June 20, 2013

HEALTHCARE/BIO AND SPECIALTY PHARMACEUTICALS

Stock Rating:

OUTPERFORM

12-18 mo. Price Target	$60.00
ICPT - NASDAQ	$38.41

3-5 Yr. EPS Gr. Rate	NA
52-Wk Range	$42.67-$15.00
Shares Outstanding	20.7M
Float	7.0M
Market Capitalization	$714.5M
Avg. Daily Trading Volume	34,646
Dividend/Div Yield	NA/NM
Book Value	$3.66
Fiscal Year Ends	Dec
2013E ROE	NA
LT Debt	NA
Preferred	NA
Common Equity	$66M
Convertible Available	No

EPS Diluted	Q1	Q2	Q3	Q4	Year	Mult.
2011A	--	--	--	--	(4.73)	NM
2012A	(1.03)	(1.75)	(1.86)	(2.02)	(7.36)	NM
2013E	(0.62)A	(0.42)	(0.39)	(0.40)	(1.83)	NM
2014E	--	--	--	--	(1.92)	NM
2015E	--	--	--	--	(0.43)	NM

Intercept Pharmaceuticals

Bullish on PBC and Follow-On Indications; Initiating at Outperform With $60 PT

SUMMARY

We are initiating coverage of Intercept (ICPT) with an Outperform rating and $60 price target. We believe Intercept is emerging as a leader in liver disease therapies, with obeticholic acid (OCA) likely to deliver positive Phase 3 results in 2Q14 for the treatment of Primary Biliary Cirrhosis (PBC), an orphan indication with a strong unmet medical need. Meanwhile, smaller proof-of-concept studies in a number of follow-on indications provide the opportunity for further share appreciation, in our view. Our bullish stance on ICPT is based on anticipated success in the Phase 3 PBC trial as well as our expectation that the shares will move higher as investors begin to assign value to OCA's follow-on indications.

KEY POINTS

- Intercept's primary asset is OCA, currently in a Phase 3 study in PBC (POISE trial), with data less than a year away (2Q14). Prior Phase 2 studies demonstrated very positive results, in our view, and we are optimistic that the POISE study will be successful.
- PBC is a well-defined orphan disease opportunity with a clear unmet medical need. We see relatively low commercialization risk as well as potential upside to our pricing estimates. We estimate peak worldwide sales of $650M for OCA in PBC.
- We see additional upside in ICPT as investors become more familiar with OCA's opportunities in Bile-Acid Diarrhea and Portal Hypertension following the release of additional proof-of-concept data later this year. We believe OCA's broad therapeutic potential positions the drug to be a pipeline within itself.
- Key upcoming milestones include additional Phase 2 data updates in portal hypertension and bile-acid diarrhea later this year followed by top-line results from Phase 3 POISE study in 2Q14.
- Our $60 PT is based on a sum-of-parts analysis, assigning $40/share for PBC, $11/share for portal hypertension and $9/share for bile-acid diarrhea. Although ICPT has mostly traded sideways YTD (following strong post-IPO performance last year), we believe the approaching milestones position the stock to outperform during 2H13 and 2014.

Stock Price Performance

Company Description

Intercept is a biopharmaceutical company focused on the development of novel treatments for liver diseases. Lead drug OCA is in Phase 3 for the treatment of Primary Biliary Cirrhosis.

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Investment Thesis

We are initiating coverage of Intercept Pharmaceuticals (ICPT) with an Outperform rating and a 12- to 18-month price target of $60. We believe Intercept is well positioned as a leading drug developer for the treatment of liver diseases. The company's lead asset, obeticholic acid (OCA), is set to report Phase 3 results in 2Q14 for the treatment of Primary Biliary Cirrhosis (PBC), an orphan indication with a strong unmet medical need. Meanwhile, smaller proof-of-concept studies in a number of follow-on indications should provide the opportunity for further share appreciation, in our view. Our bullish stance on the company is based on our positive expectations for OCA's Phase 3 POISE trial, regulatory approval, well-defined commercial potential and an overall risk/reward profile that appears highly favorable at the current share price.

- **OCA's Phase 3 study in Primary Biliary Cirrhosis is de-risked by strong results demonstrated in prior Phase 2 studies.** Intercept's expertise in bile-acid therapies led to the identification of OCA as a promising therapy for the treatment of a rare liver disease called PBC. Intercept has completed two Phase 2 studies of OCA in PBC, which demonstrated highly statistically significant results and a favorable safety profile. The company is currently conducting a Phase 3 study in PBC, entitled POISE, expected to report data in less than 12 months (2Q14). Based on our analysis of the Phase 2 results, we believe the POISE study is likely to succeed.

- **PBC is a well-defined orphan disease opportunity with a clear unmet medical need.** An autoimmune-mediated disease, PBC primarily affects adult women. Persons with PBC often exhibit symptoms of extreme fatigue and/or pruritis (itching), but even more worrisome is the high risk of liver failure leading to death or the need for a transplant. The current standard of care for PBC is the generic drug ursodeoxycholic acid (typically referred to as Urso). Although most patients diagnosed with PBC receive Urso therapy, the drug is not sufficiently effective in about 40-50% of patients, who continue to remain at high risk of liver failure and death. The shortcomings of Urso present a clear unmet medical need for the treatment of PBC. Because these patients are already identified and receiving treatment, we believe the market opportunity for OCA is relatively well-defined compared to other orphan diseases, which reduces commercial risk.

- **We believe Intercept will be successful in seeking accelerated approval for PBC.** Intercept currently intends to file for accelerated approval in the US based on the results of the Phase 3 POISE study. This approach adds a degree of regulatory risk as the company will attempt to secure approval based on a surrogate marker of PBC disease activity (reduction in alkaline phosphatase [ALP] enzyme levels). However, given the impracticality of conducting an outcomes study in PBC as well as a more-favorable regulatory environment at the FDA with regard to orphan diseases, we believe that Intercept will be able to secure accelerated approval without significant controversy. Our optimism here is boosted by a recent "supergroup" analysis of thousands of PBC patients that demonstrates the link between the ALP biomarker and risk of death. We expect incremental clarity later this year after the company receives additional feedback from the FDA (although a definitive yes/no answer is unlikely, in our view). Specifically, we believe that the initiation of a confirmatory outcomes study, expected in 4Q13, would signal that the company is increasingly confident with the FDA's stance on accelerated approval.

- **We expect OCA approval in PBC in mid-2015 and model peak worldwide sales of $650M in 2025.** For the US, our estimates assume peak OCA penetration of 60% in patients not responding to first-line therapy with Urso (an estimated 14-15,000 total patients available for treatment) and initial pricing of $40,000 annually. We believe our pricing estimate may err on the conservative side given comparable pricing of innovative life-saving drugs that treat rare diseases.

- **OCA development in other liver diseases provides additional news flow and represents significant upside potential for ICPT.** OCA's unique mechanism of action leads to the potential for the drug to be used in a variety of liver diseases related to bile-acid dysfunction and hepatic fibrosis. Although the data are early, results in initial studies of OCA for portal hypertension and bile-acid diarrhea appear promising. Meanwhile, an ongoing Phase 2 study in NASH is expected to read out in late-2014. With no currently approved therapies for these diseases, we believe follow-on indications of OCA offer the potential for significant future value-inflection points upon the release positive data. In short, we are encouraged by the possibility for OCA to develop into a "pipeline" within itself, and believe this potential is not currently reflected in ICPT shares.

- **In our view, ICPT shares are currently trading at an attractive valuation with a favorable risk/reward profile. We expect the shares to appreciate in 2H2013-2014 in conjunction with the POISE study results and as investors begin to assign value to follow-on indications as Intercept moves these programs into later-stage development.**

  Please see our initiation of Amarin Corp. (Perform), and our Company Reports on Antares (Outperform), The Medicines Company (assuming at Perform from prior Outperform), Questcor (assuming at Outperform from prior Perform), and Zogenix (Perform), also released today.

Overview

Intercept Pharmaceuticals, Inc. is a biopharmaceutical company specializing in developing novel drugs that target the hepatic system (the liver). The company's lead development program is the drug OCA (formerly known as INT-747) for the treatment of Primary Biliary Cirrhosis (PBC), a potentially serious disease that can lead to liver failure and death. Intercept's development pipeline also consists of OCA for the treatment of other liver-related indications such as NASH, portal hypertension and bile-acid diarrhea, as well as preclinical programs for the treatment of fibrosis (INT-767) and Type-2 diabetes (INT-777).

Exhibit 1: ICPT Clinical Development Pipeline

Product	Indication	Clinical Stage				Status
		Preclinical	Phase I	Phase II	Phase III	
OCA	Primary Biliary Cirrhosis					▪Anticipate topline data from pivotal Phase 3 POISE study in 2Q14
OCA	Portal Hypertension					▪Anticipate updated data from phase 2a trial (PESTO study) in 2H13
OCA	Bile Acid Diarrhea					▪Anticipate updated data from phase 2a trial OBADIAH study) in 2H13
OCA	NASH					▪Anticipate updated data from phase 2a trial (FLINT study) in 4Q14
INT-767	Fibrosis					
INT-777	Type-2 Diabetes					▪Expect Intercept to look to out-license INT-777

Source: Company Documents

Intercept's lead drug is called obeticholic acid, commonly referred to by the abbreviation OCA. OCA is a bile acid analogue and an agonist of FXR (farnesoid X receptor), a receptor associated in a number of liver-related diseases. OCA was first described in 2002 by an academic laboratory at the University of Perugia in Italy, the head of which co-founded Intercept along with the company's current CEO.

Although the precise etiology is not fully known, PBC is a chronic liver disease resulting from an autoimmune response against the bile ducts. This can lead to cirrhosis and liver failure, causing death or the need for liver transplantation. PBC is classified as an orphan disease, and is estimated to affect roughly 65,000 to 85,000 patients in the US. A key hallmark of PBC (especially in early stages of the disease) is elevated levels of the liver enzyme alkaline phosphatase (ALP). Phase 2 studies of OCA demonstrated highly significant reductions of ALP levels in patients with PBC.

Intercept initiated enrollment of the Phase POISE 3 study in PBC in early-2012, and completed enrollment ahead of schedule in December. The company expects to report top-line results from the trial in 2Q14. In addition to the PBC, Intercept is also studying OCA in other indications including NASH, Portal Hypertension and Bile Acid Diarrhea, all currently in Phase 2.

Intercept owns worldwide rights to OCA with the exception of Japan and China, where Dainippon Sumitomo Pharma has an exclusive license from Intercept. Intercept also has an existing collaborative agreement with French drug company Servier for the discovery and development of bile-acid related drugs and technology.

The remainder of Intercept's pipeline consists of INT-767, a dual agonist of FXR and TGR5, in early stage development for fibrosis, and INT-777, a TGR5 agonist in early-stage development for diabetes. We expect Intercept to attempt to out-license INT-777, as diabetes appears to be outside of the company's core focus. Importantly, we believe the positioning of INT-767 as a second-generation drug to OCA may provide Intercept the

flexibility to command orphan-drug pricing for PBC (and possibly portal hypertension) while pricing INT-767 for larger indications such as NASH and bile-acid diarrhea.

Valuation

Our $60 price target for ICPT is derived from a sum-of-parts analysis of the company's development pipeline for the treatment of PBC, Portal Hypertension and Bile Acid Diarrhea.

We value ICPT using a probability-adjusted net present value (pNPV) approach, calculating anticipated profits from OCA (or INT-767) through 2026, discounted at 10.5% with no terminal value. We then adjust for clinical and regulatory risk by assigning an estimated probability of success, based on stage of clinical development and our assessment of the available clinical data and characteristics of the proposed indication. Our valuation does not include Intercept's cash, as we expect these funds to be fully utilized for development purposes.

Exhibit 2: ICPT Probability-Adjusted NPV

Drug/Indication	Expected Launch	Peak Sales Estimate ($MM)	Est. Probability of Success	P-Adj NPV ($MM)	P-Adj Value / Share
OCA - Primary Biliary Cirrhosis	2015	$646	74%	$851	$40
OCA/INT-767 - Portal Hypertension	2018	$637	28%	$231	$11
OCA/INT-767 - Bile Acid Diarrhea	2018	$541	33%	$190	$9
Pipeline Value				**$1,271**	**$60**
Net Cash				$143	$7
Total Equity Value				**$1,271**	**$60**
Diluted Shares Outstanding Used for Valuation (MM)					21.4

Source: Oppenheimer & Co.

As shown above, the largest contribution to our valuation comes from OCA for the treatment of PBC, which is a result of the drug's late development stage and our optimism for Phase 3 success and subsequent FDA approval. We estimate peak worldwide sales of $646M in PBC, which assumes annual pricing of $40,000 in the US. Although we believe that Portal Hypertension and Bile Acid Diarrhea represent significant market opportunities, we assign a lower chance for success given the more limited data generated in these indications to date.

We see upside to our $60 target in the event that OCA's follow-on indications demonstrate additional positive clinical data proof-of-concept studies, as well as potential upside to our pricing estimates for OCA. Of note, the share count used in our calculations includes dilution from potential financings to support future clinical development for indications other than PBC.

Upcoming Catalysts

As listed below in Exhibit 3, we see several catalysts during 2013-2015 that could create upside for the stock. In our view, the release of Phase 3 OCA data in 2Q14 represents the most important milestone for Intercept during the next 12 months. Although we believe expectations for the Phase 3 results are relatively high, we still anticipate ICPT shares to trade upward on positive data given the binary nature of these results and their importance to the long-term outlook for Intercept.

Exhibit 3: ICPT Potential Upcoming Milestones

Expected Date	ICPT Milestone
2H13	Additional presentations of PBC "supergroup" data
2H13	Updated data from Phase 2a trial of OCA in portal hypertension (PESTO study)
2H13	Updated data from Phase 2a trial of OCA in bile-acid diarrhea (OBADIAH study)
2H13	Initiation of Phase 3 OCA confirmatory outcomes study in PBC
2Q14	Topline data from pivotal Phase 3 POISE study of OCA in PBC
4Q14	Topline data from Phase 2 study of OCA in NASH (FLINT study)
4Q14	FDA and EMA regulatory filings for OCA in PBC
2014	Potential updates regarding clinical development plans for portal hypertension and bile acid diarrhea
Mid-2015	Potential approval of OCA for PBC

Source: Company reports, Oppenheimer & Co. Inc. research

Other potential catalysts over the coming year include additional data from the PBC supergroup analysis, the initiation of a confirmatory outcomes study in PBC (signaling increased assurance of the accelerated approval pathway) and incremental data updates from ongoing Phase 2a studies of OCA in portal hypertension and bile-acid diarrhea.

PBC Background

Primary Biliary Cirrhosis is a rare liver disease that primarily affects adult women beginning in middle age. PBC is marked by autoimmune-mediated damage to the bile ducts that circulate bile acids out of the liver. Over time, destruction of these bile ducts causes bile acids to accumulate to toxic levels in the liver, leading to its progressive degradation from chronic inflammation. Obstruction of normal bile flow, also known by the medical term cholestasis, is damaging to the healthy liver. Bile is a key component of the hepatic and GI systems as it serves to help digest fats and fat-soluble vitamins in the small intestine. While the degree of severity is somewhat heterogeneous, over time PBC can eventually lead to liver failure and death. Notably, before the recent emergence of obesity-related diseases such as NASH (Non-Alcoholic Steatohepatitis), PBC was once the most frequent cause of liver transplantation in the US.

Because PBC is a progressive disease with a relatively lengthy course, clinical determinations such as diagnosis, progression, and treatment-response rely upon measuring the levels of a biomarker called alkaline phosphatase (ALP). ALP is an enzyme released by the liver in response to certain toxicities, which in the case of PBC is the buildup of bile acids (which become toxic at high concentrations) due to cholestasis. As discussed later, ALP levels are crucial to measuring disease course and determining whether a patient has adequately responded to therapy.

New patients with PBC typically present in one of two ways: (1) by complaints of frequent pruritus (itching or tingling) and fatigue, or (2) via findings of elevated ALP during a routine blood-test. A diagnosis of PBC following elevated ALP levels can be made following a confirmatory test for serum anti-mitochondrial-antibodies. A liver biopsy is not necessary for confirming a diagnosis of PBC, although biopsies may be performed at intervals throughout the disease course to check for progression and the onset of cirrhosis. Importantly, biopsies are not required prior to initiating therapy or as inclusion criteria in the POISE study.

Exhibit 4: Outline of Primary Biliary Cirrhosis (PBC)

Incidence	Up to 1:1,000 females over 40 years old. 10:1 female to male preponderance.
Prevalence	Estimate approximately 300,000 persons with PBC in developed countries, with 60,000 diagnosed and treated.
Diagnosis	PBC is typically suspected when a middle-aged female presents with complaints of pruritus and fatigue. Diagnosis is confirmed via elevated ALP levels + positive AMA. A liver biopsy is not required.
Course	Progressive disease course, although severity and rate of progression are highly variable. Some patients exhibit minimal symptoms, others rapidly progress to liver failure. Median survival in untreated patients is reported to be 7.5 to 16 years.
Symptoms/ Complications	Pruritus and fatigue are key hallmarks of PBC, present in up to 85% and 70% of PBC patients. Other complications include jaundice, hypercholesterolemia, xanthomas and osteomalacia; progressing to portal hypertension, hyperbilirubinemia, ascites, encephalopathy, and liver failure (resulting in a liver transplant or death).
Standard of Care	The only approved therapy is Urso. Immunosuppressants are sometimes used off-label.
Treatment duration	PBC patients typically require chronic therapy.

Source: FDA review documents for URSO, Oppenheimer & Co. Inc.

Our conversations with physicians suggest that PBC is not an especially difficult or elusive diagnosis for a general practitioner to make, although patients are usually referred to a specialist following diagnosis. Under-diagnosis of PBC exists primarily in patients with more mild disease and/or patients who do not receive routine medical exams.

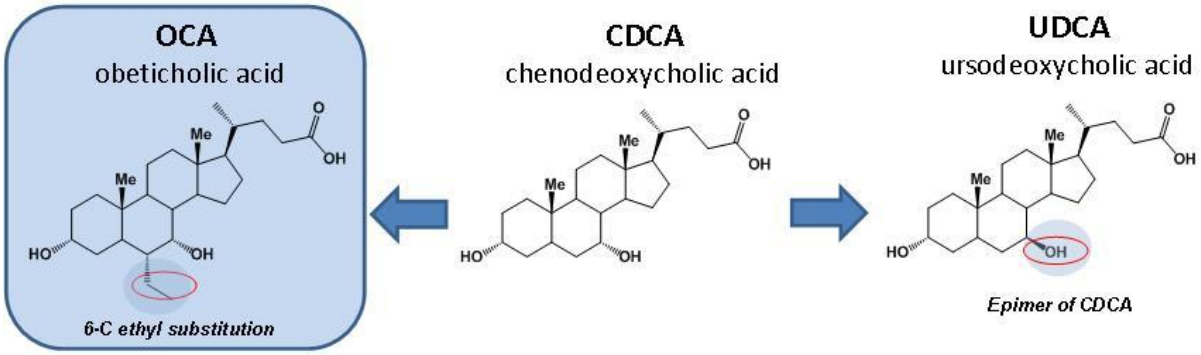
PBC is designated as an orphan disease in the US and Europe. As discussed in more detail below, while making a highly accurate determination of the disease's prevalence is difficult, Intercept estimates that there are approximately 300,000 persons with PBC in developed countries, of whom roughly 60,000 have been diagnosed and are being treated with Urso. Our own analysis of the US market size suggests the total population of diagnosed and treated PBC patients to be approximately 34,000, with approximately 14-15,000 of these patients being non-responders to Urso or otherwise eligible for second-line therapy with OCA.

Ursodeoxycholic acid (Urso)—Current Standard of Care Is Insufficient in a Large Subset of PBC Patients, Presenting an Unmet Medical Need

Currently, the only approved drug for the treatment of PBC is the generic drug ursodeoxycholic acid, abbreviated as UDCA, but often referred to as Urso. Urso is one of several naturally occurring bile acids, and in humans it comprises only small percentage (less than 5%) of a person's total bile acid pool. Upon administration of synthetic Urso as a therapeutic agent, the drug enters the enterohepatic circulation (a flow path consisting of the liver, biliary tract and intestine) and increases the concentration of Urso to 50-60% of a patient's total bile acid pool.

The primary mechanism of action by which Urso functions as a therapeutic for PBC is physiochemical in nature as it has less of a detergent effect compared to other bile acids. In short, Urso is less toxic to hepatocyte cell membranes than the bile acid CDCA, normally the largest component of the bile acid pool. Urso therapy dilutes the normally large concentration of CDCA. In cholestatic patients, high levels of CDCA can have toxic effects as its accumulation in the liver damages hepatocyte membranes to the point of triggering apoptosis (cell death).

Exhibit 5: Chemical Structures of CDCA, UDCA (Urso) and OCA



Source: Company Documents

Urso was first approved in 1987 (brand-name ACTIGALL by Ciba-Geigy, now owned by Novartis) for the treatment of gallstones, but quickly emerged as an off-label treatment for PBC. A clinical study of a tablet formulation (which held a number of advantages over ACTIGALL) was conducted specifically for PBC. This drug, branded URSO by Axcan Pharmaceuticals (now Aptalis Pharma, private), was approved for PBC by the FDA in late 1997.

Although Urso's clean safety profile and modest efficacy led to its widespread use in PBC, a significant number of patients do not respond sufficiently to bring the disease under control. We believe this is likely due to PBC's wide spectrum of disease severity along with Urso's non-specific mechanism of action.

Recall that PBC's disease course is measured by the level of ALP enzyme, particularly in the early-to-middle stages of the disease. Patients whose ALP levels remain high despite therapy with Urso are considered to be non-responders and remain at high risk of liver failure and death.

The consensus treatment goal for PBC therapy is a reduction in ALP to below 1.67x the upper limit of normal (ULN). The unmet medical need in PBC exists in those patients whose ALP levels remain above 1.67x ULN after treatment with Urso.

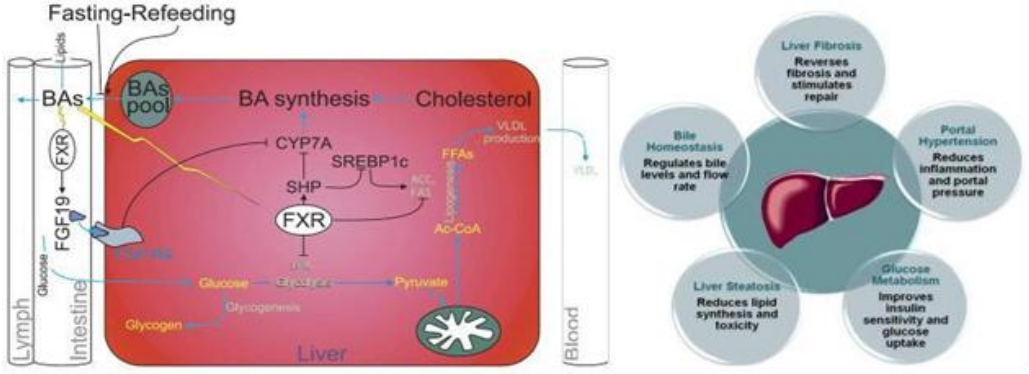
ICPT's OCA Addresses Unmet Clinical Need

Mechanism of Action in PBC and other Liver Diseases

Intercept is developing OCA as a second-line therapy for PBC in patients who do not adequately respond to treatment with Urso. Unlike Urso, OCA is a bile acid analogue discovered through medicinal chemistry, and does not exist as part of the normal bile acid pool in humans. OCA is structurally similar to CDCA, with the only difference being a 6-ethyl substitution. This substitution allows the molecule to fit more tightly inside the "pocket" of the FXR-receptor binding domain and is the driver of its activity as a potent FXR agonist, unlike Urso which does not bind to FXR. Moreover, OCA is not converted back into CDCA under physiological conditions and can be administered at much lower doses as a therapeutic agent compared to Urso (10 mg/day vs. 1,000 mg/day).

The farnesoid X Receptor (FXR) was discovered in 1999 and subsequently identified as a bile acid receptor. Further research elucidated FXR's function as playing an important role in regulating the flow and synthesis of bile acids. FXR is necessary for the normal regeneration of the liver, and its downstream targets encompass a myriad of liver disease related deregulations (Exhibit 6)

Exhibit 6: FXR Pathway and Mechanisms in Liver Disease



Source: Company Documents

Positive Phase 2 Studies Provide Confidence for Pivotal Trial Success

As discussed below in more detail, our review of the existing data suggests that OCA provides a clinically significant benefit for PBC patients.

Our optimism for OCA is based upon the robust efficacy demonstrated in two Phase 2 studies conducted by Intercept. In both studies, OCA demonstrated highly statistically significant reductions in ALP levels compared to placebo ($p<0.0001$ for all doses tested). These reductions occurred when OCA was administered as monotherapy ('201 trial n=59) or in combination with Urso ('202 trial; n=165). Overall, 80% of OCA patients experienced a greater than 10% reduction in ALP levels (the cut-off for a clinically meaningful effect), compared to only 13% of placebo patients. Moreover, in a retrospective analysis of the Phase 2 studies using the same responder criteria employed in the Phase 3 trial, 40-45% of the OCA patients would have met the endpoint, compared to 5-9% of placebo patients.

Exhibit 7: Top-line Results from OCA Phase 2 Studies in PBC

	Primary Endpoint: Mean ALP change (12 weeks)	**p-value**
OCA/Urso Combo (Study '202)		
10 mg (n=38)	-24%	<0.0001
25 mg (n=47)	-25%	<0.0001
50 mg (n=39)	-21%	<0.0001
Placebo/Urso (n=37)	-3%	NS
OCA Monotherapy (Study '201)		
10 mg (n=20)	-45%	<0.0001
50 mg (n=16)	-38%	<0.0001
Placebo (n=23)	0%	NS
	Retrospective "Responder" Analysis (12 months)	**p-value**
OCA vs. Placebo pooled across both Phase 2 trials	40-45% 10mg OCA pts. Vs. 5%-9% placebo pts. achieved ALP reduction below 1.67x ULN and a ≥15% drop from baseline, along with normal bilirubin (the Ph 3 POISE endpoint)	<0.0025

Source: Company reports, NS = not significant.

Although we acknowledge the general caveat in drug development that Phase 3 studies rarely perform as well as their Phase 2 counterparts, the overwhelming level of efficacy demonstrated in the OCA Phase 2 studies should provide a substantial cushion for the unforeseen clinical risks that may surface in larger Phase 3 studies.

Phase II OCA Combination Study (202 Trial)

The more comprehensive of Intercept's two completed clinical studies is the 202 combination trial, which tested OCA as an add-on to Urso in patients with persistently elevated ALP levels despite being treated with Urso. Eligible patients were required to exhibit ALP levels greater than 1.5x ULN while receiving Urso for at least six months prior to screening.

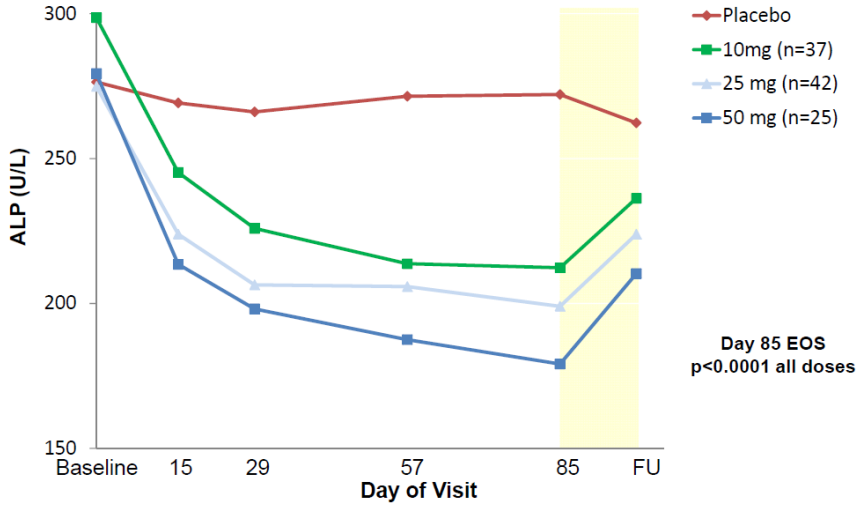
Initiated in Noveber 2008, the combination trial enrolled a total of 165 patients in the US, Canada and Western Europe. Patients were randomized to receive OCA 10mg, 25mg, 50mg or placebo once daily for 12 weeks in combination with Urso. The study's primary

endpoint was reduction in ALP, with secondary endpoints including measurements in GGT, ALT, AST and bilirubin levels.

As shown previously in Exhibit 7, each OCA dose cohort exhibited a statistically significant reduction in ALP levels compared to the Urso alone (the placebo arm), with mean reductions of 23.7%, 24.7% and 21.0% at 10mg, 25mg and 50mg of OCA, respectively, compared to a 2.6% reduction in the placebo arm. Of note, these results reflect a modified intent-to-treat (mITT) analysis which includes patients who withdrew from the study. This accounts for the lower response seen in the 50mg cohort, which contained a higher number of patients who withdrew from the study due to pruritis (discussed in more detail later in this report).

When looking at ALP levels over the course of the trial, OCA demonstrated rapid reductions in ALP followed by sustained declines through the end of the study. ALP levels then rebounded when patients were removed from the drug and returned for a follow-up visit two weeks later (Exhibit 8).

Exhibit 8: Primary Endpoint of Phase 2 OCA Combination Study



Source: Company documents.

Of note, the data in Exhibit 8 excludes patients who withdrew from the study, and a dose-response effect is more pronounced compared to the mITT analysis. We highlight that the ALP reductions seen at the 10 mg dose were also reflective of a very high level of efficacy. This is important because the 10mg dose of OCA is significantly better tolerated by PBC patients with regards to pruritis.

In our view, the Phase 2 combination study offers clear and convincing evidence that OCA is effective at reducing ALP levels in patients who have reached the limit of benefit from Urso. As demonstrated in the PBC “supergroup” study, achieving target reductions in ALP levels strongly correlates to improved survival.

Additionally, the study demonstrated statistically significant reductions in a number of other liver function markers, such as GGT, ALT, AST, and bilirubin, the latter of which is of more importance because bilirubin is a component of the primary endpoint in the Phase 3 POISE study.

Phase II OCA Monotherapy Study (201 Trial)

Intercept initiated the smaller Phase II OCA monotherapy study in December 2007. This multi-center (US, Canada and Western Europe), double-blind, placebo-controlled study enrolled 59 PBC patients for 12 weeks of treatment and a 2-week follow-up. Patients were randomized to receive OCA 10mg, 50mg or placebo once daily. The study's primary endpoint was reduction in ALP levels, with secondary endpoints assessing levels of GGT, ALT, AST and bilirubin (other liver-related enzymes). In order to meet eligibility criteria, patients must have had a baseline ALP value above 1.5x ULN and must have not received treatment for PBC within the prior 3 months.

The study overwhelmingly met its primary endpoint, with OCA groups demonstrating average decreases in ALP levels of 45% at 10mg and 38% at 50mg, which was statistically significant ($p<0.0001$ for both doses) compared to placebo patients (ALP unchanged).

Safety and Tolerability of OCA in Phase 2 studies

In our view, the Phase 2 studies also demonstrate an encouraging safety profile for OCA, with no drug-related serious adverse events observed and little difference among other safety events when compared to placebo.

Although the drug appears to have a clean safety profile, OCA does noticeably differ from placebo in the rate of pruritus, the medical term for itching. We note the important distinction that pruritus is chiefly a tolerability issue and not a safety issue in PBC. As discussed previously, pruritus is a hallmark symptom of PBC, and many patients are first diagnosed after presenting with persistent pruritus.

Exhibit 9: Withdrawal and Pruritus Rates In Phase 2 PBC Studies

	Phase 2 Combination Study (202)				Phase 2 Monotherapy Study (201)		
	10mg	25mg	50mg	Placebo (+Urso)	10mg	50mg	Placebo
Started Trial (n=)	38	48	41	38	20	16	23
Completed Trial (n=)	37	42	25	37	16	9	23
Withdrew (n, %)	1 (3%)	6 (13%)	16 (39%)	1 (3%)	4 (20%)	7 (44%)	0 (0%)
Patients with SAE*	0 (0%)	1 (2%)	5 (12%)	1 (3%)	0 (0%)	0 (0%)	1 (4%)
Pruritus	18 (47%)	41 (85%)	33 (80%)	19 (50%)	14 (70%)	15 (94%)	8 (35%)

Source: Company reports. *all SAEs deemed unrelated to study drug

The table above shows the withdrawal rate and incidence of pruritis in the completed Phase 2 studies. Aside from pruritis, there was no difference in OCA adverse events compared to the placebo arms of the trial. Moreover, the small number of serious adverse events (SAEs) seen in the study were deemed to be unrelated to treatment (SAEs included salivary gland neoplasm, angina pectoris, GI hemorrhage, jaundice and angioedema in the OCA arms, and dyspnea and rash in the placebo arms).

As it relates to OCA's ongoing Phase 3 study and eventual commercial potential, pruritus becomes relevant when it is serious enough to lead to discontinuation of treatment. While many patients experience relatively mild and/or temporary pruritus on OCA, the higher doses tested in Phase 2 did show an elevated rate of study withdrawal (39% at 50 mg). Fortunately, the 10mg dose retains comparable levels of efficacy to the higher doses yet

demonstrated much lower rates of pruritus (47% in '202 study, comparable Urso) and drop outs (3%).

Most important, we are encouraged by the fact that Intercept recently disclosed that the dropout rate due to pruritus observed in the fully enrolled Phase 3 trial is lower thus far then what was observed in Phase 2, even assuming that all withdrawals occurred in the OCA dosing groups.

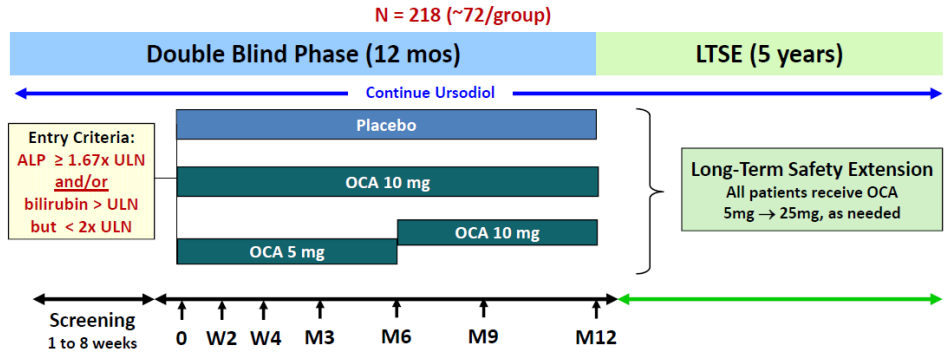
We account for the likelihood of some degree of patient discontinuation due to pruritus in a real-world setting. We have incorporated a ~5% treatment discontinuation rate in our market model for OCA in PBC.

Pivotal Phase 3 POISE Study

Intercept is conducting a pivotal Phase 3 study of OCA in PBC called the POISE trial. A randomized, double-blind, placebo-controlled trial, POISE is being conducted across 59 centers in 13 countries. Enrollment began in January 2012 and was targeted to enroll a total of 180 patients. However, following heavy demand for the trial, enrollment completed well-ahead of schedule and above the target number, with a total 218 patients.

The design of the Phase 3 POISE study is summarized below. Patients are randomized to one of three arms: OCA 10mg, OCA 5mg titrated to 10mg after 6 months, or placebo. The total treatment duration is 12 months, and following the study, all patients are eligible to continue OCA treatment in a long-term safety extension trial.

Exhibit 10: Phase 3 POISE Trial Design



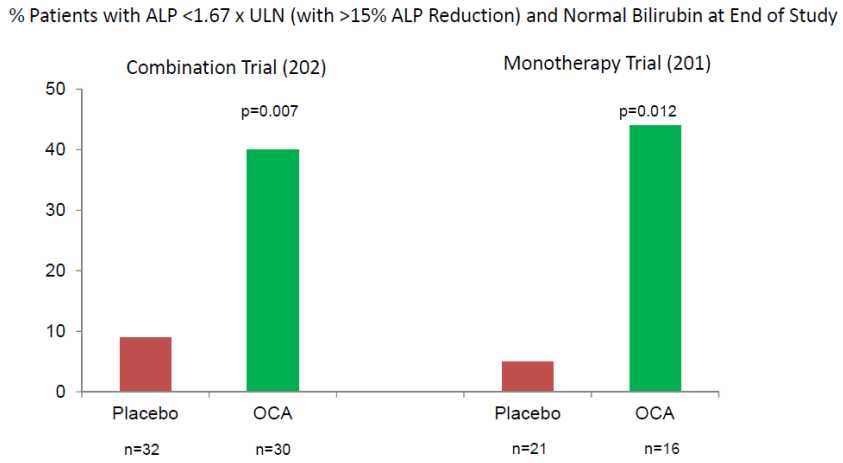
Source: Company Documents

Unlike the Phase 2 studies which measured ALP change as their primary endpoints, the Phase 3 POISE study incorporates ALP reduction into a responder endpoint. In POISE, the primary endpoint is the percentage of patients meeting the defined response criteria: (1) reduction of ALP to < 1.67x ULN; (2) absolute reduction of ALP by at least 15%; and (3) normal bilirubin levels.

The restructured primary endpoint (compared to Phase 2) not only serves to align the study with clinically relevant practices, but also better positions the study to support FDA approval, in our view. As discussed below, we believe the change does not represent a significant risk to the study's succeeding.

Further optimism for the POISE trial comes from Intercept's analysis of the Phase 2 studies, which retrospectively applied the composite primary endpoint used in POISE to determine which patients would have met the responder criteria.

Exhibit 11: Analysis of Phase 2 Patient Outcomes (POISE Criteria)



Source: Company Documents

As shown in Exhibit 11 above, roughly 40% of 10mg OCA treated patients would have met the composite endpoint compared to < 10% for placebo-treated patients. This difference was statistically significant despite the smaller sample size and shorter treatment duration compared to the ongoing Phase 3 study. Although retrospective analyses are inherently less conclusive compared to prospective studies, these results significantly diminish our perceived risk of the Phase 3 study's underperforming due to the use of a different primary endpoint.

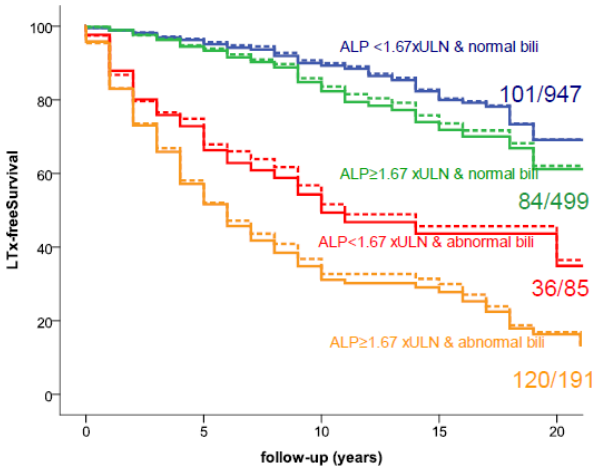
Regulatory Pathway for OCA in PBC and Importance of "Supergroup" Analysis

OCA's regulatory pathway in Europe appears rather straightforward, with the company indicating that the POISE trial design has been deemed acceptable to support approval by the European Medicines Agency (EMA). In the US, Intercept intends to seek accelerated approval (Subpart H) using data from the POISE study and running a longer-term outcomes study to corroborate approval in the future. Under accelerated approval, the FDA is allowed to approve a drug based on demonstrated benefit in a surrogate endpoint, rather than a clinical outcome measurement such as survival. For OCA, the surrogate endpoint is the ALP/bilirubin response criteria employed in the Phase 3 POISE study (ALP below 1.67x ULN and a reduction of at least 15%, along with normal bilirubin).

An accelerated approval strategy is most often sought for significant unmet medical needs in which conducting an outcomes-based study would be impractical or otherwise significantly delay approval. Instead, a surrogate endpoint that is reasonably likely to predict clinical benefit is used in lieu of an outcome endpoint.

Intercept has provided very strong evidence for ALP's validity as a surrogate endpoint via a large "supergroup" study of close to 4,000 PBC patients treated at 13 centers around the world. Researchers collected individual patient data on clinical characteristics (including ALP levels) and long-term outcomes. Through this study, Intercept is able to demonstrate that patients that have adequately controlled ALP levels experience significantly improved transplant-free survival compared to patients whose ALP levels remain elevated.

Exhibit 12: Transplant-free Survival Curves of PBC Patients



Source: Lammers et al. EASL 2013

An initial analysis from the study on the first 2,100 patients was presented at the EASL conference in April 2013. As shown in Exhibit 12 above, PBC patients with ALP levels below 1.67x ULN demonstrate significant improvements in transplant-free survival ($p<0.000001$). The result is more pronounced when patients are further stratified by normal or abnormal bilirubin levels. Recall that the Phase 3 POISE endpoint is a composite of ALP and bilirubin.

In summary, we believe OCA is likely to qualify for accelerated approval (assuming positive Phase 3 results) due to a number of reasons:

(1) Conducting an outcomes study in PBC would require a very large number of patients and a multi-year duration, significantly delaying access to treatment for a serious unmet medical need.

(2) Key opinion leaders appear vocal in their support for ALP as a valid surrogate marker for survival in PBC patients.

(3) The FDA's prior review of Urso highlights that demonstrating a survival benefit in PBC would be onerous for a pivotal study, and Urso was approved without demonstrating a statistically significant reduction in survival.

(4) In our view, the FDA (as well as advisory committees) has been increasingly amenable to approving safe drugs based on surrogate endpoints for orphan indications with high unmet medical need. Moreover, because PBC is predominantly a disease of middle-aged women, we see a heightened sensitivity to avoid undue controversy resulting from delaying a safe and efficacious drug to this specific patient population.

That said, we acknowledge that regulatory risk is relatively higher for accelerated approval, and FDA actions in general are inherently unpredictable.

Market Size Estimates and OCA Sales Potential

Based on industry data, Intercept estimates approximately 60,000 diagnosed PBC patients in developed countries are receiving Urso therapy (out of 300,000 total individuals thought to have the disease). Of these 60,000 PBC patients, Intercept estimates that up to 50% do not adequately respond to Urso and would be candidates to receive OCA as a second-line treatment. While this estimate is supported by the recent "supergroup" analysis, we take a slightly more conservative approach with our estimates as our conversations with physicians suggest that the 50% non-responder rate is likely at the upper end of estimates.

We note that our estimate of approximately 14,000 US patients eligible for OCA compares to Intercept's estimate of 15,000-20,000 patients. For Europe, we model a higher slightly higher number of PBC patients, but expect lower OCA penetration and pricing compared to the US. We see upside potential to our peak revenue estimates from pricing and a potentially higher real-world rate of Urso non-responders compared to our assumptions.

Competitive Space in PBC

With regards to competition in PBC, we note that the small number of drugs in active development is made up mostly of potent immunosuppressants which we believe would be likely relegated to third-line use, if they reach the market. Although recent publications have also highlighted small clinical studies of fibrates as a potential therapy for PBC, we believe fibrates are very unlikely to reach widespread use as they are specifically contraindicated in PBC patients due to risk of serious liver damage.

Moreover, we believe there is ample room in our model assumptions to allow for niche use of fibrates or other off-label therapies (we estimate OCA's peak penetration at 60% in US and 30% in Europe).

Development of OCA and INT-767 for Additional Indications: Significant Longer-term Value Potential Beyond PBC

As we believe ICPT's current share price is being driven mostly by PBC, we see significant upside potential from a number of follow-on indications under development. Intercept's active clinical programs are listed in the table below.

Exhibit 13: Assumptions for US PBC Market Opportunity and Peak Revenue Estimates

Drug	Indication	Stage	Comments / Expected Milestones	Expected Data
OCA	PBC	Phase 3	Pivotal POISE trial.	2Q14
OCA	Bile Acid Diarrhea	Phase 2a	OBADIAH study. Investigator sponsored (UK). 30 patient target enrollment. 14 day treatment duration.	2H13
OCA	Portal Hypertension	Phase 2a	PESTO study. Investigator sponsored (UK). 39 patient target enrollment. Initial cohort presented at AASLD 2012.	2H13
OCA	NASH	Phase 2b	FLINT study. NIDDK sponsored. 280 patient target enrollment.	4Q14
INT-767	Fibrosis	Preclinical	Dual FXR/TGR5 agonist. Potential follow-on compound to OCA.	IND filing in 2014
INT-777	Type 2 diabetes	Preclinical	Available for partnering but expect minimal development by ICPT.	

Source: Company reports.

We are encouraged by early proof-of-concept results seen in Phase 2a studies of Portal Hypertension and NASH.

OCA's mechanism of action and various preclinical data suggest that the drug may have therapeutic utility for a number of liver-related diseases. We are encouraged by the potential for OCA to turn into a viable franchise on its own, as a pipeline within a drug. We also note that Intercept may choose to promote INT-767 (second-generation OCA) for further study in these indications which could allow more flexible pricing options, if necessary.

As discussed below, early clinical data in a number of studies have recently begun to establish proof-of-concept in a number of indications. As more data become available (we expect several updates over the coming 12 months), we believe investors will begin to assign greater value to these programs.

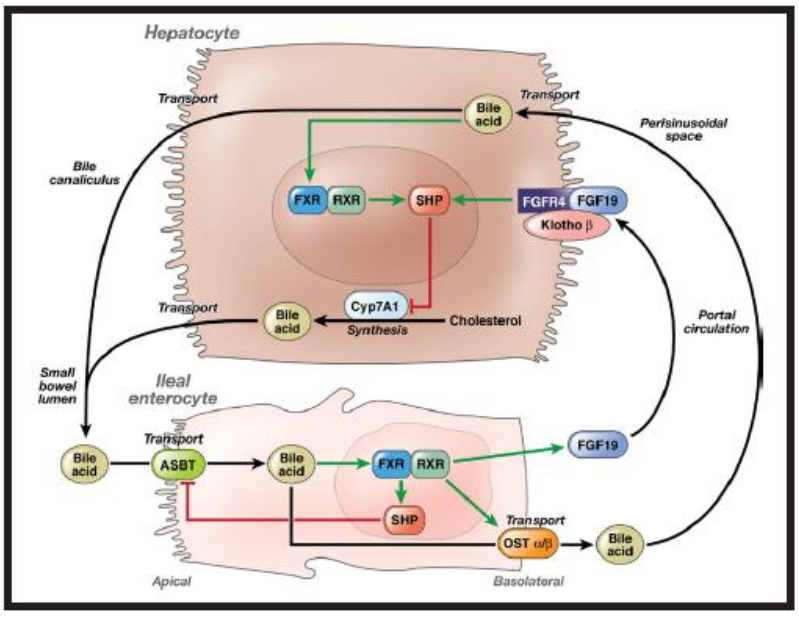
Bile Acid Diarrhea—Strong Rationale for OCA and a Significant Market Opportunity

Bile Acid Diarrhea (BAD) refers to a condition whereby a disturbance in normal bile-acid homeostasis leads to watery stools and erratic bowel function. Patients with BAD have severe diarrhea as a result of excess bile exiting the small intestine. Under normal conditions, roughly 95% of bile that enters the gut is reabsorbed in the terminal ilieum, which is located at the distal end of the small intestine. If excess amounts of bile reach the colon, colonic secretion and motility increase, causing symptoms of diarrhea, bloating, and fecal urgency. These symptoms are generally severe and can significantly impact quality of life.

BAD can arise due to ileal damage/insufficiency, although in many cases there is no clear underlying etiology (i.e., the ileum appears to be healthy). Therefore, patients are categorized as either having Primary BAD (P-BAD) or Secondary BAD (S-BAD). P-BAD refers to patients with no definitive etiology (no demonstrative ileal disease), whereas S-BAD patients develop the condition due to an underlying ileal disease or have undergone ileal resection or bypass. The most common cause of S-BAD is due to Crohn's disease, as a result of requiring ileal resection or the presence of significant damage to the ileum.

Numerous studies have demonstrated that the biological basis for BAD is a dysfunction in FGF19 (fibroblast growth factor 19) signalling. FGF19 is a hormone produced in the ileal enterocyte in response to bile-acid-mediated signaling through FXR, where it is then carried through the portal vein to the liver. At the liver, FGF19 binds to the FGFR4 receptor on the surface of hepatocytes, which inhibits the synthesis of bile-acids via a negative feedback loop.

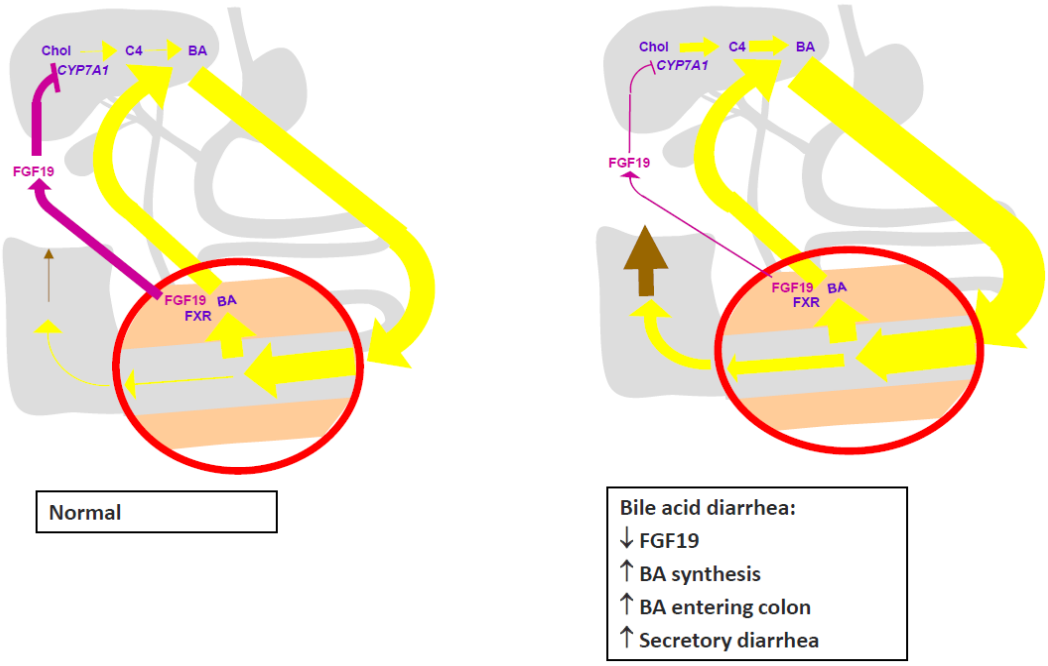
Exhibit 14: FGF19 Signaling Pathway



Source: Rao et al. *Gastroenterology 2010.*

In patients with primary BAD, the dysregulation of this negative feedback loop results in overproduction of bile acids which cannot be adequately reabsorbed by the ileum. , This overproduction can lead to excess bile acid entering the colon (Exhibit 15).

Exhibit 15: Schematic of FGF19-Mediated Bile Acid Diarrhea



Source: Johnston et al. DDW presentation 2013.

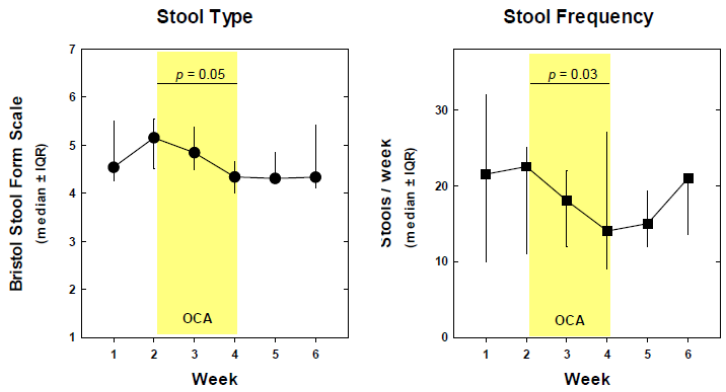
The evidence linking FGF19 to BAD was strengthened by a prospective study that measured fasting FGF19 levels in BAD patients compared to control patients with non-bile-acid-mediated chronic diarrhea, showing statistically significant lower median levels in the former.

As would be expected by the drug's mechanism of action via FXR, OCA has demonstrated a dose-dependent increase in FGF19 levels in patients receiving the drug. This finding was observed in Phase 2 studies conducted in PBC and NASH. Moreover, an ongoing Phase 2a study of OCA in BAD, described in detail below, showed significant improvements in symptoms coinciding with increases in FGF19.

The Phase 2a OBADIAH study of OCA in bile acid diarrhea is an investigator-sponsored study conducted by the Imperial College London group in the UK. This hybrid pilot/proof-of-concept study is an open label trial, enrolling 30 patients to receive 25 mg of OCA once daily for 2 weeks, bracketed by a 2-week lead-in phase and follow-up period. Enrollment was divided into 3 cohorts (n=10 each) of P-BAD, S-BAD and chronic diarrhea controls. Of note, the S-BAD cohort consists of patients with ileal resection due to Crohn's disease.

Initial results from the P-BAD cohort were presented at the Digestive Diseases Week conference (DDW) on May 19, 2013. The data showed clinical improvements in all patients, including a reduction in median stool frequency from 23 to 14 times per week (p=0.03) and improvements in the median Bristol Stool Form Scale (BSFS) from 5.15 to 4.34 (p=0.05). Additionally, stool frequency was observed to return to baseline levels during the two-week follow-up period after stopping OCA therapy (Exhibit 16).

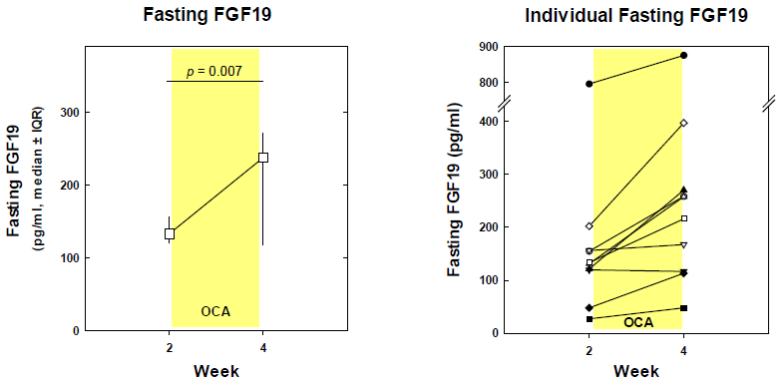
Exhibit 16: Schematic of FGF19-Mediated Bile Acid Diarrhea



Source: Johnston et al. DDW Presentation 2013

Importantly, the overall improvements in clinical measures were accompanied by a demonstrated increase in fasting FGF19 plasma levels (measured at baseline and after 2 weeks) that was statistically significant (237 pg/ml vs. 133 pg/ml; p=0.007). The investigators reported that most patients had an increase of 60% or greater in fasting FGF19.

Exhibit 17: OBADIAH Cohort 1 FGF19 Results



Source: Johnston et al. DDW Presentation 2013

We expect data from the second two cohorts of the study to be available in 2H13. Upcoming results from the S-BAD patient cohort may be especially informative as Intercept weighs its options for conducting Phase 2b/3 studies (design of an S-BAD study would likely be more straightforward than P-BAD and could be completed more quickly, in our view).

Current treatment for BAD is limited to bile acid sequesterants such as cholestyramine and Welchol, which are associated with a number of side effects and can interfere with the absorption of nutrients and commonly used medications.

In terms of market size, estimates of P-BAD prevalence as a percentage of all patients with Irritable Bowel Syndrome (IBS) vary in the literature. BAD was once thought to be relatively rare, but following the availability of a diagnostic test in Europe called the

SeHCAT retention test, studies of IBS patients suggest that roughly 1 in 9 patients with IBS (or 1 in 3 patients with IBS-D) can be classified as having BAD. Given the high degree of IBS prevalence, 1% of the general population in Western countries may suffer from P-BAD. However, due to challenges in diagnosing and identifying patients with P-BAD, we estimate that of the 3-4 million persons in the US who may have the disease, the market opportunity for OCA or INT-767 is likely to start at 150,000 patients. We estimate peak penetration of 25%, leading to peak US sales of roughly $400M annually.

Moreover, we would expect the bile-acid diarrhea market to grow significantly upon the introduction of effective therapies, representing upside to our estimates.

Portal Hypertension – Significant Cause of Mortality in Chronic Liver Diseases

Portal Hypertension is a major hallmark of cirrhosis arising from the increased resistance to blood flow found in cirrhotic livers, which elevates the portal venous pressure gradient (between the portal vein and inferior vena cava). Although Portal Hypertension can present in a number of ailments, cirrhosis is the most common cause in the Western World.

In clinical settings, portal pressure is commonly measured by a metric known as the hepatic vein pressure gradient (HVPG). Currently, HVPG measurements require a somewhat invasive procedure, although development of non-invasive ultrasound tests is underway. Elevated HVPG is responsible for a number of the complications of liver cirrhosis, with varices forming when HVPG rises above 10 mm Hg and subsequent bleeding when HVPG exceeds 12 mm Hg.

Portal Hypertension remains a significant unmet medical need with no approved therapies. Although beta blockers and certain procedures can be used to delay the occurrence of variceal bleeding (at the expense of other serious complications), a liver transplant is the only means of extending long-term survival.

Phase 2a PESTO Trial of OCA

Intercept's OCA is hypothesized to be a potential treatment for portal hypertension by reducing inflammation and hepatic resistance through a vasodilatory mechanism via intra-hepatic nitric oxide. An investigator-sponsored Phase 2a study called PESTO is enrolling 39 patients with alcoholic cirrhosis and Portal Hypertension (HVPG ≥ 12mm Hg) to receive OCA 10 mg/day for 7 days. The primary endpoint is patient response, defined as either HVPG reduction to less than12 mmHg or a decrease of at least 15% from baseline.

Initial results from the first 12 patients in the study (4 were assessed for drug safety and tolerance and 8 were assessed for HVPG reduction) were presented in a late-breaking poster at the AASLD conference in November 2012. Among the 8 patients assessed for efficacy, 5 met the criteria for a positive response. A 6th patient had a 14% fall in HVPG (overall, HVPG was reduced by an average of 24% in the 6 patients). Two patients did not respond to OCA.

Although the current dataset is small, we view the PESTO results as promising and a justification to begin additional clinical studies of OCA in portal hypertension. Given the unmet medical need and the potential for OCA to meaningfully improve survival in these patients, we believe investors will begin to assign value to this opportunity as ICPT reports additional data from the full PESTO cohort (expected later this year) and outlines a plan for moving into a larger clinical study.

NASH – Complicated Development Pathway, But Large Unmet Medical Need Exists

Following skyrocketing rates of obesity in the US, Non-Alcoholic Steatohepatitis (NASH) has become an increasingly prevalent medical problem in recent years. According to the National Institutes of Health, NASH has an incidence rate of approximately 25% in obese individuals, and is estimated to affect roughly 7.5 million adults in the US, increasing to 25 million by 2025. Patients with NASH are in danger of progressing to liver cirrhosis, which puts them at a high risk of liver failure and liver cancer. Currently, there are no approved drugs for treating NASH.

In 2010, the National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK) selected OCA as the most promising new NASH treatment in development and provided funding for the FLINT trial to test OCA's potential to treat the disease. This Phase 2b study began enrollment in early 2011 and completed enrollment of 280 patients in November 2012. The study has a 72-week treatment duration (25 mg OCA vs. placebo) and a primary endpoint of biopsy-determined improvement in NAFLD Activity Score. Intercept expects results from the study to be available in 4Q14. We note that the study passed an interim futility analysis in June 2012, conducted after the first 100+ patients reached at least 24 weeks of therapy (with the earliest enrolled patients having reached up to 15 months of therapy).

Given the longer timelines for the FLINT data availability and conducting Phase 3 studies, we do not include NASH in our ICPT valuation at this time. However, we note that NASH is a massively prevalent disease, and a drug that reaches the market could be a blockbuster opportunity.

Company Background

Recent Financing History

Intercept completed its initial public offering in October 2012, generating $78.7 million in net proceeds through the sale of 5.75 million shares at $15. The company announced a follow-on equity offering on June 20, 2013, proposing to sell up to 2.0 million shares for gross proceeds of up to $63.3 million.

Prior to the company's IPO, Intercept raised approximately $100 million cumulatively since inception via the sale of common stock, convertible notes, preferred stock and warrants. In addition, the company received non-dilutive financing in 2011 through collaboration and licensing agreements with Dainippon Sumitomo Pharma (DSP) and Les Laboratories Servier (Servier). Payments related to the licensing agreements are amortized as revenue.

Sales, Marketing and Manufacturing

As a development-stage company, Intercept relies on third-party contract manufacturers to supply its drugs for clinical trials. We do not expect Intercept to pursue internal manufacturing capabilities for the foreseeable future.

As OCA remains roughly two years from potential commercialization, we do not expect the company to make near-term investments in sales and marketing. We note the possibility that Intercept may sign commercialization partnerships with larger drug companies in specific countries or territories. However, we believe Intercept will seek to self-commercialize OCA in the US, where believe PBC specialists could be targeted by a relatively small and inexpensive sales force.

Intellectual Property and Exclusivity

As of March 15, 2013 Intercept reported to hold 51 issued or granted US and non-US patents relating to OCA, in addition to 9 pending applications. We view Intercept's key US patents as follows: (1) OCA composition of matter patent expiring in November 2022 and eligible for a ~5 year extension (7,138,390); (2) OCA method of use patent for treatment of cholestasis expiring in February 2022 (7,786,102); (3) OCA manufacturing method patent expiring December 2028 (7,994,352); (4) INT-767 composition of matter patent expiring in 2029.

Importantly, OCA has received orphan drug designation in the US and Europe which provides for marketing exclusivity of 7 and 10 years, respectively, following approval.

Key Risks

Clinical Risk. Intercept's drugs will be required to demonstrate efficacy and safety in clinical trials before they can be approved by regulatory agencies. Complicated, large clinical trials increase the risk of not meeting specified endpoints. It is also important to note that past successes in Phase 2 trials may not translate into successes in Phase 3 trials, given potential differences in study design and treatment protocol. We highlight that safety signals are usually more pronounced in large Phase 3 studies and that if unexpected safety problems were to arise, the company may be required to halt further development, representing a significant setback.

Regulatory Risk. ICPT has yet to submit for or receive approval for any of its drugs in the US, and may face difficulties in doing so, potentially delay commercialization. The regulatory process to attain approval of drugs is complex, requiring collection and production of extensive sets of data from expensive and time-consuming studies. Decisions on approval are at the discretion of the respective regulatory agencies, which can be unpredictable. We expect ICPT to pursue accelerated approval for PBC, utilizing data from the Phase 3 POISE study. Should the FDA disagree with the accelerated approval suitability, ICPT may be required to conduct additional clinical studies, which could significantly delay estimated approval timelines. Last, following potential approvals of drugs, the regulatory agencies retain the power and ability to remove these drugs from the market if deemed to present sufficient danger.

Commercialization Risk. Despite ICPT's ability to potentially attain approval of its development candidates, the company may face unpredictable commercialization challenges. These challenges include the presence of a competing drug with a superior efficacy and/or safety profile, greater physician preference and ease of reimbursement, and lower cost. ICPT may also depend on partnerships to commercialize its drugs. Should these partnerships derail, the company may face significant challenges or delays to successfully commercialization.

Intellectual Property Risk. There is inherent uncertainty in both the interpretation of patent claims and the application of patent law, regardless of the apparent strength of ICPT's patent portfolio. Upon expiration of licensed patents, ICPT may be unable to prevent third parties from genericizing its products. Furthermore, competitors may challenge the scope/validity the patents, or simply find ways to circumvent the patents.

Manufacturing Risk. ICPT does not possess its own manufacturing capabilities to clinically or commercially supply sufficient quantities of its drugs. Any disruption or contaminant problems could result in delays to clinical studies or future commercialization until such problems are resolved. Moreover, upon commercialization, an impact to the company's supply of drug product would adversely affect revenues.

Competitive Risk. The indications being targeted by ICPT are also being targeted by several competitors, some with superior resources. Additionally, ICPT may be unable to effectively compete for market against larger companies, even if ICPT products have superior clinical profiles vs. the competitors' products.

Financing Risk. While we believe ICPT is sufficiently capitalized to reach significant value inflection points, any unexpected clinical or regulatory setbacks may prompt capital raising to before ICPT is able to generate sufficient revenues from the commercial activities. We note that such financing may to be dilutive and potentially on unfavorable terms to current investors. Moreover, if ICPT is unable to complete necessary financings, development/operations plans would be severely impacted.

Management

Mark Pruzanski, M.D.

President and Chief Executive Officer

Dr. Pruzankski is a co-founder of Intercept and currently serving as CEO and president, and has been a member of the BOD since the company's inception in 2002. Dr. Pruzanski has over 15 years of experience in life science company management, venture capital and strategic consulting. Previously, he was a venture partner at Apple Tree Partners, an early stage life sciences VC firm. Prior to that, Dr. Pruzanski was an entrepreneur-in-residence at Oak Investment Partners. Dr. Pruzanski received his M.D. from McMaster University in Ontario, a M.A. degree in international affairs from the Johns Hopkins University School of Advanced International Studies and a bachelor's degree from McGill University. He currently serves on the boards of the Emerging Company Section of the Biotechnology Industry Association (BIO) and the Foundation for the Defense of Democracies.

Barbara Duncan

Chief Financial Officer

Ms. Duncan has served as ICPT's CFO and secretary since May 2009 and as treasurer since 2010. Ms. Duncan has over 14 years of experience in the life sciences industry. Previously, Ms. Duncan served as CFO and then CEO at DOV Pharmaceuticals, a biopharmaceutical company focused on central nervous system disorders. Prior to DOV, Ms. Duncan served as a VP at Lehman Brothers in the company's corporate finance division, where she provided financial advisory services primarily to life sciences and general industrial industries. From 1994 to 1998, Ms. Duncan was an associate and director at SBC Warburg Dillon Read, in the company's corporate finance group. Ms. Duncan received her B.S. from Louisiana State University in 1985 and her M.B.A. from the Wharton School, University of Pennsylvania.

David Shapiro, M.D.

Chief Medical Officer and EVP, Development

Dr. Shapiro has served as ICPT's CMA and EVP of development since 2008. Dr. Shapiro has over 25 years of clinical development experience in the pharmaceutical industry. In 2005, he founded a consulting firm, Integrated Quality Resources, which focused on development stage biopharmaceutical companies and was active in this role until 2008. Prior to that, Dr. Shapiro was an EVP of Medical Affairs and CMO of Idun Pharmaceuticals, Inc., prior to its acquisition by PFE. From 1995 to 1998, he was president of the Scripps Medical Research Center at Scripps Clinic. Dr. Shapiro received his medical degree from Dundee University & Medical School.

Luciano Adorini, M.D.

Chief Scientific Officer

Dr. Adorini has served as ICPT's CSO since 2008. Dr. Adorini has over 20 years of industry experience, encompassing his time as CSO at BioXell, from 2002 to 2007, where he was responsible for advancing a broad pipeline of products in multiple disease indications. Prior to that, he served as associate director of Roche Milano Richerche, where he contributed to the development of several drugs. Dr. Adorini received his M.D. degree from Medical School of Padova University and conducted his post-doctoral studies at UCLA.

Stock prices of other companies mentioned in this report (as of 6/18/2013):

AstraZeneca (AZN-NYSE, $50.98, Not Covered)
Dainippon Sumitomo Pharm (4506:TKS, ¥1256.0, Not Covered)
GlaxoSmithkline (GSK-NYSE, $52.47, Not Covered)
Novartis (NVS-NYSE, $73.12, Not Covered)
Pfizer (PFE-NYSE, $29.40, Not Covered)

Exhibit 18: Intercept Pharmaceuticals Income Statement

Intercept Pharma. (ICPT) **Oppenheimer & Co.**

($000's) [FY - DEC]

	2011A	2012A					2013E					2014E	2015E
	FY:11A	Q1A	Q2A	Q3A	Q4A	FY:12A	Q1A	Q2E	Q3E	Q4E	FY:13E	FY:14E	FY:15E
Revenues/Royalties on Product Sales	-	-	-	-	-	-	-	-	-	-	-	-	37,720
OCA - Primary Biliary Cirrhosis	-					-	-	-	-	-	-	-	37,720
OCA or INT-767 - Portal Hypertension	-					-	-	-	-	-	-	-	0
OCA or INT-767 PBAD	-					-	-	-	-	-	-	-	0
Licensing revenue and Milestones	1,805	759	759	523	405	2,446	405	400	400	400	1,605	1,600	1,600
Total revenues	**$ 1,805**	**$ 759**	**$ 759**	**$ 523**	**$ 405**	**$ 2,446**	**$ 405**	**$ 400**	**$ 400**	**$ 400**	**$ 1,605**	**$ 1,600**	**$ 39,320**
Cost of Goods	-	-	-	-	-	-	-	-	-	-	-	-	3,539
Gross profit	**1,805**	**759**	**759**	**523**	**405**	**2,446**	**405**	**400**	**400**	**400**	**1,605**	**1,600**	**35,781**
Operating expenses													
Research and development	11,426	3,060	5,018	3,318	4,787	16,183	4,833	4,929	5,028	5,128	19,918	22,906	23,364
Selling, general and administrative	4,209	1,059	944	991	2,183	5,177	2,397	2,541	2,617	2,695	10,250	15,375	24,000
Other	-												
Total expenses	**15,636**	**4,119**	**5,962**	**4,309**	**6,970**	**21,360**	**7,229**	**7,470**	**7,645**	**7,824**	**30,168**	**38,280**	**47,364**
Operating income	**(13,830)**	**(3,360)**	**(5,203)**	**(3,786)**	**(6,565)**	**(18,914)**	**(6,824)**	**(7,070)**	**(7,245)**	**(7,424)**	**(28,562)**	**(36,680)**	**(11,582)**
Revaluation of warrants	1,045	678	302	(1,418)	(24,187)	(24,625)	(3,683)	-	-	-	(3,683)	-	-
Other income (expense)	0	(750)	(942)	(1,000)	(130)	(2,822)	296	26	30	29	(3,683)	109	102
Pre-tax income	**(12,738)**	**(3,430)**	**(5,836)**	**(6,187)**	**(30,821)**	**(46,274)**	**(10,210)**	**(7,044)**	**(7,215)**	**(7,394)**	**(32,245)**	**(36,571)**	**(11,481)**
Income tax expense (benefit)	3,000	-	-	-	-	-	-	-	-	-	-	-	(1,722)
Net income	**($15,738)**	**($3,430)**	**($5,836)**	**($6,187)**	**($30,821)**	**($46,274)**	**($10,210)**	**($7,044)**	**($7,215)**	**($7,394)**	**($32,245)**	**($36,571)**	**($9,759)**
Basic shares outstanding	3,330	3,330	3,330	3,330	15,223	6,283	16,558	16,883	18,488	18,638	17,642	19,013	19,623
Diluted shares outstanding					18,197		19,423	19,748	21,353	21,503	20,507	21,878	22,545
GAAP EPS (basic and diluted)	**($4.73)**	**($1.03)**	**($1.75)**	**($1.86)**	**($2.02)**	**($7.36)**	**($0.62)**	**($0.42)**	**($0.39)**	**($0.40)**	**($1.83)**	**($1.92)**	**($0.43)**
Cash and Equivalents	$ 17,707	$ -	$ -	$ 36,049	$ 110,194	$ 110,194	$ 104,220	$ 153,014	$ 150,119	$ 147,047	$ 147,047	$ 126,916	$ 129,060

Source: Oppenheimer & Co. Inc., Company Reports

Exhibit 19: Intercept Pharmaceuticals Balance Sheet

Intercept Pharma. (ICPT)	2010A	2011A	2012A					2013E					2014E	2015E
($000's) [FY - DEC]	FY:10A	FY:11A	Q1A	Q2A	Q3A	Q4A	FY:12A	Q1A	Q2E	Q3E	Q4E	FY:13E	FY:14E	FY:15E
Assets														
Current Assets:														
Cash and cash equivalents		17,707			35,971	45,512	45,512	31,711	80,505	77,610	74,538	74,538	54,408	56,551
Investment securities		-			78	64,682	64,682	72,509	72,509	72,509	72,509	72,509	72,509	72,509
Accounts Receivable		-			-	-	-	-	-	-	-	-	-	539
Inventories		-			-	-	-	-	-	-	-	-	-	970
Deferred tax assets		-			-	-	-	-	-	-	-	-	-	393
Prepaid expense and other current assets		1,197			2,106	1,584	1,584	1,572	1,572	1,572	1,572	1,572	1,572	9,830
Total current assets	**-**	**18,904**	**-**	**-**	**38,155**	**111,778**	**111,778**	**105,793**	**154,586**	**151,691**	**148,619**	**148,619**	**128,489**	**140,791**
Fixed assets, net		311			157	149	149	135	137	138	139	139	140	136
Intangible assets, net		-				-	-	-	-	-	-	-	-	-
Deferred tax assets		-				-	-	-	-	-	-	-	-	-
Other non-current assets		255			258	252	252	269	269	269	269	269	269	269
Total assets	**-**	**19,470**	**-**	**-**	**38,570**	**112,179**	**112,179**	**106,196**	**154,992**	**152,098**	**149,027**	**149,027**	**128,897**	**141,196**
Liabilities														
Current liabilities:														
Accounts payable		1,504			4,090	3,746	3,746	3,226	3,029	3,100	3,172	3,172	2,622	3,893
Short-term portion of warrant liability					334	7,597		4,786	4,786	4,786	4,786	4,786	4,786	4,786
Accrued interest payable		-			-	-	-	-	-	-	-	-	-	-
Deferred revenue		2,446			1,622	1,622	1,622	1,622	1,622	1,622	1,622	1,622	1,622	1,622
Accrued expenses and other		82			-	-	-	-	-	-	-	-	-	786
Total current liabilities	**-**	**4,032**	**-**	**-**	**6,047**	**12,964**	**5,367**	**9,633**	**9,437**	**9,507**	**9,580**	**9,580**	**9,030**	**11,087**
Long-term portion of warrant liability		5,836			5,940	22,762	22,762	25,627	25,627	25,627	25,627	25,627	25,627	25,627
Long-term portion of deferred revenue		12,162			10,946	10,541	10,541	10,135	10,135	10,135	10,135	10,135	10,135	10,135
Long-term debt		-				-	-	-	-	-	-	-	-	-
Other		-				-	-	-	-	-	-	-	-	-
Total liabilities	**-**	**22,030**	**-**	**-**	**22,932**	**46,267**	**38,670**	**45,396**	**45,199**	**45,270**	**45,342**	**45,342**	**44,792**	**46,849**
Total stockholders' equity	**-**	**(2,560)**	**-**	**-**	**15,637**	**65,912**	**65,912**	**60,800**	**109,793**	**106,829**	**103,684**	**103,684**	**84,105**	**94,346**
Total liabilities and equity	**-**	**19,470**	**-**	**-**	**38,570**	**112,179**	**104,582**	**106,196**	**154,992**	**152,098**	**149,027**	**149,027**	**128,897**	**141,196**

Source: Oppenheimer & Co., Company Reports

Exhibit 20: Intercept Pharmaceuticals Statement of Cash Flows

Intercept Pharma. (ICPT)	2010A	2011A	2012A					2013E					2014E	2015E
($000's) [FY - DEC]	FY:10A	FY:11A	Q1A	Q2A	Q3A	Q4A	FY:12A	Q1A	Q2E	Q3E	Q4E	FY:13E	FY:14E	FY:15E
Net income (loss)	(15,087)	(12,738)	(2,680)		(12,953)	(28,011)	(43,643)	(10,210)	(7,044)	(7,215)	(7,394)	(31,863)	(36,571)	(9,759)
Adjustments														
Depreciation and amortization	480	411	74		178	(51)	201	23	13	14	15	64	71	104
Stock compensation expense	1,693	1,866	392		1,252	1,706	3,349	1,607	4,250	4,250	4,250	14,357	17,000	20,000
Revaluation of warrants	(672)	(1,045)	(678)		438	24,865	24,626	3,683	-	-	-	3,683	-	-
Impairment of bonds		-	-		-	-		-	-	-	-	-	-	-
Amortization of intangible asset		-	-		-	-		-	-	-	-	-	-	-
(Gain) loss from sale of assets	-	217	-		-	-		-	-	-	-	-	-	-
Deferred tax provision	-	-	-		-	-		-	-	-	-	-	-	-
Excess tax benefit from share based compensation	-	-	-		-	-		-	-	-	-	-	-	-
Other	-	-	-		192	118	310	-	-	-	-	-	-	-
Changes in operating assets and liabilities												-	-	
Cash flow from operations	**(13,657.823)**	**2,606**	**(3,893)**	**-**	**(11,457)**	**(399)**	**(15,749)**	**(5,636)**	**(2,978)**	**(2,880)**	**(3,057)**	**(14,551)**	**(20,051)**	**2,243**
Purchase of available for sale securities	87	(3)	(4)		120	(64,935)	(64,819)	(8,243)	-	-	-	(8,243)	-	
Purchase of fixed assets	(29)	(63)	(20)		(24)	5	(39)	(9)	(15)	(15)	(15)	(54)	(80)	(100)
Other	-	-				-		-				-		
Cash flow from investing	**57.859**	**(66)**	**(24)**	**-**	**96**	**(64,930)**	**(64,857)**	**(8,252)**	**(15)**	**(15)**	**(15)**	**(8,297)**	**(80)**	**(100)**
Proceeds from issuances of common stock	24,888	-			29,714	78,786	108,500	439	50,680			51,119	-	-
Proceeds from issuance of warrants	-	-				-		-				-	-	-
Payments of capital lease obligation	(270)	(250)	(59)		(82)	59	(82)	-				-	-	-
Proceed from the exercise of options and warrants	-	-				-		70				70	-	-
Proceeds from the issuance of convertible notes	-	-	-	-	-	-		-	-	-	-	-	-	
Excess tax benefit from stock-based compensation exp	-	-				-		678				678	-	-
Other	-	-				-		-				-	-	-
Cash flow from financing	**24,618.090**	**(250)**	**(59)**	**-**	**29,632**	**78,845**	**108,418**	**1,187**	**50,680**	**-**	**-**	**51,867**	**-**	**-**
Effect of FX	(29)	(6)					(7)							
Net increase (decrease) in cash	10,989	2,284	(3,976)	-	18,271	13,516	27,811	(12,701)	47,687	(2,895)	(3,072)	29,019	(20,131)	2,143
Cash and equivalents at beginning	4,435	15,424	17,707	13,732	13,732	32,003	17,707	45,519	32,818	80,505	77,610	45,519	74,538	54,408
Cash and equivalents at end	**15,424**	**17,707**	**13,732**	**13,732**	**32,003**	**45,519**	**45,519**	**32,818**	**80,505**	**77,610**	**74,538**	**74,538**	**54,408**	**56,551**

Source: Oppenheimer & Co., Company Reports

Exhibit 21: OCA/INT-767 Market Model (2015-2025)

($000's) [FY - DEC]	2015E	2016E	2017E	2018E	2019E	2020E	2021E	2022E	2023E	2024E	2025E
Revenues											
OCA - PBC	37,720	141,343	200,163	324,948	482,768	556,992	573,813	591,142	608,995	627,386	646,334
OCA (or INT-767) - PH	0	0	0	126,284	231,857	321,632	439,197	565,735	588,591	612,370	637,110
OCA (or INT-767) - NASH	0	0	0	28,653	59,620	124,058	193,605	268,569	349,274	436,061	529,291
OCA (or INT-767) - P-BAD	0	0	0	33,382	108,264	178,454	297,394	494,379	509,309	524,690	540,536
Total OCA	**37,720**	**141,343**	**200,163**	**513,266**	**882,510**	**1,181,136**	**1,504,009**	**1,919,825**	**2,056,169**	**2,200,508**	**2,353,271**
Total revenue	**$37,720**	**$141,343**	**$200,163**	**$513,266**	**$882,510**	**$1,181,136**	**$1,504,009**	**$1,919,825**	**$2,056,169**	**$2,200,508**	**$2,353,271**

Source: Oppenheimer & Co. Inc., Company Reports

Investment Thesis
We believe ICPT's lead asset, obeticholic acid (OCA), which is in a Phase 3 trial for the treatment of primary biliary cirrhosis (PBC), and in earlier stages of development for the treatment of portal hypertension, bile acid diarrhea and NASH, has well-defined commercial potential and an overall risk/reward profile that appears highly favorable at the current share price. Specifically, we believe OCA will be able fill a much-needed role as a second-line therapy in PBC, as the current standard of care, Urso, is be effective in only ~40%-50% of patients. We also believe OCA's development in other liver diseases represents significant upside potential for ICPT.

Price Target Calculation
Our $60 price target is based on a sum-of-the-parts analysis for ICPT's lead asset, OCA, being developed for the treatment of PBC, portal hypertension, and bile acid diarrhea. We value ICPT using a probability-adjusted net present value (pNPV) approach, calculating anticipated profits from OCA (or the follow-on drug INT-767) through 2026, discounted at 10.5% with no terminal value. We then adjust for clinical and regulatory risk by assigning an estimated probability of success (i.e., reaching commercialization), based on stage of clinical development and our assessment of the available clinical data and characteristics of the proposed indication. Specifically, we estimate a $40/share valuation for OCA in PBC assuming a 74% chance of success and peak sales of ~$650M; $11/share for OCA/INT-767 in portal hypertension assuming a 28% chance of success and peak sales of ~$640M; and $9/share for OCA/INT-767 in bile acid diarrhea assuming a 33% chance of success and peak sales of $540M.

Key Risks to Price Target

Clinical Risk. Intercept's drugs will be required to demonstrate efficacy and safety in clinical trials before they can be approved by regulatory agencies.

Regulatory Risk. ICPT has yet to submit for or receive approval for any of its drugs in the US, and may face difficulties in doing so, potentially delaying commercialization.

Commercialization Risk. Despite ICPT's ability to potentially attain approval of their development candidates, the company may face unpredictable commercialization challenges.

Intellectual Property Risk. There is inherent uncertainty in both the interpretation of patent claims and the application of patent law, regardless of the apparent strength of ICPT's patent portfolio.

Manufacturing Risk. ICPT does not possess its own manufacturing capabilities to clinically or commercially supply sufficient quantities of its drugs.

Competitive Risk. The indications being targeted by ICPT are also being targeted by several competitors, some with superior resources.

Financing Risk. While we believe ICPT is sufficiently capitalized to reach significant value inflection points, any unexpected clinical or regulatory setbacks may prompt capital raising to before ICPT is able to generate sufficient revenues from the commercial activities.

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Stock Prices as of June 20, 2013

Antares Pharma, Inc. (ATRS - NASDAQ, $4.10, OUTPERFORM)
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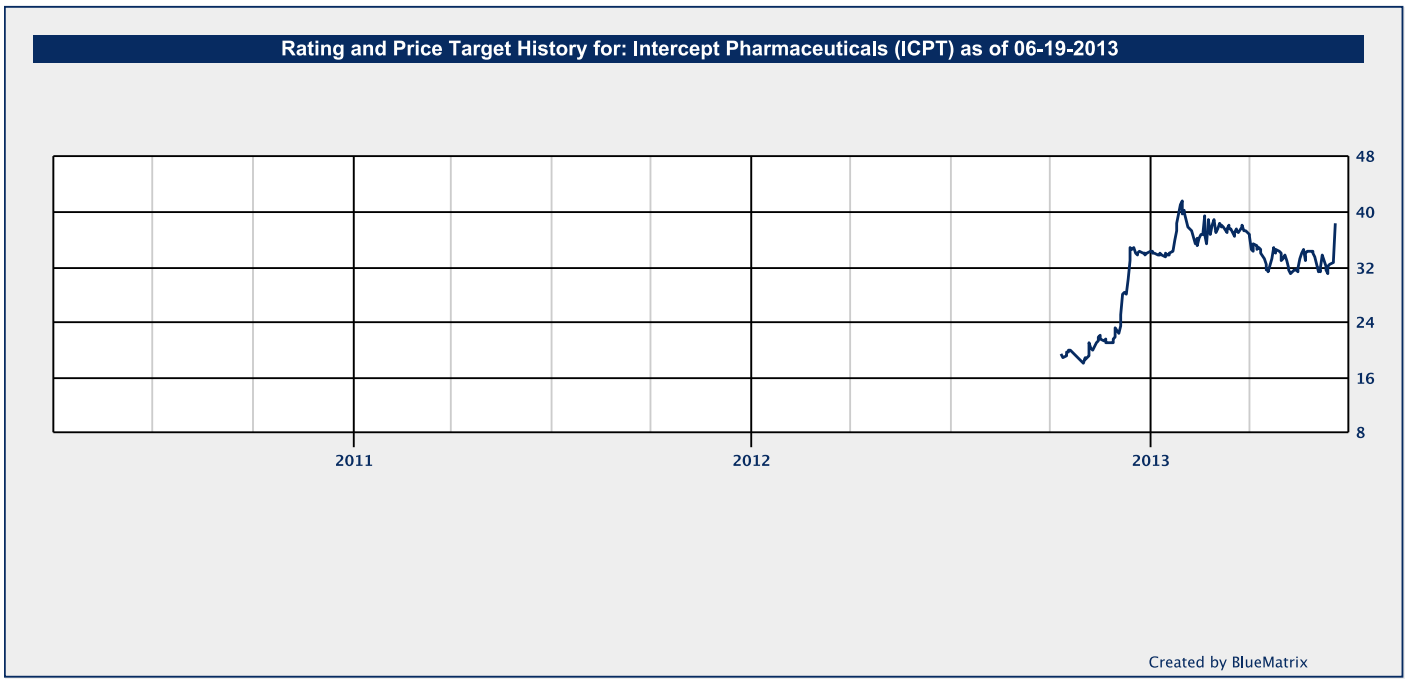
The Medicines Company (MDCO - NASDAQ, $33.52, OUTPERFORM)
Questcor Pharmaceuticals (QCOR - NASDAQ, $47.46, PERFORM)
ViroPharma (VPHM - NASDAQ, $28.80, OUTPERFORM)



All price targets displayed in the chart above are for a 12- to- 18-month period. Prior to March 30, 2004, Oppenheimer & Co. Inc. used 6-, 12-, 12- to 18-, and 12- to 24-month price targets and ranges. For more information about target price histories, please write to Oppenheimer & Co. Inc., 85 Broad Street, New York, NY 10004, Attention: Equity Research Department, Business Manager.

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