Janney CAPITAL MARKETS

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Intercept Pharmaceuticals, Inc.

ICPT - BUY

February 20, 2013

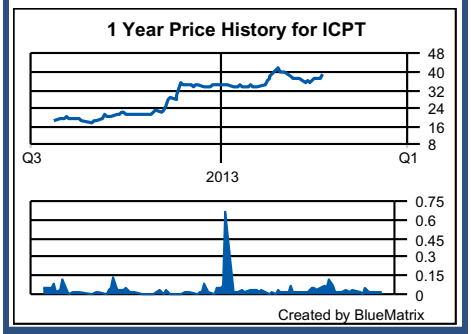
Specialty Pharmaceuticals

Intercept Pharmaceuticals, Inc.

(ICPT) - BUY

Price:	**$39.53**
Fair Value Estimate:	$48.00
52-Week Range:	$15.00-$42.67
Market Cap (MM):	$652
Shr.O/S-Diluted (mm):	16.5
Average Daily Volume:	35,250
Dividend:	NA
Book Value:	$0.52

FYE: Dec	2012E	2013E	2014E
Revenue (M):	$2.6E	$2.4E	$2.4E
Quarterly Revenue (M):			
Q1	$0.8A	$0.6E	$0.6E
Q2	$0.8A	$0.6E	$0.6E
Q3	$0.5A	$0.6E	$0.6E
Q4	$0.5E	$0.6E	$0.6E
FYE: Dec	2012E	2013E	2014E
EPS:	$(1.11)E	$(1.67)E	$(1.87)E
Prior EPS:	NC	NC	NC
Quarterly EPS:			
Q1	$(0.18)A	$(0.40)E	$(0.46)E
Q2	$(0.26)A	$(0.41)E	$(0.46)E
Q3	$(0.32)A	$(0.42)E	$(0.47)E
Q4	$(0.35)E	$(0.43)E	$(0.48)E



Equity Research
Basic Report

Initiate with a BUY, $48 FV: Building Better Livers through Bile Acid Chemistry

INVESTMENT CONCLUSION:

We are initiating coverage on Intercept Pharmaceuticals (ICPT) with a Buy rating and a $48 fair value estimate. ICPT is a specialty pharmaceutical company focused on novel therapeutics to treat chronic liver diseases through bile acid chemistry. ICPT targets both orphan and more prevalent liver diseases that have limited FDA approved treatment options. The lead product, obeticholic acid (OCA), is a bile acid analog that appears to have broad liver-protective properties. The lead indication is primary biliary cirrhosis (PBC), an orphan indication primarily affecting women where the standard of care fails ~50% of the time. OCA is currently in phase 3 trials (data mid-2014). ICPT is also investigating OCA for treating NASH, a significantly larger treatment opportunity. OCA is in phase 2 trials for NASH (data 2H14).

KEY POINTS:

- **Obeticholic acid (OCA) for primary biliary cirrhosis (PBC).** OCA is a first-in-class FXR agonist targeting the significant unmet medical need to treat PBC Urso failures. With approximately 30,000 patients in the US & EU each and pricing in the ~$100,000/year typical of an orphan drug, we believe that OCA could be north of $600M by 2018 in the US alone. We believe that the PBC phase 3 trial results (due mid-2014) are set up for success, given the similarity between the phase 3 trial design and the design of the successful phase 2 trial. The key question remains if the FDA will ultimately accept the surrogate endpoints of lowering alkaline phosphatase (ALP) while maintaining normal bilirubin as predictive of the clinical outcome of maintaining liver function. We believe the FDA will accept those endpoints.

- **If OCA is approved for PBC, nonalcoholic steatohepatitis (NASH) is next.** NASH is similar to alcoholic cirrhosis of the liver, and the advanced form of NASH affects ~8M people in the US alone with no FDA approved treatments available. OCA is currently in a phase 2 NASH trial sponsored by the National Institute of Diabetes and Digestive and Kidney Diseases. We anticipate top-line data in 2H14 and should OCA demonstrate the magnitude of liver sparing activity (as measured by changes in NAFL activity score & fibrosis) in phase 2 NASH trials like it did in phase 2 PBC trials, we believe that the NASH indication could dwarf the PBC indication, even if NASH is unlikely to be priced like an orphan drug.

- **Deep pipeline of follow-on indications.** ICPT is looking at using OCA for the treatment of a variety of liver-related conditions such as: portal hypertension, which has no FDA approved treatment options and has shown some activity in a phase 2a open label trials with additional safety data expected in 1H13; and bile acid diarrhea, which is currently in a phase 2a open label trial with data expected in 1H13. Separately, ICPT is also looking at INT-767, a more potent FXR agonist for diabetic nephropathy & chronic kidney disease, which could be up to 5x as potent as OCA. ICPT anticipates filing an IND for INT-767 in 2H13.

Research Analyst Certifications and Important Disclosures are on pages 17 - 19 of this report

Summary and Investment Thesis

We are initiating coverage on Intercept Pharmaceuticals (ICPT) with a Buy rating and a $48 fair value estimate. ICPT is a specialty pharmaceutical company focused on novel therapeutics to treat chronic liver diseases through bile acid chemistry. ICPT targets both orphan and more prevalent liver diseases that have limited FDA approved treatment options. The lead product obeticholic acid (OCA) is a bile acid analog which appears to have broad liver-protective properties. The lead indication is primary biliary cirrhosis (PBC), an orphan indication primarily affecting women where the standard of care fails ~50% of the time. OCA is currently in phase 3 trials (data mid-2014). ICPT is also investigating OCA for treating NASH, a significantly larger treatment opportunity. OCA is in phase 2 trials for NASH (data 2H14).

Top Reasons to Own ICPT:

1. **Obeticholic acid (OCA) for primary biliary cirrhosis (PBC).** OCA is a first-in-class FXR agonist targeting the significant unmet medical need to treat PBC Urso failures. With approximately 30,000 patients in the US & EU each, and pricing in the ~$100,000/year typical of an orphan drug, we believe that OCA could be north of $600M by 2018 in the US alone. We believe that the PBC phase 3 trial results (due mid-2014) are set up for success, given the similarity between the phase 3 trial design and the design of the successful phase 2 trial. The key question remains if the FDA will ultimately accept the surrogate endpoints of lowering alkaline phosphatase (ALP) while maintaining normal bilirubin as predictive of the clinical outcome of maintaining liver function. We believe the FDA will accept those endpoints.

2. **If OCA is approved for PBC, nonalcoholic steatohepatitis (NASH) is next.** NASH is similar to alcoholic cirrhosis of the liver, and the advanced form of NSAH affects ~8M people in the US alone, with no FDA approved treatments available. OCA is currently in a phase 2 NASH trial sponsored by the National Institute of Diabetes and Digestive and Kidney Diseases. We anticipate top line data in 2H14, and should OCA demonstrate the magnitude of liver sparing activity (as measured by changes in NAFL activity score & fibrosis) in phase 2 NASH trials like it did in phase 2 PBC trials, we believe that the NASH indication could dwarf the PBC indication, even if NASH is unlikely to be priced like an orphan drug.

3. **Deep pipeline of follow-on indications.** ICPT is looking at using OCA for the treatment of a variety of liver-related conditions such as: portal hypertension, which has no FDA approved treatment options, and has shown some activity in a phase 2a open label trials with additional safety data expected in 1H13, and bile acid diarrhea, which is currently in a phase 2a open label trial with data expected in 1H13. Separately, ICPT is also looking at INT-767, a more potent FXR agonist for diabetic nephropathy & chronic kidney disease, which could be up to 5x as potent as OCA. ICPT anticipates filing an IND for INT-767 in 2H13.

4. **IPO provides cash through the phase 3 PBC trial in mid-2014.** With their very successful IPO behind them, ICPT has secured sufficient financing to see the results of the phase 3 trial of OCA to treat PBC. We estimate that ICPT will end 2012 with ~$80M in cash following its $86M IPO in 4Q12. We believe it is likely that ICPT may choose to accelerate its earlier stage clinical programs, which would require additional funding.

Upcoming potential catalysts

EXHIBIT 1

Event	Expected Timing
Phase 2a PESTO trial (portal hypertension) results for 2nd cohort	nid-2013
Phase 2a OBADIAH trial (Bile Acid Diarrhea) results	mid-2013
Phase 3 POISE trial (PBC) results	mid-2014
Phase 2b FLINT trial (NASH) results	2H14

Source: Janney estimates

Valuation

We value ICPT as a sum-of-parts with a $48/share based on a sum-of-the-parts. We value the US sales of OCA at $29/share based on a 3.5x multiple of 2018 US PBC sales and the EU royalties at $4/share based on a 5x multiple of 2018 EU PBC royalties, both discounted 5 years at 25% to account for the risks remaining in this program. We value OCA for NASH at $9/share based on a 3.5x multiple of 2020 sales of $1.1B discounted 7 years at 50% to account for risk. Our remaining $6/share value is based on cash (end 2013) and technology value. We estimate a ~$90M raise in 2013.

EXHIBIT 2

Sum-of-the-parts valuation: ICPT		
Segment	Valuation (000's)	Per share value
OCA for PBC in the US	$698,755	$33
OCA for NASH in the US	$225,332	$9
Cash (end of '13E) & tech value	$158,068	$6
	$1,180,845	**$48**
2013 fully diluted shares out		24,488

Source: Janney estimates

EXHIBIT 3

Multiple analysis						
2018 PBC revs ($M)	multiple	value ($M)	years	disc rate	disc val. ($M)	disc val.
$609	**3.5**	$2,132	5	25%	$699	**$29**
2018 PBC royalties ($M)	multiple	value	years	disc rate	disc val.	disc val.
$60	**5.0**	$301	5	25%	$99	**$4**
2020 NASH revs ($M)	multiple	value	years	disc rate	disc val.	disc val.
$1,100	**3.5**	$3,850	7	50%	$225	**$9**

Source: Janney estimates

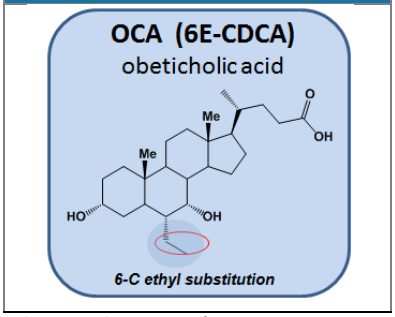
Company Description

ICPT Pharmaceuticals is a biopharmaceutical company focused on the development and commercialization of novel therapeutics to treat chronic liver disease by utilizing its expertise in bile acid and chemistry. ICPT's goal is to develop treatments for both orphan liver diseases - more prevalent liver diseases - for which there are currently limited therapeutic solutions. Its lead product candidate is obeticholic acid (OCA), is a bile acid analog derived from the primary human bile acid chenodeoxycholic acid (CDCA). OCA also selectively binds to and induces activity in the farnesoid X receptor (FXR), which ICPT believes has broad liver-protective properties.

ICPT initially developed OCA for primary biliary cirrhosis (PBC) as a second line treatment for patients who have an inadequate response to or who are unable to tolerate the current standard of care. ICPT is also looking at portal hypertension, nonalcoholic steatohepatitis (NASH) and bile acid diarrhea as other possible indications.

The company is currently running a 12 month, multi-center, 3 arm, phase 3 clinical trial in for the PBC indication, recently reported positive phase 2a open-label data for 10mg OCA for portal hypertension, is enrolling a phase 2b clinical trial for NASH in collaboration with the U.S. National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK). Investigators at the Imperial College of London, looking at OCA for the treatment of bile acid diarrhea, initiated a phase 2a clinical trial in July 2012 with data possible by mid-2013.

EXHIBIT 4 – OCA



Source: Company documents

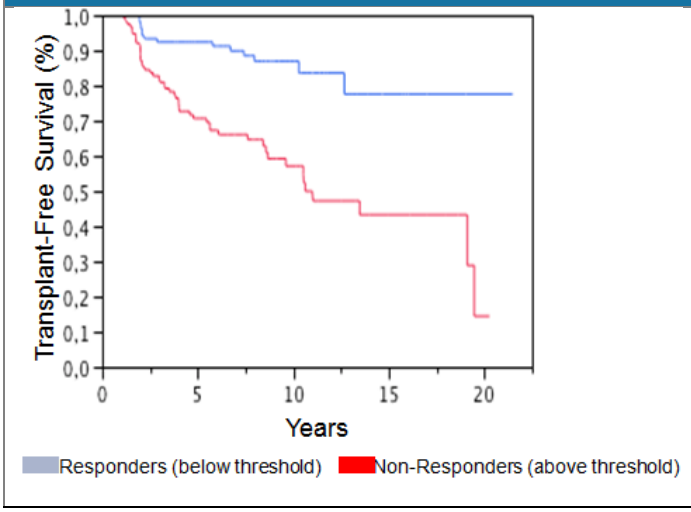
OCA for Primary Biliary Cirrhosis - $33/share

Key Issues: It remains unknown if the FDA will ultimately accept the surrogate endpoint of improvement of alkaline phosphatase (ALP) & normal bilirubin as being reasonably predictive of clinical benefit. We believe it is predictive, and that the FDA will ultimately agree.

Background on Primary Biliary Cirrhosis (PBC)

PBC is a chronic liver disease that primarily results from autoimmune destruction of the small bile ducts within the liver. White blood cells known as T lymphocytes, which are a part of the immune response system, begin to accumulate in the liver. These cells invade and destroy the cell lining of the small bile ducts, causing irritation and inflammation. With the ducts blocked, bile acid builds up in the liver, slowly progressing to toxic levels. As the inflammation of the ducts spreads, it also destroys the nearby liver cells. As these cells are destroyed, they are replaced by scar tissue (fibrosis) that contributes to cirrhosis, which can lead to diminishing liver function.

EXHIBIT 5 – lowering ALP improves survival



Source: Intercept company documents

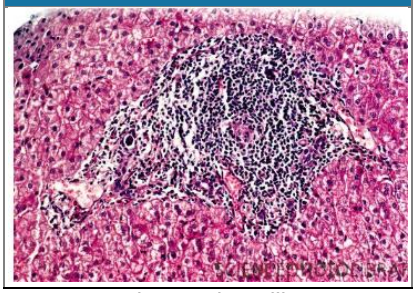
Disease progression in PBC varies significantly but usually is relatively slow, with median survival in untreated patients of 7.5 years if symptomatic at diagnosis and up to 16 years if asymptomatic at diagnosis. Diagnosis for PBC is confirmed through a routine liver function test for alkaline phosphatase (ALP) which any primary care physician can do. ALP is the main biomarker for treatment response as well as prognosis (see Exhibit 5).

Standard Treatment with Urso only ~50% effective

Currently, the only FDA approved treatment for PBC is Urso (ursodiol), which is the standard initial course of therapy for all PBC patients. Ursodiol (ursodeoxycholic acid), is a naturally occurring bile acid found in small quantities in humans and it is the least detergent of the various types of bile acids that make up the bile pool. Ursodiol assists the liver by diluting the more detergent acids and thus making the pool less toxic. The typical daily dose of ursodiol is approximately one gram spread over divided doses. Of note is that even though ursodiol is the current standard of care, at least 50% of patients fail to respond adequately to treatment.

There are limited options for these patients and though a full liver transplant can be curative, many fail to receive a donor organ in time. Organ transplantation also comes with severe clinical risks such as infection and rejection, and with PBC, the disease reoccurrence rate is as high as 18% at five years and up to 30% at ten years after transplant.

EXHIBIT 6 – Image of PBC



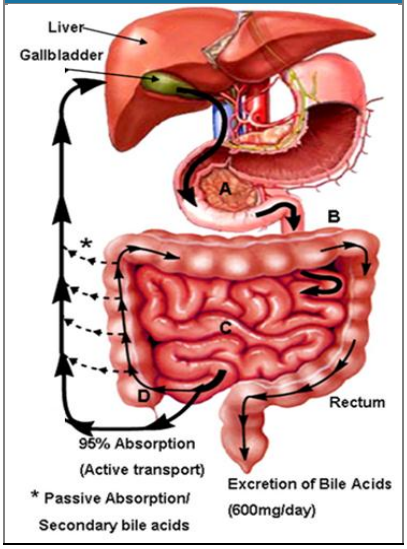
Source: Science Photo library.com

Patient Population

While PBC is rare, it is the most common cholestatic liver disease and is the fifth most common cause of liver transplant in the United States. It also accounts for approximately 2% of deaths attributed to cirrhosis. The majority (~90%) of patients are women with approximately 1 in 1,000 women over the age of 40 afflicted with the disease. The mean age of diagnosis is about 40 years old. Most people with PBC are diagnosed before symptoms even begin with the most common symptoms being fatigue and pruritus, or itchy skin.

ICPT estimates that there are approximately 300,000 people with PBC in developed countries, with ~60,000 in the US which have been diagnosed and are on the standard treatment, ursodiol. As previously noted, 50% of these patients do not respond to this drug which means that 30,000 PBC patients are eligible for treatment with OCA. Approximately 30% of non-responders to Urso progress to liver failure, transplant, or death within 5 years, so there is a significant unmet medical need for new therapies. Disease progression in PBC varies significantly but usually is relatively slow, with median survival in untreated patients of 7.5 years if symptomatic at diagnosis and up to 16 years if asymptomatic at diagnosis.

EXHIBIT 7 – Enterohepatic recirculation



Source: 2009 American Society for Biochemistry and Molecular Biology

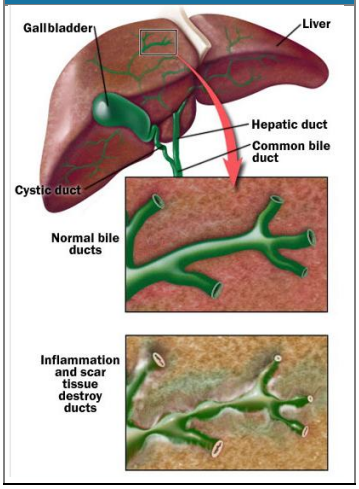
The Solution: OCA

The liver performs many essential functions including the regulation of bile acid metabolism. Large amounts of bile acids are secreted into the small intestine every day but only a small quantity is actually lost from the body. The vast majority of bile acids are reabsorbed back into the gallbladder.

In the liver, ingested cholesterol is synthesized into two primary bile acids. These acids are secreted into the bile canalicular lumen for storage in the gallbladder. Once food enters into the small intestines, the gallbladder releases bile acids which act as a detergent, emulsifying and breaking down the ingested material thus creating more surface area for digestive enzymes to work with.

Once the process reaches the end of the small intestines known as the ileum, a system of small veins passively absorbs the acids into the larger main portal vein which delivers the bile acids back into the liver. Liver cells known as hepatocytes, which are the main cells of the liver extract the bile acids and actively secrete them into the bile ducts and back into the gallbladder. This digestive cycle of bile flow from gallbladder to intestine to liver and back is called the enterohepatic recirculation of bile (see Exhibit 7 below).

EXHIBIT 8 – Bile duct damage



Source: Mayoclinic.com

The amount of bile acid produced is a tightly regulated process within the liver and intestines. Mediation is accomplished through dedicated receptors. One of the best understood is the farnesoid X receptor (FXR) which is a nuclear receptor that is activated by high concentrations of bile acid. Once triggered, it inhibits bile acid synthesis and promotes bile clearance from the liver. This makes FXR an attractive target for the treatment of liver diseases.

OCA is a novel, oral, first-in-class FXR agonist that is believed to have broad liver-protective properties and may effectively counter a variety of chronic liver diseases. Not only does ICPT believe that OCA will prove effective in lowering alkaline phosphatase (ALP) levels, but as an FXR agonist, OCA is ~100x more potent than other known FXR agonist, chenodeoxycholic acid (CDCA). Finally, as a once daily dose medication, patient compliance should be higher compared the current multi-doses needed for ursodiol.

Phase 2 Clinical Trials: Monotherapy and Combination therapy

Monotherapy Therapy:
In the monotherapy trail, 59 patients participated in the double-blind, placebo controlled Phase 2 trail. The trail measured OCA dosages of 10mg and 50mg as compared to placebo. Again, at the end of the 12-week period, patients treated with OCA experienced statistically significant reductions in ALP levels. Mean ALP levels saw a 38%-45% reduction with even greater reductions (63%-75%) in GGT as well as significant improvements in ALT and bilirubin levels (see Exhibit 10 below).

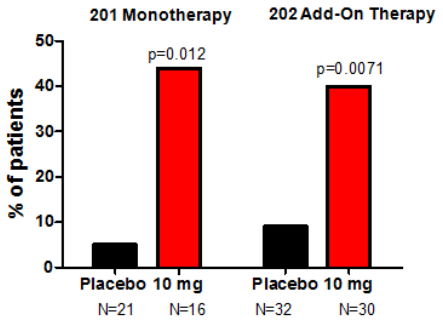
Combination Therapy:
ICPT has completed a double-blind, placebo-controlled Phase 2 clinical trial of OCA that involved 165 patients with PBC. The trail evaluated three doses of OCA (10mg, 25mg and 50mg) or placebo in patients with high ALP levels currently on ursodiol but not getting a good response to the drug. The patients continued with their urodiol regiment throughout the 12-week period. At the end of the study, all three doses of OCA in conjunction with ursodiol produced statistically significant reductions in ALP levels as compared to placebo with ursodiol. The mean ALP level reduction in OCA-treated patients was 21%-25%. In addition, patients who had been given OCA also experienced significant decreases in other relevant liver enzymes such as gamma glutamyl transferase (GGT), aspartate transaminase (AST), and alanine transaminase (ALT) (see Exhibit 11 below). In both trials, itching or pruritus was the most common side effect with severity increasing with dosage. See Exhibit 9 for a comparison of the ALP reductions for both the monotherapy and the combo-therapy phase 2 trials.

Phase 3 Preview?
Data pulled from the 10mg patient population from both Phase 2 trials who would have met the Phase 3 POISE trial inclusion criteria showed that after 12-weeks of treatment, ~40%-45% of OCA-treated patients would have met the Phase 3 primary endpoint (see Exhibit 9). While it's premature to draw conclusions about a phase 3 trial from phase 2 results, we would note that the in many ways the phase 3 trial is essentially a larger repetition of the successful phase 2 trial. While not a guarantee, we believe this design gives the phase 3 trial as much opportunity for success as possible.

Phase 3 Clinical Trial
ICPT is currently enrolling a Phase 3 POISE trial. In this trial, eligible PBC patients taking a stable therapeutic dose of ursodiol will continue with their ursodiol treatment and be

EXHIBIT 9 – Mono/combo therapy phase 2 results

% Patients who had ALP <1.67x ULN and total bilirubin ≤ULN and ≥15% reduction in ALP value at Day 85/ET



Patients included if ALP ≥ 1.67x ULN or total bilirubin > ULN at baseline.
*Statistical comparison was performed using the Fishers Exact Test.

Source: Intercept company documents

randomized into one of three arms of 60 patients each, adding either: 1) 10mg; 2) 5mg of OCA titrating up to 10mg; or 3) placebo. The double-blind phase of the trial is designed to be 12 months in duration with patients having the option upon completion, to continue in an open label, long-term safety extension phase for another five years.

The primary endpoint of the Phase 3 POISE study is the achievement of both a reduction in ALP level to below threshold of 1.67x upper limit normal (ULN), with a minimum of 15% reduction in ALP level from baseline, and a normal bilirubin level, compared to placebo after 12 months of therapy. Bilirubin is a marker of liver function and is monitored to provide an indication of how well the liver is functioning. PBC patients whose disease is progressing have persistently elevated levels of ALP and other liver enzymes, with abnormal bilirubin levels heralding more advanced disease (see Exhibit 12 below).

EXHIBIT 10

Phase 2: Monotherapy OCA for patients with PBC	
Aim	Efficacy & safety study of OCA (Obeticholic acid) in patients with primary biliary cirrhosis compared to placebo
Design	Multi-site (US &Canada), double-blind, randomized, placebo-controlled; Patientswith baseline ALP >1.5x ULN; Pts never taken Ursodiol or no ursodiol for 3-mo before trial
Dosing	OCA (10 mg, 50mg) administered orally, once daily for 12 weeks, 2 week follow-up
Endpoints	1': Reduction in alkaline phosphatase (ALP) levels - % change from baseline 2': Hepatocellular Injury and Liver Function: GGT - % change from baseline 2': Hepatocellular Injury and Liver Function: ALT - % change from baseline 2':Hepatocellular Injury and Liver Function: AST - % change from baseline 2': Hepatocellular Injury and Liver Function: Bilirubin - % change from baseline
Patients	N=59, 1:1 active vs placebo
Results	March 31, 2011 - Reduction of ALP was significant with p<0.0001 on both doses. Primary End Point: Change: 10mg: - .45% 50mg: -38% placebo: 0% Signif Improvements in GGT, CRP and IgM2 Side-effects: Pruritus (itching) - common and increased with dose

Source: Company Reports

EXHIBIT 11

Phase 2: Combotherapy OCA for patients with PBC	
Aim	Efficacy & safety study of OCA (Obeticholic acid) in patients with primary biliary cirrhosis compared to placebo
Design	Multi-site (North America and Europe), double-blind, placebo-controlled, randomized, dose response
Dosing	OCA (10 mg, 25mg, 50mg) administered orally fpr 12 weeks, 2 week follow-up
Endpoints	1': Reduction in Alkaline Phosphate (AP) levels - % change from baseline 2': Hepatocellular Injury and Liver Function: GGT - % change from baseline 2': Hepatocellular Injury and Liver Function: ALT - % change from baseline 2': Plasma Trough Concentrations of INT-747 and its major known metabolites
Patients	N=165, 1:1 active vs placebo
Results	April 13, 2010 - Reduction of AP was significant with p<0.0001 on both doses. Primary End Point: Lowered AP by 21-24.7% - Absolute -66-77U/L compared to %U/L placebo GGT reduced 48-63% compared to 7% increase in placebo (p<0.0001) ; ALT reduced 21-35% with no change in placebo(p<0.0001) Side-effects: Pruritus (itching) - notable with half of the patients. 50mg dose had 16 drop out (24% of the group)

Source: Company Reports

FDA and Accelerated Approval

Assuming the phase 3 POISE trial succeeds, ICPT plans to submit an NDA for US approval for OCA and an MAA European approval. The EMA has agreed to accept their current clinical program of reviewing for approval based on surrogate endpoints (ALP & bilirubin) based on written scientific advice that the EMA themselves have given.

The FDA remains a bit murkier. ICPT intends to request that the POISE trial primary endpoint be accepted as a basis for approval of OCA under the FDA's accelerated approval regulation that enables the use of surrogate endpoint reasonably likely to predict a clinical benefit. This means that ICPT must offer the FDA compelling evidence that a biochemical

response, namely a decrease in ALP levels and an increase to normal in bilirubin levels, leads to an increase in survival of PBC patients without the need for a liver transplant. ICPT is pursuing this channel of approval as a clinical endpoint of liver transplantation or death is highly unfeasible.

EXHIBIT 12

Phase 3: POISE - OCA (obeticholic acid) for primary biliary cirrhosis (PBC)	
Aim	Efficacy & safety study of OCA (Obeticholic acid) in patients with primary biliary cirrhosis compared to placebo
Design	Multicenter, random, 2x blind, 12 months, 5mg or 10mg OCA vs placebo
Dosing	OCA (5-10 mg) vs. placebo, OCA 5mg for 6 months the titrating up to 10mg, 5mg-10mg for 12 months; All patients eligible to roll over to 5 yr LT safety extension
Endpoints	1': Composite endpoint Alkaline Phosphate (ALP) below than 1.67x upper limit of normal & at least 15% reduction from baseline, normal bilirubin 2': Alkaline Phosphatase response rates of 10%, 20% and 40% change 2': Alkaline Phosphate less than or equal to 3x upper limit normal and aspertate aminotransferase less than or equal to 2x upper limit normal bilirubin 2': Alkaline Phosphate less than or equal to 1.5x upper limit normal and aspertate aminotransferase less than or equal to 1.5x upper limit normal bilirubin 2': Bilirubin and albumin normal limits
Patients	N=180, 1:1 active vs placebo
Results	Enrollment completed 12/19/12; Results expected mid-2014

Source: Company Reports

With the accelerated approval process, ICPT will conduct a phase 3 clinical outcomes trial to confirm the clinical benefit predicted by the biochemical therapeutic response (decrease ALP, normal bilirubin). This phase 3 trial only has to be "substantially underway" at the time of the NDA submission and would be completed after accelerated approval. ICPT has been in discussions with the FDA about the details of such a clinical trial, and they are planning to initiate the trial as early as the second half of 2013. In the meantime, the FDA can approve OCA, assuming they agree that the reduction of ALP & normal bilirubin is in fact predictive of clinical benefit.

ICPT will also be relying on a number of published clinical studies that have demonstrated that reductions in these biomarkers are predictive of positive clinical outcomes. Aside from the base of published studies, ICPT is also sponsoring an independent study (the "PBC Supergroup") involving more than ten leading academic PBC centers in Europe and North America that are pooling their long-term patient data to further substantiate the POISE trial endpoint. The PBC Supergroup is anticipated to submit data of at least 4,000 patients in 1H13. Also a recent study in the Journal of Gastroenterology of 2,353 patients (Sex and Age Are Determinants of the Clinical Phenotype of Primary Biliary Cirrhosis and Response to Ursodeoxycholic Acid; Carbone et al) also demonstrated that surrogate endpoints are predictive of clinical outcomes.

EXHIBIT 13 – Ultra Orphan pricing

Drug	Company	Symbol	Indication	Launch Date	2011 Cost/ Year (000)	2011 Revenues (M)
Soliris	Alexion	ALXN	Paroxysmal Nocturnal Hemoglobinuria	2007, 2011	$409	$783
Eleprase	Shire	SHPGY	Hunter's Syndrome	2006	$375	$465
Cinryze	Viropharma	VPHM	Hereditary Angioedema	2009	$350	$251
Naglazyme	Biomarin	BMRN	Maroteaux-Lamy Syndrome	2005	$365	$225
Adagen	Sigma-Tau		ADA Deficiency	1990	$360	NA
Myozyme	Sanofi	SNY (GENZ)	Pompe's Syndrome	2006	$300	$128
Gattex	NPS Pharma	NPSP	Short bowel syndrome	2013	$295	NA
Kalydeco	Vertex	VRTX	Cystic Fibrosis	2012	$294	NA
Arcalyst	Regeneron	REGN	Inflammatory disorders	2008	$260	$20
Aldurazyme	Biomarin	BMRN	Hurler syndrom	2003	$250	$83
Folotyn	Allos	ALTH	T-cell lymphoma	2009	$220	$50
Cerezyme	Sanofi	SNY (GENZ)	Gaucher's Disease	1994	$215	$727
Fabrazyme	Sanofi	SNY (GENZ)	Fabry's Disease	2003	$200	$175
Acthar	Questcor	QCOR	Multiple Sclerosis, Nephrotic Syndrome, Infantile Spasms, Dermatomyositis/Polymyositis	1952	$50-$215	$216

Source: Company reports, Janney estimates

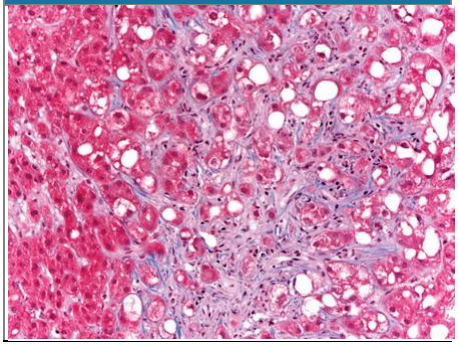
Pricing likely in the orphan drug range

One of the surprising developments over the past few years in pharma & biotech has been the pricing power of drug companies with orphan indications. Long relegated to the developmental backwaters as unprofitable, more recently a number of new, niche-focused orphan drugs (ie: less than 200,000 patients in the US) have appeared on the market. And even more recently the "ultra-orphan" drugs (not strictly defined, but generally recognized as less than 10,000 patients in the US) have surprised many by their ability to command astronomical prices (see Exhibit 12 – Ultra Orphan pricing). While the top-line pricing numbers for some of these products (ie: Soliris at $410,000/year) are stunning, since there are so few patients for ultra orphan indications there has been relatively little push-back from managed care as the drug is unlikely to break the bank. Combine this with the small trial sizes to get FDA approval, 7 years of exclusivity on approval, and typical pharma margins (~10%-12% COGS) the orphan drug space has become one of the most interesting areas for drug development in recent years, with ~200 orphan drugs in development.

There are approximately ~30,000 patients who fail Urso and are candidates for OCA treatment, which places PBC squarely as an Orphan Indication, and OCA has received Orphan Drug designation in both the US & EU. But given the patient population, small though it is, it's unlikely that OCA will be priced in the "ultra-orphan" $200,000-$300,000 range like Soliris, Eleprase, etc. But with only 30,000 patients OCA is close to ultra-orphan, and given the unmet medical need for Urso failures who will may otherwise proceed to dialysis, which can cost $50,000-$100,000 per year. In acute liver failure, liver support devices are more realistically being used as a 'bridge' to liver transplantation rather than to transplant-free survival, according to the Journal of Alimentary Pharmacology and Therapeutics, and a liver transplant can cost in excess of $500,000 according to the United Network for Organ Sharing. This cost does not include the $50,000-$100,000 per year of supportive medications/services required post-transplant.

We think it's reasonable to estimate that OCA could command a "middle ground" price of at least $100,000 annually, well below the $300,000+ range of the "Ultra-orphans" but still a significant premium to a standard course of therapy, especially given the enormous costs of the unmet medical need in treating Urso-failures as they progress to acute liver failure, dialysis, and ultimately a liver transplant or death.

EXHIBIT 14 – Image of NASH



Source: Longo DL et al: Harrison's Principles of Internal Medicine, 18th Edition.

OCA for Nonalcoholic Steatohepatitis (NASH) - $9/share

Key Issues: This is the "home-run" indication for ICPT, in our opinion. With ~8M patients in the US alone with advanced NASH this indication could be the one that drives the most value to shareholders. Partnered with the NIDDK we anticipate phase 2 data for NASH in 2H14.

Background on nonalcoholic steatohepatitis (NASH)

Nonalcoholic steatohepatitis (NASH) is a disease subset of nonalcoholic fatty liver disease (NAFLD). NAFLD occurs when fat is deposited (steatosis) in the liver in individuals that drink little to no alcohol and is the most common chronic liver disease worldwide. In most people, it causes no symptoms, but in some it can cause inflammation and scaring. In approximately 30% of NAFLD patients, this inflammation leads to extensive scaring and cirrhosis with liver function progressively declining and eventually leading to failure

Off-label treatments only
Currently there are no FDA approved drugs for the treatment of NAFLD or NASH. According to ICPT, in 2010 there were approximately $615M in off-label sales of various therapeutics. These drugs typically treat symptoms of the disease and of various co-morbidity diseases such as diabetes, obesity and high cholesterol. The standard of care in most cases is healthy lifestyle changes and exercise to reduce body weight and treatment of any underlying diseases. These treatments however, have not conclusively shown to prevent disease progression.

Patient Population:
It is estimated that there are more than 75 million patients in the US with NAFLD. Approximately 30% of NAFLD patients develop NASH or ~22.5 million Americans with ~8 million of that in advanced stages. NASH is currently the third leading indication for liver transplant in the US. The vast majority (~90%) of patients are female with most being middle-aged, overweight or obese individuals. Other contributing factors are insulin resistance, release of toxic inflammatory protein by fat cells (cytokines), and oxidative stress (deterioration of cells) inside the liver cells.

OCA activation of FXR key in NASH
What makes OCA relevant in NASH is that FXR activation has been shown to play a key role in the regulation of the metabolic pathways disrupted by the disease. FXR is viewed as a potential drug target for treatment of the disease as pre-clinical studies in rodents suggest that FXR activation inhibits hepatic de novo lipogenesis (the enzymatic pathway for converting carbohydrates to fats in the liver), increases insulin sensitivity and protects liver cells against bile acid-induced cytotoxicity.

Phase 2 Clinical Trial
OCA is currently being used in a phase 2b NASH trial called FLINT. In this trial, 280 patients will receive a 25mg single daily dose of OCA vs. placebo for 72-weeks. It is a double-blind trial with primary endpoint based on liver biopsy and is defined as improvement of two or more points in the NAFLD activity score (a system of scoring the histopathological features in the liver) with no worsening of fibrosis. The FLINT trial should have full enrollment by 2012 with final results expected in late 2014 (see Exhibit 15 below). The trial is a collaboration between ICPT and the US National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK) with the NIDDK overseeing the clinical research network and providing the majority of funding for the trial.

EXHIBIT 15

Phase 2b: FLINT - OCA (obeticholic acid) for nonalcoholic steatoheptitis (NASH)	
Aim	Evaluate whether treatment with OCA 25mg for 72 weeks compared to placebo improves the severity of nonalcoholic fatty acid liver disease (NAFLD)
Design	multicenter, random, 2x blind, parallel assignment, OCA vs placebo; Sponsored by the US National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK)
Dosing	OCA (25 mg) vs. placebo, both administered orally daily for 72 weeks
Endpoints	1': Hepatic histological improvement in NAFLD activity score (NAS) - No worsening in fibrosis (confirmed by liver biopsy at month 18); - A decrease in NAFLD Activity Score of at least 2 points
Patients	N=280, parallel - active vs placebo
Results	DSMB interim look June 2012, recommended continuing study Enrollment completed 11/12/12, results expected late 2014

Source: Company Reports & Janney estimates

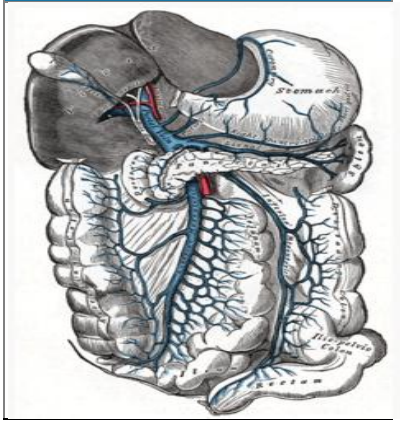
Other indications: Portal Hypertension & Bile Acid Diarrhea - $1/share
Key Issues: Part of the pipeline "technology value" at ICPT, these are Interesting indications for OCA, but earlier stage phase 2a programs. ICPT is focusing resources on their later stage candidates at this point, but these represent interesting early stage pipeline opportunities.

Background in Portal Hypertension
Portal hypertension is the increase in pressure within the portal vein, which is the vein that carries blood from the digestive organs to the liver. It results as the liver becomes cirrhotic and more rigid, thereby offering more and more

resistance to blood inflow from the vein. Thus, many patients with liver cirrhosis go on to develop portal hypertension and it is a common cause of morbidity and mortality at the end stage of all chronic liver diseases.

The main symptoms and complications of portal hypertension are gastrointestinal bleeding which is marked by black, tarry stool, accumulation of fluid in the abdomen and confusion or forgetfulness due to poor liver function. Another early manifestation and complication is the development of esophageal varices (extreme vein dilation) which causes the veins in the lower part of the esophagus to distend and weaken. In this condition, the veins can burst and lead to catastrophic bleeding.

EXHIBIT 16 – Portal Vein & Tributaries



Source: Gray's Anatomy

No approved treatment

Just as with NASH, there are currently no FDA approved treatments for portal hypertension. Treatment focuses on preventing or managing the complications, especially the bleeding of varices. The most common treatment used is beta blockers, which reduce blood pressure. This treatment however, is only effective in 25%-33% of patients and has significant safety issues.

Patient Population

While the total incidence and prevalence of portal hypertension is not known, it has been reported that in the US, 2000 for per 100,000 people have liver cirrhosis and that more than 90% of those individuals develop portal hypertension. As there are many causes for liver cirrhosis, the patient population of portal hypertension is just over 5.5 million in the US alone.

Phase 2a PESTO Trial

OCA is currently being tested in an open label Phase 2a trial, called PESTO, in patients with portal hypertension to evaluate the ability of OCA to reduce hepatic portal venous pressure in patients with end-stage liver disease. The primary endpoint of the trial is to lower the hepatic venous pressure gradient (HVPG) after seven days of treatment by 15% or more; a level at which the risk of adverse clinical outcomes has been shown to be significantly reduced.

ICPT recently reported that the 10mg dose was well tolerated and that in 5 of 8 patients treated for efficacy OCA hit the primary endpoint of lowering HVPG by 15% or more, while a 6th patient dropped 14%. Data from the 25mg group is expected in 1H13 (See Exhibit 16 below). If the PESTO trial supports the further development of OCA for the treatment of portal hypertension, ICPT will initiate a Phase 2b clinical trial in patients with portal hypertension.

EXHIBIT 17

Phase 2a: PESTO - OCA (obeticholic acid) for patients with liver cirrhosis and portal hypertension	
Aim	Efficacy, Tolerability & Safety study of OCA (obeticholic acid) in CF patients with Portal Hypertension and cirrhosis of the liver
Design	Open-label, multi-site, 7-day from baseline, OCA only
Dosing	OCA 10mg (safety & efficacy) or 25mg (safety only) orally for 7 days
Endpoints	1': Improvement of portal pressure as assessed by Hepatic Venous Pressure Gradient (HVPG) after 7 days of treatment (>=15%, or to less than 12 mm Hg) 2': Hepatic haemodynamivs: hepatic blood flow and intrahepatic resistance ; Liver and renal function: GGT, ALT, ALP, albumin, prothrombin time, INR, bilirubin; Pharmacokinteics; Inflammation.
Patients	10mg: N=12, 1st 4 drug safety only, next 8 safety & efficacy; patients with established alcoholic cirrhosis & portal hypertension
Safety	10mg: Well tolerated in all patients, no sig changes in aminotransferases, creatinine, bilirubin, or
Results - 11/8/12	10mg: 5/8 patients in efficacy group lowered HVPG 15% or more, 6th was lowered -14% 25mg: still awaiting data 1H13

Source: Company Reports

Background on Bile Acid Diarrhea

Bile acid diarrhea is a common subtype of inflammatory bowel syndrome with diarrhea (IBS-D) which is marked by chronic watery diarrhea. Fibroblast growth factor 19 (FGF19) levels in these patients are substantially low, resulting in impaired feedback inhibition of bile acid synthesis. The resulting excess bile acids spill into the intestine where they produce diarrhea by stimulating intestinal secretion. FGF19 is synthesized in the small intestine under the direct regulation of FXR.

Phase 2 OBADIAH trial

In July of 2012, enrollment began at the Imperial College of London for an open label Phase 2a trial called OBADIAH. The primary outcome measure of the trail will be to assess the change in FGF19 levels over a two-week period of ten

patients with bile acid diarrhea and in two control groups. As the Imperial College of London is acting as the sponsor of the OBADIAH trial, if positive results support further development of OCA for bile acid diarrhea, then additional funding will need to be secured.

EXHIBIT 18

Phase 2a: OBADIAH - OCA (obeticholic acid) for bile acid diarrhea	
Aim	Safety and to define change over 2 weeks in serum fibroblast growth factor (FGF19)
Design	multi-site, open-label, 2 weeks, OCA only; Sponsored by the Imperial College of London
Dosing	OCA (25 mg) 1x/day orally for 15 days
Endpoints	1': Define change over 2 weeks in serum fibroblast growth factor (FGF19) in 3 patient groups 2': 7a-hydroxy-4-cholesten-3-one 2': Individual and bile acids, tolerability and change in symptoms
Patients	N=30, 1:1 active
Results	Anticipated 1H13

Source: Company Reports & Janney estimates

Additional Pipeline opportunities

INT-767: This is an orally administered FXR and TGR5 agonist that is derived from the primary human bile acid CDCA. TGR5 is another dedicated bile acid receptor like FXR that is a target of interest for the treatment of type 2 diabetes. INT-767 has been shown to be approximately 5x more potent that OCA as an FXR agonist and has consistently shown greater anti-fibrotic and anti-inflammatory effect than OCA in animal models. Currently ICPT plans to advance INT-767 through preclinical studies with a focus on developing it as a novel treatment for chronic kidney diseases.

INT-777: This is another orally administered TGR5 agonist that showed potential to selectively target TGR5 in the intestine in vitro, with resulting insulin sensitizing effects. In animal models, treatment with INT-777 induced GLP-1 secretion, a glucagon like peptide-1, with resulting insulin sensitivity and normalizing of glycemic control increased basal energy expenditure and prevention of weight gain and a reduction in the blood lipid levels together with liver steatosis and fibrosis. ICPT believes that there results could support further development in the treatment of type 2 diabetes and associated metabolic disorders.

Management

Mark Pruzanski, M.D., President and CEO. Dr. Pruzanski is a co-founder of the company and has served as the chief executive officer and president and has been a member of the board of directors, since company inception in 2002. HE has over 15 years of experience in life sciences company management, venture capital and strategic consulting. Dr. Pruzanski was previously a venture partner at Apple Tree Partners, an early stage life sciences venture capital firm he co-founded in 1999. Prior to that, he 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 in Bologna, Italy and Washington, D.C., and a bachelor's degree from McGill University in Montreal, Quebec.

David Shapiro, M.D. Chief Medical Officer and EVP of Development. Dr. Shapiro has served as the chief medical officer and executive vice president of development since 2008. He has over 25 years of clinical development experience in the pharmaceutical industry. Dr. Shapiro founded a consulting company, Integrated Quality Resources, which 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, Inc., 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 & 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.

Barbara Duncan, Chief Financial Officer – Ms. Duncan has served as chief financial officer and secretary since May 2009 and as our treasurer since 2010. She has over 14 years of 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, Inc. in 2010. Prior to joining DOV, Ms. Duncan served as a vice president of Lehman Brothers Inc. in its corporate finance division from August 1998 to August 2001. From September 1994 to August 1998, Ms. Duncan was an associate and director at SBC Warburg Dillon Read. 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.

Luciano Adorini, M.D. Chief Scientific Officer – Dr. Adorini has served as chief scientific officer since 2008. Dr. Adorini has over 20 years of industry experience. From January 2002 through December 2007, Dr. Adorini served as chief scientific officer at BioXell S.p.A., where he was responsible for advancing a broad pipeline of products in multiple disease indications. From January 1993 to December 2001, he served as associate director of Roche Milano Richerche, where he contributed to the development of several drugs. Earlier in his career, Dr. Adorini was research director of a unit at the Preclinical Research Center, Sandoz Pharma, Ltd., in Basel, Switzerland. Dr. Adorini has authored over 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. 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.

RISKS TO FAIR VALUE ESTIMATE:

Exogenous events could impact our outlook. We believe that pharmaceutical companies have the least control over competitive, political, and regulatory risks. Although we have incorporated competitive assumptions into our forecasts, there may be other risks beyond the scope of our analysis. Changes in the drug reimbursement system, as well as any political or regulatory amendments, may significantly influence the earnings power of these companies.

Actual clinical results and the FDA's conclusions may deviate from expectations. Many of our assumptions are based on a review of incomplete clinical trial data available in the public domain. Often our conclusions are drawn from early-stage data, which may not be reflected by pivotal studies. Furthermore, the FDA's conclusions may not coincide with our own, materially changing our revenue and earnings assumptions.

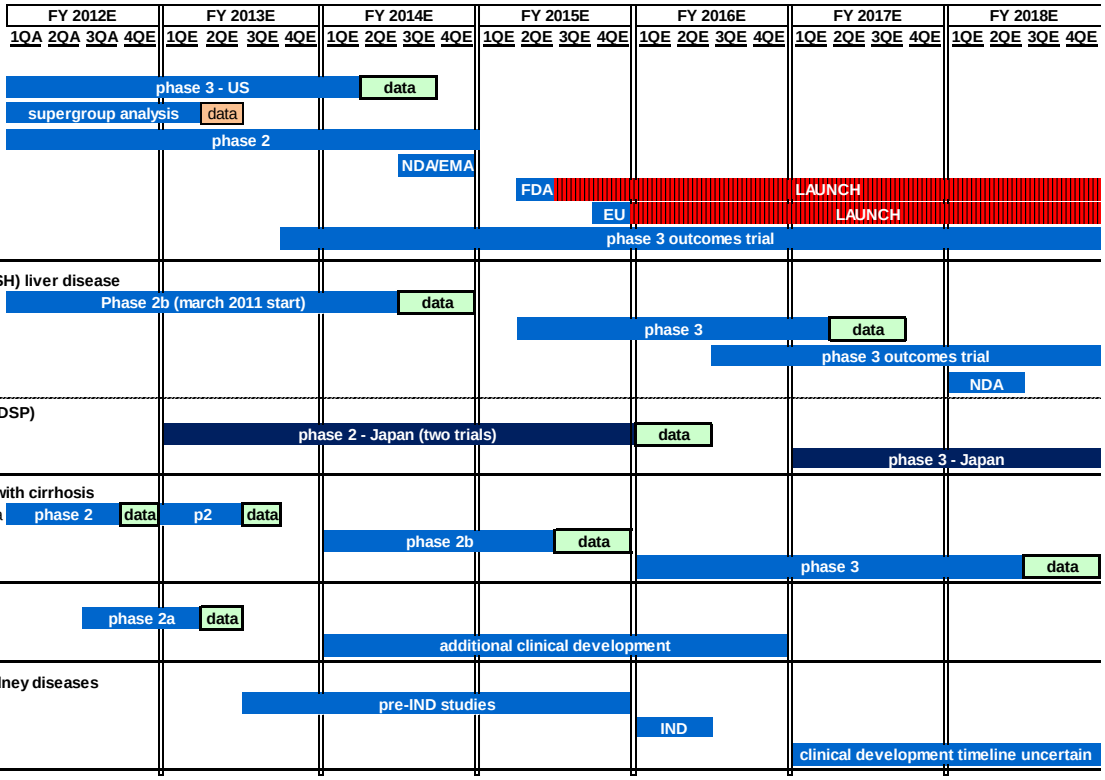
Compliance issues, product recalls, and other mandates by regulatory authorities could materially change our expectations. Regulatory compliance issues, ranging from accounting irregularities to defective manufacturing practices, could materially change our assumptions and earnings outlook. Unanticipated product recalls and labeling changes could also have adverse consequences on our earnings assumptions.

Legal risks could lead to additional liabilities and revenue loss. In addition to the expenses incurred by patent challenges, product liability and other legal suits could occur and lead to additional liabilities and revenue loss, which could substantially change our financial assumptions.

EXHIBIT 19: Potential Clinical Trial Timelines

Intercept Pharmaceuticals

Potential clinical trial timeline estimates



Source: Company reports and Janney estimates

Specialty Pharmaceuticals
Jim Molloy (617) 371-1528 jmolloy@janney.com

Intercept Pharmaceuticals
Quarterly income statement

($000's except per share)	2012E 1QA	2QA	3QA	4QE	2012E Year	2013E 1QE	2QE	3QE	4QE	2013E Year
Revenues										
License fees	$759	$759	$523	$525	$2,566	$400	$400	$400	$400	$1,600
Total Revenues	**$759**	**$759**	**$523**	**$525**	**$2,566**	**$400**	**$400**	**$400**	**$400**	**$1,600**
Expenses										
Cost of Goods Sold	0	0	0	0	**0**	0	0	0	0	**0**
Gross Margin	**759**	**759**	**523**	**525**	**2,566**	**400**	**400**	**400**	**400**	**1,600**
Research and Development	3,060	5,018	3,318	4,250	15,646	5,250	6,250	6,500	6,500	24,500
SG&A	1,059	944	991	3,000	5,994	3,250	3,500	3,500	4,000	14,250
Total Operating Expenses	**4,119**	**5,962**	**4,309**	**7,250**	**21,640**	**8,500**	**9,750**	**10,000**	**10,500**	**38,750**
Income (loss) from Ops	**(3,360)**	**(5,203)**	**(3,786)**	**(6,725)**	**(19,074)**	**(8,100)**	**(9,350)**	**(9,600)**	**(10,100)**	**(37,150)**
Warrant Revaluation income	678	301	(1,418)		**(439)**					**0**
FOREX loss on liquidation		(198)								
Interest & dividend income	10	7	13	10	**41**	10	10	10	10	**40**
Interest expense	(7)	0	3	4	**(0)**					**0**
QTP grant					**0**					**0**
Pretax Income (Loss)	**(2,680)**	**(5,092)**	**(5,187)**	**(6,711)**	**(19,670)**	**(8,090)**	**(9,340)**	**(9,590)**	**(10,090)**	**(37,110)**
Dividend of pref stock, not declared	(750)	(750)	(1,000)		(2,500)					0
Net income/(loss)	**(3,430)**	**(5,842)**	**(6,187)**	**(6,711)**	**(22,170)**	**(8,090)**	**(9,340)**	**(9,590)**	**(10,090)**	**(37,110)**
EPS as reported	**($1.03)**	**($1.75)**	**($1.86)**	**($0.41)**	**($5.05)**	**($0.48)**	**($0.48)**	**($0.49)**	**($0.50)**	**($1.95)**
Shares out (000)	3,330	3,330	3,330	16,484	16,484	16,734	19,484	19,734	19,984	18,984
Fully diluted shares (000)	14,326	14,326	14,326	19,238	19,238	19,484	22,234	22,484	22,734	21,734

Source: Company reports and Janney estimates

Specialty Pharmaceuticals
Jim Molloy (617) 371-1528 jmolloy@janney.com

Intercept Pharmaceuticals
Annual income statement

($000's except per share)	2012E	2013E	2014E	2015E	2016E	2017E	2018E
Revenues							
OCA sales	-	-	-	$27,799	$150,913	$396,849	$609,266
OCA royalties (EU & Japan)	-	-	-	-	6,849	35,454	60,236
License fees	$2,566	$1,600	$1,600	1,600	1,800	2,000	2,000
Total Revenues	**$2,566**	**$1,600**	**$1,600**	**$29,399**	**$159,562**	**$434,303**	**$671,502**
Expenses							
Cost of Goods Sold	-	-	-	6,745	22,637	59,527	73,112
Gross Margin	**2,566**	**1,600**	**1,600**	**22,654**	**136,925**	**374,776**	**598,390**
Research and Development	15,646	24,500	28,000	30,000	34,000	45,000	65,000
SG&A	5,994	14,250	18,250	25,000	41,500	93,750	132,500
Total Operating Expenses	**21,640**	**38,750**	**46,250**	**55,000**	**75,500**	**138,750**	**197,500**
Income (loss) from Ops	**(19,074)**	**(37,150)**	**(44,650)**	**(32,346)**	**61,425**	**236,026**	**400,890**
Warrant Revaluation income	(439)	-	-	-	-	-	-
FOREX loss on liquidation	-	-	-	-	-	-	-
Interest & dividend income	41	40	40	60	75	100	100
Interest expense	(0)	-	-	-	-	-	-
Pretax Income (Loss)	**(19,670)**	**(37,110)**	**(44,610)**	**(32,286)**	**61,500**	**236,126**	**400,990**
Taxes	-	-	-	-	-	47,225	136,337
Dividend of pref stock, not declared	(2,500)	-	-	-	-	-	-
Net income/(loss)	**(22,170)**	**(37,110)**	**(44,610)**	**(32,286)**	**61,500**	**188,901**	**264,654**
EPS as reported	**($5.05)**	**($1.95)**	**($2.16)**	**($1.50)**	**$2.50**	**$6.50**	**$8.75**
Shares out (000)	16,484	18,984	20,609	21,734	22,884	24,059	25,259
Fully diluted shares (000)	19,238	21,734	23,609	25,234	24,584	29,059	30,259
Margin & expense analysis							
COGS as % prod sales	NA	NA	NA	24%	14%	14%	11%
R&D	NA	NA	NA	102%	21%	10%	10%
SG&A	NA	NA	NA	85%	26%	22%	20%
Op. margin cont. ops	NA	NA	NA	-110%	38%	54%	60%
Taxes	NA	NA	NA	0%	0%	20%	34%
Net margin	NA	NA	NA	-110%	39%	43%	39%
Year-over-year change							
Net revenue	NA	NA	NA		443%	172%	55%
R&D	37%	57%	14%	7%	13%	32%	44%
SG&A	42%	138%	28%	37%	66%	126%	41%
Operating income	NA	NA	NA	NA	NA	284%	70%
Net income	NA	NA	NA	NA	NA	207%	40%

Source: Company reports and Janney estimates

Specialty Pharmaceuticals
Jim Molloy (617) 371-1528 jmolloy@janney.com

Intercept Pharmaceuticals
Balance sheet

($000's except per share)	1Q12A	2Q12A	3Q12A	2012E	2013E	2014E	2015E	2016E	2017E	2018E
Current Assets										
Cash and cash equivs	13,744	9,947	35,971	78,455	131,395	88,610	57,924	121,593	312,074	575,903
Certificates of deposit	204	79	78	78						
Prepaid expenses + Other	1,228	1,682	2,106							
Total Current Assets	**15,177**	**11,708**	**38,155**	**78,533**	**135,145**	**93,360**	**64,174**	**128,843**	**320,574**	**588,403**
Fixed assets (net)	257	176	157	175	175	175	175	175	175	175
Security deposits	255	261	258	250	250	275	275	275	300	300
Total Assets	**15,689**	**12,145**	**38,570**	**78,958**	**135,570**	**93,810**	**64,624**	**129,293**	**321,049**	**588,878**
Current Liabilites										
Total Current Liabilites	**3,609**	**5,604**	**6,047**	**7,250**	**7,750**	**8,125**	**8,750**	**9,220**	**9,300**	**9,500**
LT deferred revenue	11,757	11,351	10,946	11,000	11,000	11,000	11,000	11,000	11,000	11,000
LT warrant liability	5,158	4,569	5,940	6,000	6,000	6,000	6,000	6,000	6,000	6,000
Total Liabilities	**20,524**	**21,525**	**22,932**	**24,250**	**24,750**	**25,125**	**25,750**	**26,220**	**26,300**	**26,500**
Shareholders' Equity										
Series A pref stock	14	14	14	14						
Series B pref stock	14	14	14	14						
Series C pref stock			15	15						
Common stock	19	3	3	3						
Additional paid in capital	72,510	72,895	103,084	164,325	257,593	260,068	262,543	265,243	268,018	270,993
Accumulated other loss	(172)									
Accumulated deficit	(77,220)	(82,306)	(87,493)	(109,663)	(146,773)	(191,383)	(223,669)	(162,169)	26,731	291,385
Total SE (deficit)	**(4,835)**	**(9,380)**	**15,637**	**54,708**	**110,820**	**68,685**	**38,874**	**103,073**	**294,749**	**562,378**
Total liabilities & SE	**15,689**	**12,145**	**38,570**	**78,958**	**135,570**	**93,810**	**64,624**	**129,293**	**321,049**	**588,878**

Source: Company reports and Janney estimates

Intercept Pharmaceuticals
Statement of cash flows

($000's except per share)	1Q12A	2Q12A	3Q12A	2012E	2013E	2014E	2015E	2016E	2017E	2018E
Operating Cash Flow										
Net Income/Loss	(2,680)	(7,766)	(12,953)	(22,170)	(37,110)	(44,610)	(32,286)	61,500	188,901	264,654
Adjustments:										
Deprec & Amort	74	154	178	200	225	225	250	250	300	300
Stock based comp exp	392	761	1,252	1,850	2,000	2,250	2,250	2,500	2,500	2,750
Changes in Assets & Liabilites	(1,002)	(130)	(565)	2,165	(1,000)	(625)	(875)	(530)	(1,170)	(3,800)
Prepaid expenses and other assets	(232)	(686)	(1,110)							
Accounts payable	(11)	2,073	2,586							
Deferred revenue	(759)	(1,518)	(2,041)							
Cash from operations	**(3,893)**	**(7,768)**	**(11,457)**	**(17,956)**	**(35,885)**	**(42,760)**	**(30,661)**	**63,720**	**190,531**	**263,904**
Investing Activities										
Redemptions of CD's	(4)	116	120	125	150	150	150	175	175	175
Purchase of fixed assets	(20)	(19)	(24)	(30)	(50)	(75)	(75)	(125)	(125)	(150)
Cash from investing	**(24)**	**97**	**96**	**95**	**100**	**75**	**75**	**50**	**50**	**25**
Financing Activities										
Payments on cap leases	(59)	(82)	(82)	(82)	(100)	(100)	(100)	(100)	(100)	(100)
Proceeds from stock issuance			30,000	86,250	88,825					
Stock issuance costs			(286)	(7,550)						
Cash from financing	**(59)**	**(82)**	**29,632**	**78,618**	**88,725**	**(100)**	**(100)**	**(100)**	**(100)**	**(100)**
Forex impact	13		(7)	(10)						
Change in cash	**(3,963)**	**(7,753)**	**18,264**	**60,747**	**52,940**	**(42,785)**	**(30,686)**	**63,670**	**190,481**	**263,829**
Cash, start of period	17,707	17,707	17,707	17,707	78,455	131,395	88,610	57,924	121,593	312,074
Cash, end of period	**13,744**	**9,954**	**35,972**	**78,455**	**131,395**	**88,610**	**57,924**	**121,593**	**312,074**	**575,903**

Source: Company reports and Janney estimates

IMPORTANT DISCLOSURES

Research Analyst Certification

I, Jim Molloy, the Primarily Responsible Analyst for this research report, hereby certify that all of the views expressed in this research report accurately reflect my personal views about any and all of the subject securities or issuers. No part of my compensation was, is, or will be, directly or indirectly, related to the specific recommendations or views I expressed in this research report.

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The research analyst is compensated based on, in part, Janney Montgomery Scott's profitability, which includes its investment banking revenues.

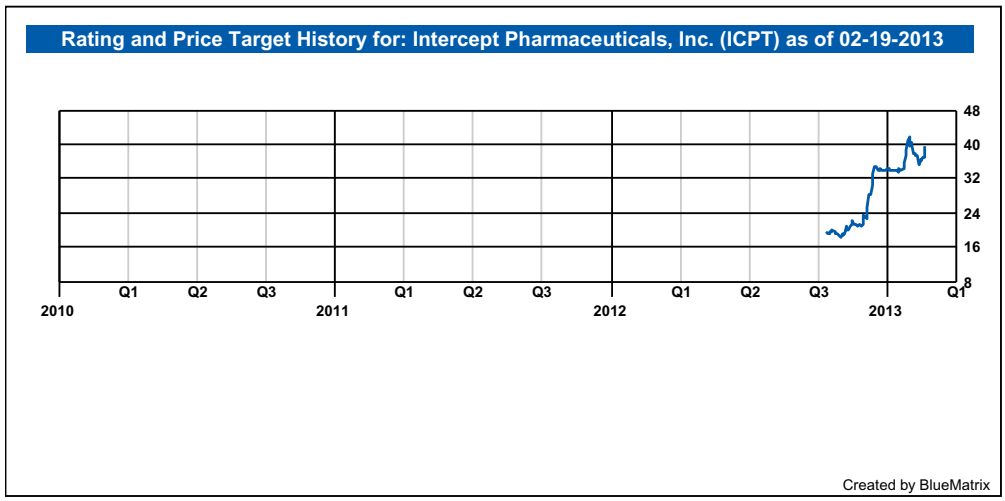
Definition of Ratings

BUY: Janney expects that the subject company will appreciate in value. Additionally, we expect that the subject company will outperform comparable companies within its sector.

NEUTRAL: Janney believes that the subject company is fairly valued and will perform in line with comparable companies within its sector. Investors may add to current positions on short-term weakness and sell on strength as the valuations or fundamentals become more or less attractive.

SELL: Janney expects that the subject company will likely decline in value and will underperform comparable companies within its sector.

Price Charts



Janney Montgomery Scott Ratings Distribution as of 12/31/12

			IB Serv./Past 12 Mos.	
Rating	**Count**	**Percent**	**Count**	**Percent**
BUY [B]	207	53.35	32	15.46
NEUTRAL [N]	177	45.61	15	8.47
SELL [S]	4	1.03	0	0.00

***Percentages of each rating category where Janney has performed Investment Banking services over the past 12 months.**

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