13.5	14.9	19.3	19.4	19.4	21.0	22.6	22.7	22.8	22.8

Source: Company reports and BMO Capital Markets.

ICPT Balance Sheet 2011A-2020E

BALANCE SHEET ($M)	2011A	2012E	2013E	2014E	2015E	2016E	2017E	2018E	2019E	2020E
Current Assets										
Cash and cash equivalents	$ 17.7	$ 79.0	$ 52.2	$ 15.1	$ 10.1	$ 48.5	$ 118.2	$ 217.9	$ 348.2	$ 509.8
Short-term investments	0.2	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1
Total cash, cash equivalents, and short-term investments	$ 17.9	$ 79.1	$ 52.2	$ 15.2	$ 10.2	$ 48.5	$ 118.3	$ 218.0	$ 348.3	$ 509.9
Accounts Receivables	-	-	-	-	-	-	-	-	-	-
Restricted Cash	-	-	-	-	-	-	-	-	-	-
Inventories	-	-	-	-	-	-	-	-	-	-
Prepaid and other current assets	1.0	1.7	1.7	1.7	1.7	1.7	1.7	1.7	1.7	1.7
Total Current Assets	$ 18.9	$ 80.8	$ 53.9	$ 16.9	$ 11.9	$ 50.2	$ 119.9	$ 219.6	$ 349.9	$ 511.5
Leasehold improvements	-	-	-	-	-	-	-	-	-	-
Property and equipment, net	0.3	2.3	6.5	10.6	14.8	19.0	23.2	27.4	31.6	35.7
Patents and licensed technology	-	-	-	-	-	-	-	-	-	-
Intangibles, net	-	-	-	-	-	-	-	-	-	-
Security deposits and other assets	0.3	0.3	0.3	0.3	0.3	0.3	0.3	0.3	0.3	0.3
TOTAL ASSETS	**$ 19.5**	**$ 83.3**	**$ 60.6**	**$ 27.8**	**$ 27.0**	**$ 69.5**	**$ 143.4**	**$ 247.3**	**$ 381.8**	**$ 547.5**
Current Liabilities										
Accounts payable	1.5	3.6	3.6	3.6	3.6	3.6	3.6	3.6	3.6	3.6
Accrued payroll	-	-	-	-	-	-	-	-	-	-
Accrued expenses	-	-	-	-	-	-	-	-	-	-
Accrued interest	-	-	-	-	-	-	-	-	-	-
Payables to related parties	-	-	-	-	-	-	-	-	-	-
Income taxes payable	-	-	-	-	-	-	-	-	-	-
Short-term portion of warrant liability	-	0.3	0.3	0.3	0.3	0.3	0.3	0.3	0.3	0.3
Short-term portion of deferred revenue	2.4	1.7	1.7	1.7	1.7	1.7	1.7	1.7	1.7	1.7
Short-term portion of capital leases	0.1	-	-	-	-	-	-	-	-	-
Other current liabilities	-	-	-	-	-	-	-	-	-	-
Total Current Liabilities	$ 4.0	$ 5.6	$ 5.6	$ 5.6	$ 5.6	$ 5.6	$ 5.6	$ 5.6	$ 5.6	$ 5.6
Convertible notes payable	-	-	-	-	-	-	-	-	-	-
Accrued interest on convertible notes payable	-	-	-	-	-	-	-	-	-	-
Long-term portion of deferred revenue	12.2	11.4	11.4	11.4	11.4	11.4	11.4	11.4	11.4	11.4
Long-term portion of warrant liability	5.8	4.6	4.6	4.6	4.6	4.6	4.6	4.6	4.6	4.6
Long-term portion of capital leases	-	-	-	-	-	-	-	-	-	-
Other liabilities	-	-	-	-	-	-	-	-	-	-
TOTAL LIABILITIES	$ 22.0	$ 21.5	$ 21.5	$ 21.5	$ 21.5	$ 21.5	$ 21.5	$ 21.5	$ 21.5	$ 21.5
Shareholder's Equity										
Series A preferred stock	0.0	-	-	-	-	-	-	-	-	-
Series B preferred stock	0.0	-	-	-	-	-	-	-	-	-
Common stock, at par	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Additional paid-in capital	72.1	151.6	151.6	151.6	151.6	151.6	151.6	151.6	151.6	151.6
Deferred compensation	-	-	-	-	-	-	-	-	-	-
Notes receivable from shareholders	-	-	-	-	-	-	-	-	-	-
Unrealized gains (losses) on short-term investments	-	-	-	-	-	-	-	-	-	-
Accumulated other comprehensive income	(0.2)	-	-	-	-	-	-	-	-	-
Accumulated deficit	(74.5)	(89.9)	(112.5)	(145.4)	(146.2)	(103.7)	(29.8)	74.1	208.6	374.4
TOTAL SHAREHOLDER'S EQUITY (DEFICIT)	$ (2.6)	$ 61.8	$ 39.1	$ 6.3	$ 5.4	$ 48.0	$ 121.9	$ 225.7	$ 360.2	$ 526.0
TOTAL LIABILITIES AND SHAREHOLDER'S EQUITY	**$ 19.5**	**$ 83.3**	**$ 60.6**	**$ 27.8**	**$ 27.0**	**$ 69.5**	**$ 143.4**	**$ 247.3**	**$ 381.8**	**$ 547.5**

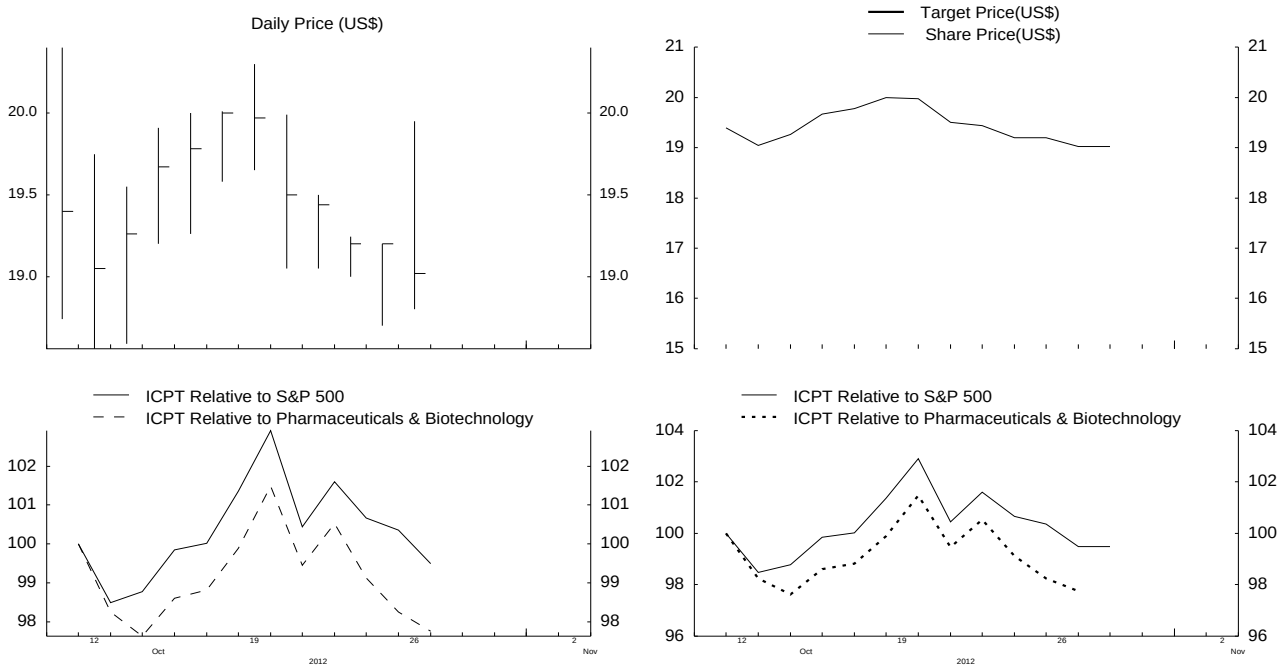
Source: Company reports and BMO Capital Markets.

ICPT Cash Flow Statement 2011A-2020E

CASH FLOW STATEMENT (M)	2011A	2012E	2013E	2014E	2015E	2016E	2017E	2018E	2019E	2020E
Cash Flow From Operating Activities										
Net Income	$ (12.7)	$ (4.1)	$ (6.6)	$ (7.3)	$ 4.0	$ 13.8	$ 21.2	$ 28.7	$ 36.4	$ 44.2
Adjustments to reconcile net income to cash from operations										
Loss from sale of assets	$ 0.2	$ -	$ -	$ -	$ -	$ -	$ -	$ -	$ -	$ -
Depreciation & amortization	0.4	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2
Foreign currency loss on liquidation	-	-	-	-	-	-	-	-	-	-
Stock-based compensation	1.9	-	-	-	-	-	-	-	-	-
Revaluation of warrants	(1.0)	(1.0)	(1.0)	(1.0)	(1.0)	(1.0)	(1.0)	(1.0)	(1.0)	(1.0)
Deferred income taxes	-	-	-	-	-	-	-	-	-	-
Other	-	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0
Working Capital Adjustments										
Prepaids and other assets	(0.6)	-	-	-	-	-	-	-	-	-
Accounts payable	(0.1)	-	-	-	-	-	-	-	-	-
Accrued payroll	-	-	-	-	-	-	-	-	-	-
Accrued expenses	-	-	-	-	-	-	-	-	-	-
Accrued interest	-	-	-	-	-	-	-	-	-	-
Payables to related parties	-	-	-	-	-	-	-	-	-	-
Income taxes payable	-	-	-	-	-	-	-	-	-	-
Deferred revenue	14.6	-	-	-	-	-	-	-	-	-
Deferred rent	-	-	-	-	-	-	-	-	-	-
Other assets and liabilities, net	-	-	-	-	-	-	-	-	-	-
Total Working Capital Increase (Decrease)	$ 13.9	$ -	$ -	$ -	$ -	$ -	$ -	$ -	$ -	$ -
TOTAL CASH FROM OPERATIONS	**$ 2.6**	**$ (3.9)**	**$ (6.4)**	**$ (7.1)**	**$ 4.2**	**$ 13.9**	**$ 21.3**	**$ 28.8**	**$ 36.5**	**$ 44.4**
Cash From Investing Activities										
Purchases of short-term investments	-	-	-	-	-	-	-	-	-	-
Maturities and sales of short-term investments	-	-	-	-	-	-	-	-	-	-
Purchases of property and equipment	(0.1)	-	-	-	-	-	-	-	-	-
Acquisitions of patents	-	-	-	-	-	-	-	-	-	-
Acquisitions of licenses	-	(1.2)	(1.2)	(1.2)	(1.2)	(1.2)	(1.2)	(1.2)	(1.2)	(1.2)
Increase in patents, deposits and other assets	(0.0)	-	-	-	-	-	-	-	-	-
TOTAL CASH FROM INVESTING	$ (0.1)	$ (1.2)	$ (1.2)	$ (1.2)	$ (1.2)	$ (1.2)	$ (1.2)	$ (1.2)	$ (1.2)	$ (1.2)
Cash From Financing Activities										
Proceeds from issuance of common stock	-	-	-	-	-	-	-	-	-	-
Proceeds from issuance of preferred stock	-	-	-	-	-	-	-	-	-	-
Proceeds from issuance of common stock warrants	-	-	-	-	-	-	-	-	-	-
Costs associated with issuance of stock	-	-	-	-	-	-	-	-	-	-
Payments of capital lease obligation	(0.2)	-	-	-	-	-	-	-	-	-
Proceeds from exercise of options	-	-	-	-	-	-	-	-	-	-
Proceeds from exercise of warrants	-	-	-	-	-	-	-	-	-	-
Proceeds from issuance of convertible promissory notes payable	-	-	-	-	-	-	-	-	-	-
Other	-	-	-	-	-	-	-	-	-	-
TOTAL CASH FROM FINANCING	$ (0.2)	$ -	$ -	$ -	$ -	$ -	$ -	$ -	$ -	$ -
Effect of exchange rate changes	(0.0)	-	-	-	-	-	-	-	-	-
Increase (decrease) in cash and cash equivalents	2.3	(5.1)	(7.6)	(8.3)	3.0	12.7	20.1	27.6	35.3	43.2
Cash and cash equivalents at beginning of year	15.4	84.1	59.8	23.4	7.1	35.7	98.1	190.2	312.9	466.6
Cash and cash equivalents at end of year	$ 17.7	$ 79.0	$ 52.2	$ 15.1	$ 10.1	$ 48.5	$ 118.2	$ 217.9	$ 348.2	$ 509.8

Source: Company reports and BMO Capital Markets.

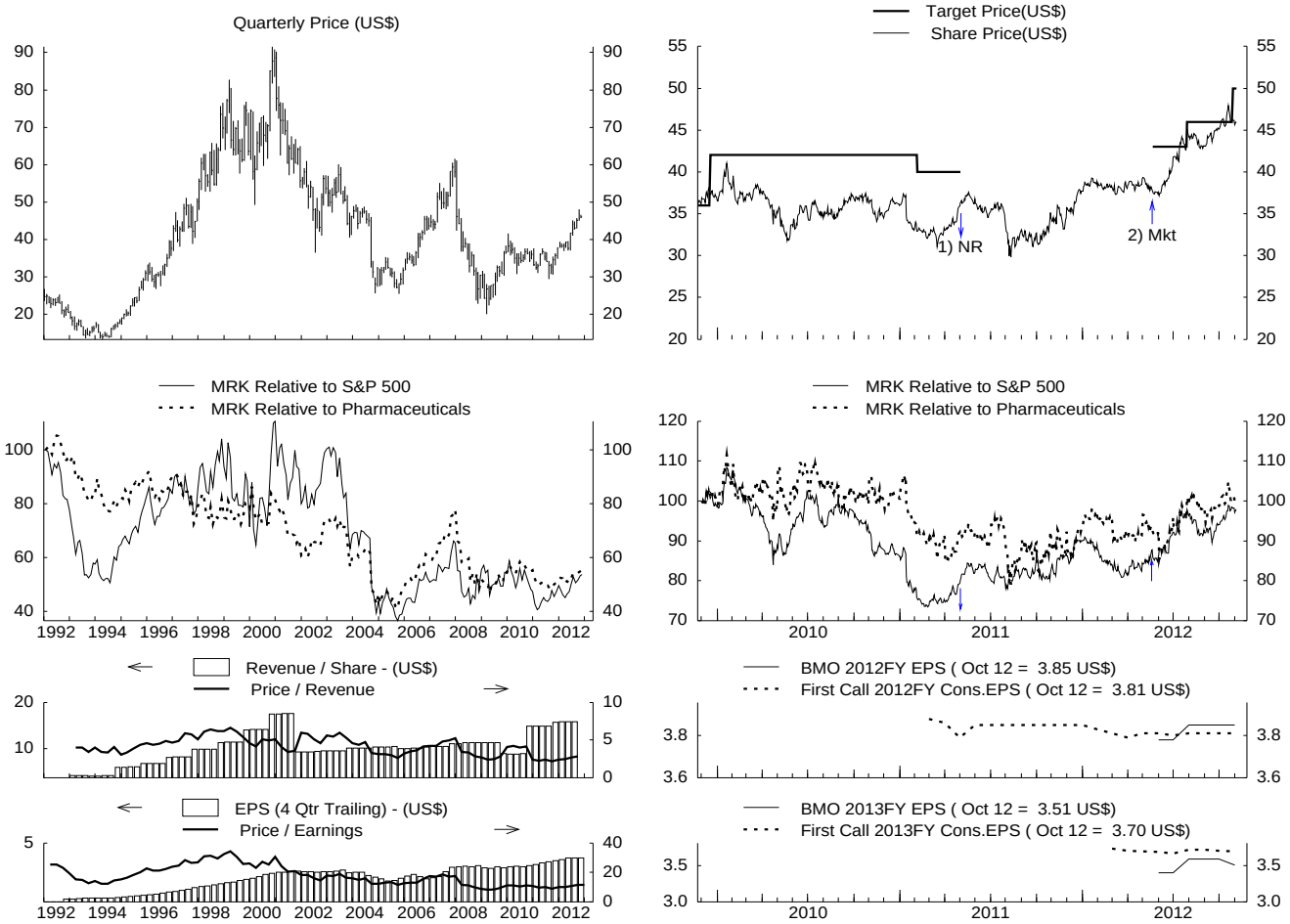
Intercept Phamaceuticals (ICPT)



ICPT - Rating as of 22-Oct-12 = NR

Last Daily Data Point: October 29, 2012

Merck & Co. Inc. (MRK)



FYE (Dec.)	EPS US$	P/E	DPS US$	Yield %	Payout %	BV US$	P/B	ROE %
1992	1.06	19	0.00	0.0	0	2.19	9.4	nm
1993	1.17	14	0.53	3.3	45	4.00	4.1	38
1994	1.19	15	0.57	3.1	47	4.46	4.0	28
1995	1.35	23	0.64	2.1	47	4.78	6.5	29
1996	1.56	24	0.76	2.0	48	4.96	7.6	32
1997	1.87	27	0.85	1.7	45	5.28	9.5	37
1998	2.15	32	1.02	1.5	47	5.42	nm	40
1999	2.45	26	1.10	1.7	45	5.69	nm	44
2000	2.90	31	1.29	1.5	45	6.43	nm	nm
2001	3.04	18	1.33	2.4	44	7.06	7.9	nm
2002	2.98	18	1.36	2.5	46	8.11	6.6	39
2003	2.92	16	1.48	3.2	51	7.01	6.6	39
2004	2.62	12	1.52	4.7	58	7.83	4.1	35
2005	2.53	13	1.52	4.8	60	8.21	3.9	32
2006	2.52	17	1.52	3.5	60	8.10	5.4	31
2007	3.31	18	1.52	2.6	46	8.37	6.9	40
2008	3.42	9	1.52	5.0	44	8.90	3.4	40
2009	3.26	11	1.52	4.2	47	19.00	1.9	23
2010	3.42	11	1.52	4.2	44	17.64	2.0	19
2011	3.77	10	1.68	4.5	44	17.93	2.1	21
Current*	3.96	12	1.68	3.7	42	17.93	2.5	22
Average:		18		3.1	48		5.2	34.7
Growth(%):								
5 Year:	5.6		2.0			17.2		
10 Year:	2.9		2.1			9.8		
20 Year:	7.6		nm			11.3		

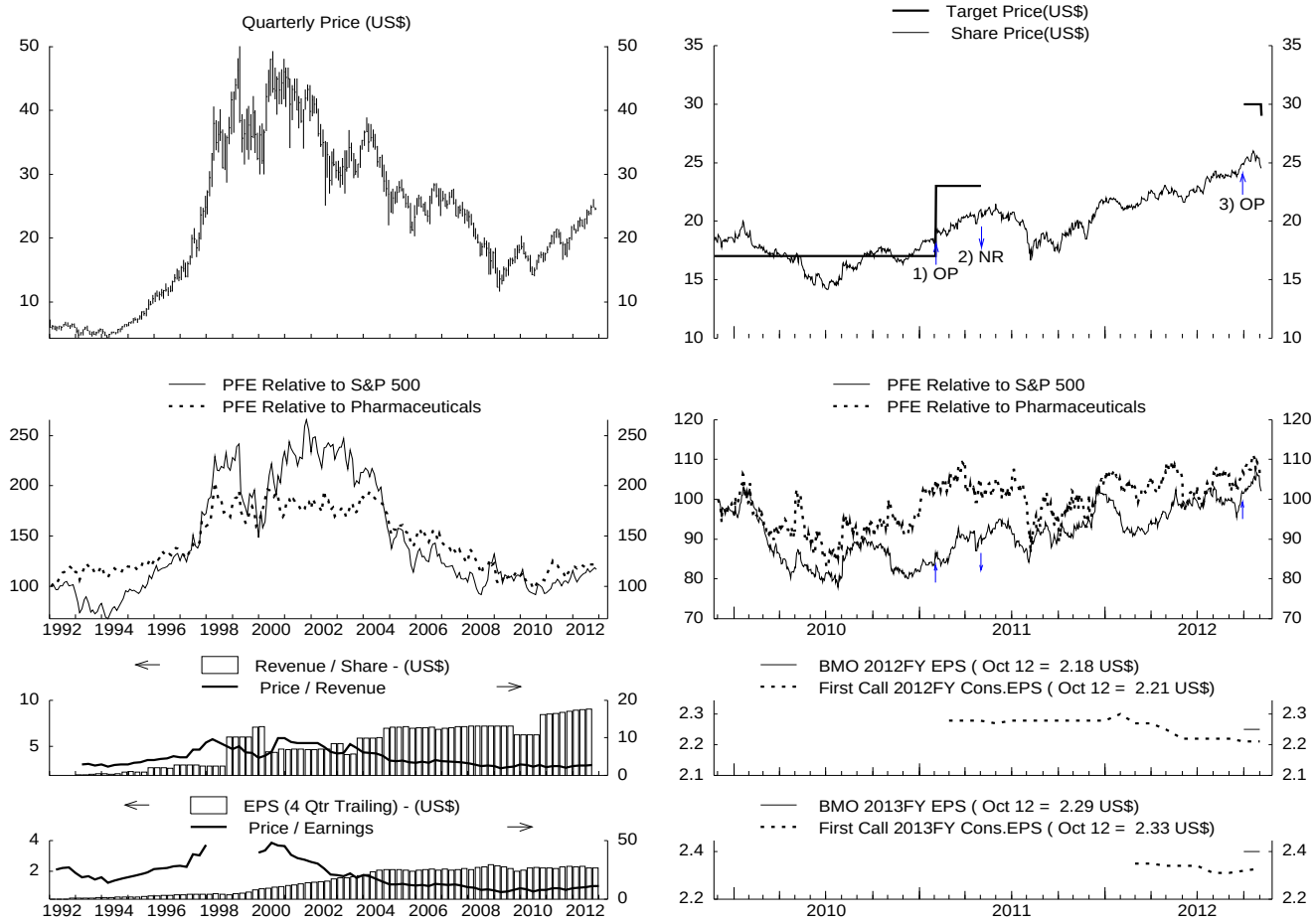
* Current EPS is the 4 Quarter Trailing to Q3/2012.

MRK - Rating as of 24-Nov-09 = OP

	Date	Rating Change	Share Price
1	3-May-11	OP to NR	$36.41
2	18-May-12	NR to Mkt	$37.82

Last Price (November 2, 2012): $46.00
Sources: IHS Global Insight, Thomson Reuters, BMO Capital Markets.

Pfizer Inc. (PFE)



FYE (Dec.)	EPS US$	P/E	DPS US$	Yield %	Payout %	BV US$	P/B	ROE %
1992	0.27	22	0.00	0.0	0	1.21	5.0	22
1993	0.31	19	0.14	2.4	45	1.00	5.7	28
1994	0.35	18	0.16	2.4	45	1.15	5.6	33
1995	0.41	26	0.09	0.8	21	1.44	7.3	32
1996	0.50	28	0.20	1.4	41	1.80	7.7	31
1997	0.53	46	0.23	0.9	42	2.04	nm	28
1998	0.51	nm	0.29	0.7	58	2.27	nm	24
1999	0.82	40	0.36	1.1	44	2.31	nm	36
2000	1.02	45	0.44	1.0	43	2.55	nm	42
2001	1.27	32	0.52	1.3	41	2.91	nm	nm
2002	1.53	20	0.60	2.0	40	3.24	9.4	nm
2003	1.75	20	0.68	1.9	39	8.54	4.1	30
2004	2.12	13	0.76	2.8	36	9.11	3.0	24
2005	2.02	12	0.96	4.1	48	8.89	2.6	22
2006	2.08	12	1.16	4.5	56	10.00	2.6	22
2007	2.20	10	1.28	5.6	58	9.60	2.4	22
2008	2.43	7	1.28	7.2	53	8.52	2.1	27
2009	2.02	9	0.72	4.0	36	11.15	1.6	21
2010	2.22	8	0.80	4.6	36	10.95	1.6	20
2011	2.31	9	0.88	4.1	38	10.84	2.0	21
Current*	2.23	11	0.88	3.5	39	10.84	2.3	21
Average:		21		2.8	43		4.5	29.4
Growth(%):								
5 Year:	1.1		-5.4			1.6		
10 Year:	4.8		5.4			14.1		
20 Year:	12.0		nm			11.1		

* Current EPS is the 4 Quarter Trailing to Q3/2012.

PFE - Rating as of 24-Nov-09 = Mkt

	Date	Rating Change	Share Price
1	2-Feb-11	Mkt to OP	$18.96
2	3-May-11	OP to NR	$20.44
3	27-Sep-12	NR to OP	$24.96

Last Price (November 2, 2012): $24.55
Sources: IHS Global Insight, Thomson Reuters, BMO Capital Markets.

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Methodology: We arrive at our price target by applying a 20x multiple to 2018 EPS estimate of $4.58 discounted at 30%.
Risks: There are a number of risks associated with investment in biotechnology companies. These risks include, but are not limited to, risk of clinical trial delay or failure, adverse regulatory decisions including product non-approval, unanticipated adverse effects of drugs which may result in removal from market, risk of manufacturing difficulties, capital market risk which may impair the ability to fund product discovery, research, regulatory filing, manufacture and/or commercialization, risk in attaining and retaining appropriate development or commercial partners, lower-than-expected product adoption, difficulties in gaining appropriate reimbursement for products from payors, unforeseen generic and branded competition, risk to patents being invalidated, and failure to meet earnings and revenue expectations.

Company Specific Disclosures for MRK

9 - BMO Capital Markets makes a market in this security.

Methodology and Risks to Our Price Target/Valuation

Methodology: DCF: Free Cash Flow to Equity and P/E multiple.
Risks: Uncertainty of Phase 3 Pipeline.

Company Specific Disclosures for PFE

9 - BMO Capital Markets makes a market in this security.

Methodology and Risks to Our Price Target/Valuation

Methodology: DCF: Free Cash Flow to Equity.
Risks: Uncertainty of Phase 3 Pipeline.

Distribution of Ratings (September 30, 2012)

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Hold	Market Perform	58.5%	7.9%	39.1%	56.4%	48.5%	40.3%
Sell	Underperform	3.1%	11.8%	3.1%	4.6%	2.0%	5.3%

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