Company Presentation
November 2013
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ICPT Investment Highlights

- Intercept (ICPT) was founded in 2002 around a bile acid chemistry drug discovery platform
  - Co-founder Roberto Pellicciari, PhD: rational design of selective bile acid derived NCEs

- OCA in Phase 3 for orphan indication primary biliary cirrhosis with data expected in Q2 2014
  - OCA (obeticholic acid) is a first-in-class farnesoid X receptor (FXR) agonist
  - Two Phase 2 randomized trials met all primary (p<0.0001) and secondary endpoints
  - Addressable population anticipated to be approximately 30,000 patients in developed markets
  - Additional indications focused primarily on chronic liver diseases
  - Patent terms projected through 2028

- Pipeline of follow-on bile acid derived NCEs

- Experienced management team and strong financial position
  - Raised approximately $245 million in equity to date with $157 million of cash as of 9/30/2013
  - Operations funded into early 2016 (through anticipated US and European approvals)
Management Team

- Experienced biotech CEO, life sciences venture capitalist
- **Inventor on several ICPT patents; author of numerous publications**
- Founder of venture capital firm **Apple Tree Partners**

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**Mark Pruzanski, MD**
Founder, President & Chief Executive Officer

- Former EVP Development & CMO at **Idun Pharmaceuticals**
- Former President of **Scripps** Medical Research Center
- Former Director, Clinical Research at **Merck**

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**David Shapiro, MD FRCP**
Chief Medical Officer

- Former CSO at **BioXell**
- Former Associate Director of **Roche** Milan Research
- Former President, **Italian Immunology Society**

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**Luciano Adorini, MD**
Chief Scientific Officer

- Former CEO/CFO of **DOV Pharmaceutical**
- Investment banking experience with **Lehman** and **Dillon Read**

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**Barbara Duncan, MBA**
Chief Financial Officer

- Former CCO at **Inspiration**
- Former SVP & GM at **Genzyme**
- Commercial experience with orphan & specialty products

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**Dan Regan**
Chief Commercial Officer
Current clinical focus primarily on chronic liver diseases with high unmet medical needs

<table>
<thead>
<tr>
<th>Product / Indication</th>
<th>Preclinical</th>
<th>Phase 1</th>
<th>Phase 2</th>
<th>Phase 3</th>
<th>Our Rights</th>
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<tbody>
<tr>
<td>OCA (FXR Agonist)</td>
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<td>Worldwide</td>
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<tr>
<td>Primary Biliary Cirrhosis (PBC)</td>
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<td>Worldwide excluding certain Asian countries incl. Japan/China (licensed to DSP)</td>
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<tr>
<td>Portal Hypertension</td>
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<tr>
<td>Nonalcoholic Steatohepatitis (NASH)</td>
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<td>Worldwide</td>
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<tr>
<td>Bile Acid Diarrhea</td>
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<td>Worldwide</td>
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<tr>
<td>INT-767 (Dual FXR / TGR5 Agonist)</td>
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<td>Worldwide</td>
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<tr>
<td>Fibrosis</td>
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<td>Worldwide</td>
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<tr>
<td>INT-777 (TGR5 Agonist)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Worldwide</td>
</tr>
<tr>
<td>Type 2 Diabetes</td>
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</tbody>
</table>

Additional OCA indications include primary sclerosing cholangitis, biliary atresia
Why Bile Acids Are Important As a Basis for Novel Therapies

- Bile acids are synthesized in the liver from cholesterol

- Facilitate absorption of dietary lipids and nutrients based on their natural detergent properties
  - Released by gallbladder into gut during digestion
  - Reabsorbed & conserved (enterohepatic recirculation)

- Discovery of dedicated bile acid receptors (e.g., FXR): act as hormones regulating multiple biological pathways
  - Key regulators of liver, intestinal & kidney function
  - Modulate metabolic, inflammatory & immune pathways

- Proprietary capability to rationally modify bile acids used to efficiently generate our pipeline of potent NCEs
Obeticholic Acid (OCA), a First-in-Class FXR Agonist for Chronic Liver Disease

- Overview of PBC
- OCA Mechanism of Action
- PBC Development Program Overview
- Additional Clinical Indications for OCA
| Chronic Orphan Disease | - PBC is an autoimmune, chronic liver disease  
                             - Orphan drug designation in US and EU |
|-------------------------|---------------------------------------------------------------------------------|
| Significant Unmet Need  | - Up to 50% of PBC patients fail to respond adequately to SOC ursodiol therapy  
                             - Limited options for end-stage PBC patients: long liver transplant waiting list |
| Favorable Market Dynamics | - Significant costs of treating complications of liver failure and liver transplant  
                                  - Specialty care market with limited number of treating physicians  
                                  - Chronic disease requiring long term treatment |
| OCA Product Profile     | - Efficacy demonstrated in two Phase 2 trials and well tolerated for up to 3 years  
                                  - Ease of use with low single daily oral dose of 10 mg |
| Strong IP Position      | - Composition of matter patent expires in 2022 with up to 5 year term extension  
                                  - Additional patents issued and pending, anticipated to be in force through 2028 |
PBC Disease Profile: Pathology, Presentation & Management

- Most common cholestatic liver disease with progressive destruction of bile ducts
  - Primarily female disease (~90% of patients): ~1 in 1,000 women over 40

- Typical initial presentation is either asymptomatic or with complaint of itching (pruritus) and fatigue, two signature symptoms of PBC
  - Median survival in untreated patients of 7.5 years (symptomatic) to 16 years (asymptomatic)

- Diagnosis based mainly on non-invasive serum liver markers
  - Elevated alkaline phosphatase (ALP) + anti-mitochondrial antibody (AMA) titer
  - Liver biopsy not required and becoming increasingly rare

- Ursodiol is the only approved therapy
  - Several other drugs tested in trials but none found to be both safe and effective
Our PBC Market Opportunity

**Size Of Initial Market**

- ~300,000 Estimated patients with PBC in developed countries
- ~60,000 estimated diagnosed and on ursodiol therapy
- ~30,000 patients believed eligible for OCA treatment
- Up to 50% of patients have an inadequate response to ursodiol

**Targeted Commercial Investment**

- KOL driven specialty market
- Well-organized physician and patient community
- Plan to address with relatively small specialty sales force

**US and EU**

**Asia: DSP Agreement**

- DSP exclusive license of OCA for PBC and NASH in Japan and China
- $15MM up front with ~$300MM milestones
- Tiered sales royalties in the 10s – 20s %
- Option to expand to other Asian territories and indications

**Estimated Patients**

- ~300,000 patients with PBC in developed countries
- ~60,000 patients diagnosed and on ursodiol therapy
- ~30,000 patients believed eligible for OCA treatment
- Up to 50% of patients have an inadequate response to ursodiol
OCA: Potent First-in-Class FXR Agonist and Bile Acid Analog

- **OCA (6E-CDCA)**
  - obeticholic acid
  - Close analog to bile acid CDCA but 100x more potent on FXR
  - Metabolic stability
  - First-in-class with novel mechanism of action

- **CDCA**
  - chenodeoxycholic acid
  - Endogenous FXR agonist

- **UDCA**
  - ursodeoxycholic acid
  - Epimer of CDCA
  - Only product approved for PBC
  - Displaces more detergent bile acids in pool

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<table>
<thead>
<tr>
<th>FXR EC&lt;sub&gt;50&lt;/sub&gt;</th>
<th>OCA (0.09 μM)</th>
<th>CDCA (8.6 μM)</th>
<th>UDCA (No activity)</th>
</tr>
</thead>
<tbody>
<tr>
<td>~100x increased potency</td>
<td></td>
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</tr>
</tbody>
</table>

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- OCA
  - Close analog to bile acid CDCA but 100x more potent on FXR
  - Metabolic stability
  - First-in-class with novel mechanism of action

- CDCA
  - Endogenous FXR agonist

- UDCA (Ursodiol)
  - Only product approved for PBC
  - Displaces more detergent bile acids in pool
FXR: Amplifies the Regenerative Capacity of the Liver

FXR is a nuclear receptor expressed in the liver

Liver Fibrosis
Reverses fibrosis and stimulates repair

Portal Hypertension
Reduces inflammation and portal pressure

Glucose Metabolism
Improves insulin sensitivity and glucose uptake

Liver Steatosis
Reduces lipid synthesis and toxicity

Bile Homeostasis
Regulates bile levels and flow rate

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Regulates bile levels and flow rate

FXR
Bile uptake
Bile synthesis

Liver
Cholesterol
Bile acids
Reabsorption
Intestine
FXR
Bile secretion
Bile flow

FXR
Inflammation
Fibrogenesis
Fibrolysis
OCA Reverses Liver Cirrhosis in Animal Disease Models

Data from Scott Friedman lab (Mt. Sinai) with representative rat liver specimen photos and histology from control and treated animals
# OCA Clinical Development in PBC

<table>
<thead>
<tr>
<th>Trial</th>
<th>Description</th>
<th>Duration</th>
<th>Dose</th>
<th>Status</th>
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<tbody>
<tr>
<td>202</td>
<td>Combination therapy in non-responders on ursodiol</td>
<td>12 weeks</td>
<td>10mg, 25mg or 50mg</td>
<td>Completed: met primary and secondary endpoints</td>
</tr>
<tr>
<td>201</td>
<td>Monotherapy in treatment naïve or intolerant patients</td>
<td>12 weeks</td>
<td>10mg or 50mg</td>
<td>Completed: met primary and secondary endpoints</td>
</tr>
<tr>
<td>POISE</td>
<td>Combination therapy in non-responders on ursodiol</td>
<td>1 year</td>
<td>5mg or 10mg</td>
<td>Enrollment completed</td>
</tr>
</tbody>
</table>

All trials randomized, double-blind, placebo-controlled with long-term safety extension (LTSE) phases
Phase 2 Trial in PBC: Combination Therapy (202 Trial)

- Multi-center, double-blind, randomized, placebo-controlled trial in combination with ursodiol in PBC patients with persistently elevated ALP
- 12 week treatment duration with 2 week follow-up
- Enrollment: 165 patients
- OCA 10mg, 25mg, 50mg and placebo once daily
- Primary endpoint: statistically significant reduction in ALP
- Secondary analyses: other liver function tests, including gamma glutamyl transferase (GGT), alanine aminotransferase (ALT), aspartate aminotransferase (AST), and bilirubin
Primary Endpoint: ALP Δ% (Combination Therapy)

OCA treatment for 12 weeks led to statistically significant ALP reductions of >20% at all doses tested vs. placebo.

- Placebo: n=37
- 10 mg: n=38
- 25 mg: n=47
- 50 mg: n=39

Mean values + SEM

≥10% ALP reduction
- Clinically significant
- Correlates with reduced risk of liver failure

Modified intention to treat (ITT) analysis with last ALP value carried forward
Completed Patients: ALP Δ (Combination Therapy)

Rapid therapeutic response after initiating OCA therapy with continuing improvement through 12 week treatment period

Day 85 EOS
p<0.0001 all doses
Other Liver Function Markers (GGT, ALT, Bilirubin, AST)

**GGT**
- Placebo (N=37)
- 10 mg (N=32)
- 25 mg (N=42)
- 50 mg (N=25)

Day 85-EOS
- p<0.0001 all doses

**ALT**
- Placebo (N=37)
- 10 mg (N=32)
- 25 mg (N=42)
- 50 mg (N=25)

Day 85-EOS
- p<0.0001 10 & 25mg
- p<0.005 50mg

**Bilirubin**

Day 85-EOS
- p<0.005 25 & 50mg

**AST**

Day 85-EOS
- p<0.005 all doses

Intercept Pharmaceuticals
Patients on long term OCA therapy have a durable therapeutic response.
Adverse Events (Combination Therapy)

OCA is well tolerated with pruritus (itching) the only side effect of note

- Pruritus is a signature symptom in PBC patients: managed with cholestyramine therapy

<table>
<thead>
<tr>
<th>Adverse Event</th>
<th>Placebo (%)</th>
<th>10mg (%)</th>
<th>25mg (%)</th>
<th>50mg (%)</th>
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<tr>
<td>Pruritus</td>
<td>50</td>
<td>47</td>
<td>85</td>
<td>80</td>
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<tr>
<td>Headache</td>
<td>11</td>
<td>8</td>
<td>10</td>
<td>17</td>
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<tr>
<td>Fatigue</td>
<td>13</td>
<td>18</td>
<td>6</td>
<td>12</td>
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<tr>
<td>Constipation</td>
<td>8</td>
<td>8</td>
<td>8</td>
<td>7</td>
</tr>
<tr>
<td>Diarrhea</td>
<td>8</td>
<td>8</td>
<td>8</td>
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</tr>
<tr>
<td>Nausea</td>
<td>3</td>
<td>11</td>
<td>6</td>
<td>10</td>
</tr>
<tr>
<td>Abdo Distension</td>
<td>3</td>
<td>5</td>
<td>0</td>
<td>10</td>
</tr>
<tr>
<td>Dry Eye</td>
<td>3</td>
<td>0</td>
<td>6</td>
<td>5</td>
</tr>
<tr>
<td>Oropharyngeal Pain</td>
<td>3</td>
<td>5</td>
<td>8</td>
<td>0</td>
</tr>
<tr>
<td>Pain, extremities</td>
<td>0</td>
<td>0</td>
<td>2</td>
<td>10</td>
</tr>
<tr>
<td>Vomiting</td>
<td>0</td>
<td>0</td>
<td>6</td>
<td>0</td>
</tr>
<tr>
<td>Pyrexia</td>
<td>0</td>
<td>8</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>UTI</td>
<td>8</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>
Phase 2 Trial in PBC: Monotherapy (201 Trial)

- Multi-center, double-blind, randomized, placebo-controlled study in treatment naïve or ursodiol intolerant PBC patients
- 12 week treatment duration with 2 week follow-up
- Enrollment: 59 patients
- OCA 10mg, 50mg and placebo once daily
- Primary endpoint: statistically significant reduction in ALP
- Secondary analyses: GGT, ALT, AST, bilirubin
Primary Endpoint: ALP Δ% (Monotherapy)

OCA monotherapy for 12 weeks led to statistically significant ALP reductions of ≥38% at all doses tested vs. placebo.

Mean % Change in ALP

Placebo
n=23

10 mg
n=20

50 mg
n=16

-45%
-38%

p<0.0001

Mean values + SEM
Comparison of Phase 2 Trial Results: ALP Δ

**Combination Trial with Ursodiol**
- Placebo (n=37)
- 10 mg (n=38)
- 25 mg (n=47)
- 50 mg (n=39)

**Monotherapy Trial**
- Placebo (n=23)
- 10 mg (n=20)
- 50 mg (n=16)

All drug vs placebo comparisons: p<0.0025
**POISE**: Phase 3 trial of OCA add-on therapy in PBC patients with an inadequate response to ursodiol or monotherapy in patients unable to tolerate ursodiol

- Status: enrollment completed 3 months ahead of schedule with 217 patients (59 centers, 13 countries)
- Surpassed target enrollment by 20% with resulting increased power from 90% to 95%
- Results from POISE expected to be available in Q2 2014
- Primary Endpoint: ALP < 1.67x ULN (with ≥ 15% reduction) and normal bilirubin

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**Double Blind Phase (12 mos)**

- Entry Criteria:
  - ALP ≥ 1.67x ULN
  - and/or bilirubin > ULN but < 2x ULN

- N = 217 (~72/group)

<table>
<thead>
<tr>
<th>Time</th>
<th>Placebo</th>
<th>OCA 5 mg</th>
<th>OCA 10 mg</th>
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<tbody>
<tr>
<td>0</td>
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<td>M9</td>
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<tr>
<td>M12</td>
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**LTSE (5 years)**

- Long-Term Safety Extension
  - All patients receive OCA 5mg → 25mg, as needed
POISE Primary Endpoint: Correlation with Clinical Benefit

PBC patients achieving an ALP <1.67x ULN with normal bilirubin after one year of ursodiol therapy have a significantly lower risk of progressing to liver failure

- Supergroup analysis ongoing: 15 centers with ~4,000 patients’ outcomes data
- After 1 year of UDCA, 59% (n=576/981) had inadequate response*
- Non-responders: 2.4x increased risk of liver transplant or death
- Increased hazard ratio in younger cohorts:
  - <65yrs: 3.3x
  - <60yrs: 3.6x (p=1x10E-7)

*Response: ALP <1.67xULN (with ≥15% reduction from baseline) & normal bilirubin, among patients with initial ALP>1.67x ULN and/or abnormal bilirubin (n=981 at t=0)
UK PBC Database: Additional Support for Endpoint

- Clinical outcomes data for 2,353 patients in UK
  - ~25% of total UK PBC patients
  - Collected from every hospital in UK

- Demonstrates statistically significant correlation with clinical outcomes:
  - ALP alone (Barcelona & Toronto)
  - ALP + bilirubin (Paris I & II)

- Greatest differentiation seen with ALP + bilirubin

PBC KOLs Aligned On Clinical Validity Of POISE Endpoint

Hepatology (July 2010)

American Association for the Study of Liver Diseases
Endpoints Conference: Design and Endpoints for Clinical Trials in Primary Biliary Cirrhosis

Marina G. Silveira,1 Elizabeth M. Brunt,2 Jenny Heathcote,3 Gregory J. Gores,1
Keith D. Lindor,1 and Marlyn J. Mayo4

“The inclusion of biochemical markers is a satisfactory primary endpoint for therapeutic trials.”

Liver International (May 2012)

Optimizing biochemical markers as endpoints for clinical trials in Primary biliary cirrhosis

Njideka Momah1, Marina G. Silveira2, Roberta Jorgenson2, Emmanouil Sinakos2, and Keith Lindor2

“We found that patients with ALP ≤ 1.67x ULN and bilirubin ≤ 1 mg/dl at 12 months of UDCA therapy had the lowest occurrence of endpoints… These values seem to be optimal for defining treatment.”
Phase 2 Results Analyzed on the POISE Primary Endpoint

~40% of Phase 2 patients on 10mg of OCA met the POISE trial primary endpoint

% Patients with ALP <1.67 x ULN (with >15% ALP Reduction) and Normal Bilirubin at End of Study

Combination Trial (202)

- Placebo: 10% (n=32)
- OCA: 40% (n=30)
  \[ p=0.007 \]

Monotherapy Trial (201)

- Placebo: 5% (n=21)
- OCA: 34% (n=16)
  \[ p=0.012 \]
PBC Regulatory Path to Approval

**EU Regulatory Status**
- POISE trial designed in accordance with EMA scientific advice concerning requirements for approval of OCA for PBC

**US Regulatory Status**
- Company intends to file with FDA for accelerated approval (under Subpart H), conditional on conducting an additional Phase 3 confirmatory clinical outcomes trial for full approval
  - Plan to agree on confirmatory trial design and initiate trial in 1H 2014
Follow On Indications
Portal hypertension results from increasing backflow pressure in the portal vein as the liver becomes cirrhotic and more rigid.

- Common cause of morbidity and mortality at end stage of all chronic liver diseases

As portal pressure rises, veins at base of esophagus become distended (varices) and can burst, resulting in catastrophic bleeding.

- ~40% of patients already have varices when diagnosed with cirrhosis
- ~1/3 of patients have a bleeding episode within 2 years with ~20% mortality risk

No therapies currently approved but beta blockers used as SOC

- Effective in only ~1/3 of patients but significant safety issues: e.g., lower systemic blood pressure

OCA animal data demonstrated acute reversal of portal hypertension via localized vasodilatory mechanism of action with no change in systemic blood pressure.
PESTO: Phase 2a trial in ~25 patients

- Primary endpoint after 7 days of therapy: lower HVPG by \( \geq 15\% \) and/or to <12 mmHg

Initial results reported at 2012 AASLD for 10mg dose group

- 5 of 8 patients met the endpoint(s); 6\(^{th}\) patient had >14\% fall in HVPG
- OCA well tolerated in all 12 patients

Additional 10mg & 25mg dose groups being enrolled

- Trial completion expected in 4Q 2013
OCA for Nonalcoholic Steatohepatitis: Phase 2b FLINT Trial

- In NASH, liver fat accumulation causes inflammation & fibrosis leading to cirrhosis
  - Prevalence: ~12% of US population, ~2.7% (>8 million patients) with advanced disease
  - Third leading indication for liver transplant in the US
  - No therapeutics approved, but ~$615M in off label sales reported in US (2010)

- FLINT: 72 week NIDDK-sponsored Phase 2b trial of OCA vs. placebo in 283 patients
  - NIDDK selected OCA based on positive Phase 2 trial data in diabetic fatty liver patients
  - Primary endpoint: liver biopsy-determined improvement in NAFLD Activity Score (NAS) by ≥2 points, with no worsening of fibrosis
  - Positive interim futility analysis in June 2012 based on data from >100 patients
  - Trial results expected in 4Q 2014

- DSP currently enrolling a 200 patient placebo-controlled NASH trial in Japan
Primary bile acid diarrhea (BAD) accounts for up to an estimated 1/3 of all IBS-D patients (i.e., up to 1% of the general population)
- FGF19 production defect: no ‘shut off’ signal for bile acid production
- Patients have high bile acid production and resulting chronic diarrhea

Secondary bile acid diarrhea occurs in Crohn’s patients (potential orphan indication)
- Deficient FGF19 due to insufficient ileal surface area

Current treatment is with bile acid sequestrants (e.g., colesevelam)
- However, ~20% of primary BAD patients do not respond
- Non-response in Crohn’s ranges from 40% (w/o resection) to 60% (w/ resection)

Rational therapeutic approach with OCA: FGF19 is directly regulated by FXR
- Dose dependent induction by OCA seen in 3 completed Phase 2 trials
**OCA for Bile Acid Diarrhea: Phase 2a OBADIAH Trial**

- **OBADIAH**: Phase 2a trial in 30 patients (3 cohorts)
  - Primary BAD
  - Secondary BAD (Crohn’s / ileal resection)
  - IBS-D (normal FGF19)

- Initial results reported at 2013 DDW for Primary BAD patients
  - OCA increased fasting FGF19 from 133 to 237 pg/ml (p=0.007) at 2 weeks
  - Most patients had >60% increases in fasting FGF19
  - Clinical improvements in all patients, including stool frequency (from 23 to 14/week, p=0.03), Bristol Stool Form Scale (from 5.15 to 4.34, p=0.05)

- Final results expected in YE 2013
# OCA Clinical and Regulatory Milestones

<table>
<thead>
<tr>
<th>Indication</th>
<th>Milestone</th>
<th>Anticipated Timing</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Primary Biliary Cirrhosis (PBC)</strong></td>
<td>Initiate Phase 3 clinical outcomes trial</td>
<td>1H 2014</td>
</tr>
<tr>
<td></td>
<td>POISE Phase 3 results available</td>
<td>2Q 2014</td>
</tr>
<tr>
<td></td>
<td>File NDA and MAA for PBC</td>
<td>4Q 2014</td>
</tr>
<tr>
<td><strong>Portal Hypertension</strong></td>
<td>Completion of PESTO trial</td>
<td>4Q 2013</td>
</tr>
<tr>
<td><strong>Bile Acid Diarrhea</strong></td>
<td>OBADIAH final results available</td>
<td>4Q 2013</td>
</tr>
<tr>
<td><strong>NASH</strong></td>
<td>FLINT results available</td>
<td>4Q 2014</td>
</tr>
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Potential Future Product Candidates
Preclinical Pipeline: INT-767, INT-777 and Our TGR5 Program

- INT-767 is a potent FXR agonist with TGR5 agonist activity
  - ~5x more potent than OCA on FXR
  - Potent anti-fibrotic effects in multiple animal models of liver, intestinal and kidney fibrosis

- Servier partnership: focus on discovery of TGR5 agonists for type 2 diabetes and other indications
  - Up to €108.5MM in research funding and milestones, plus tiered single digit percent royalties

- INT-777 is a selective TGR5 agonist
  - Potent inducer of GLP-1 release in animal models of diabetes

Financial Overview
## Key Financial Information

<table>
<thead>
<tr>
<th></th>
<th>March 31, 2013</th>
<th>Sept. 30, 2013</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cash, cash equivalents, investments</td>
<td>$104 million</td>
<td>$157 million</td>
</tr>
<tr>
<td>Debt</td>
<td>$0</td>
<td>$0</td>
</tr>
<tr>
<td>Shares Outstanding</td>
<td>16.6 million</td>
<td>19.3 million</td>
</tr>
</tbody>
</table>
Conclusion
ICPT Investment Highlights

- Intercept (ICPT) was founded in 2002 around a bile acid chemistry drug discovery platform
  - Co-founder Roberto Pellicciari, PhD: rational design of selective bile acid derived NCEs

- OCA in Phase 3 for orphan indication primary biliary cirrhosis with data expected in Q2 2014
  - OCA (obeticholic acid) is a first-in-class farnesoid X receptor (FXR) agonist
  - Two Phase 2 randomized trials met all primary (p<0.0001) and secondary endpoints
  - Addressable population anticipated to be approximately 30,000 patients in developed markets
  - Additional indications focused primarily on chronic liver diseases
  - Patent terms projected through 2028

- Pipeline of follow-on bile acid derived NCEs

- Experienced management team and strong financial position
  - Raised approximately $245 million in equity to date with $157 million of cash as of 9/30/2013
  - Operations funded into early 2016 (through anticipated US and European approvals)