SPRING BANK PHARMACEUTICALS, INC.
(Exact Name of Company as Specified in Charter)

Delaware 001-37718 52-2386345
(State or Other Jurisdiction of Incorporation) (Commission File Number) (IRS Employee Identification No.)

35 Parkwood Drive, Suite 210
Hopkinton, MA 01748
(Address of Principal Executive Offices) (Zip Code)

Company's telephone number, including area code: (508) 473-5993
(Former Name or Former Address, if Changed Since Last Report)

Check the appropriate box below if the Form 8-K filing is intended to simultaneously satisfy the filing obligation of the registrant under any of the following provisions (see General Instruction A.2. below):

☐ Written communications pursuant to Rule 425 under the Securities Act (17 CFR 230.425)
☐ Soliciting material pursuant to Rule 14a-12 under the Exchange Act (17 CFR 240.14a-12)
☐ Pre-commencement communications pursuant to Rule 14d-2(b) under the Exchange Act (17 CFR 240.14d-2(b))
☐ Pre-commencement communications pursuant to Rule 13e-4(c) under the Exchange Act (17 CFR 240.13e-4(c))

Indicate by check mark whether the registrant is an emerging growth company as defined in as defined in Rule 405 of the Securities Act of 1933 (§230.405 of this chapter) or Rule 12b-2 of the Securities Exchange Act of 1934 (§240.12b-2 of this chapter).

Emerging growth company ☒

If an emerging growth company, indicate by check mark if the registrant has elected not to use the extended transition period for complying with any new or revised financial accounting standards provided pursuant to Section 13(a) of the Exchange Act. ☒
Spring Bank Pharmaceuticals, Inc. (the “Company”) is furnishing a presentation, attached as Exhibit 99.1 to this Current Report on Form 8-K, that the Company intends to reference on a publicly available conference call and webcast on April 12, 2019. The presentation will also be available on the Company's website at http://ir.springbankpharm.com/events-and-presentations/presentations.

The information in this Item 7.01 and Exhibit 99.1 attached hereto is furnished and shall not be deemed “filed” for purposes of Section 18 of the Securities Exchange Act of 1934, as amended (the “Exchange Act”), or otherwise subject to the liabilities of that section, nor shall it be deemed incorporated by reference in any filing under the Securities Act of 1933, as amended, or the Exchange Act, except as expressly set forth by specific reference in such filing.

On April 12, 2019, the Company issued a press release announcing results from the Company’s Phase 2 ACHIEVE clinical trial evaluating the use of inarigivir in patients infected with chronic hepatitis B virus. The press release also includes results and findings from additional inarigivir studies. A copy of the press release is attached hereto as Exhibit 99.2 and is incorporated herein by reference.

<table>
<thead>
<tr>
<th>Exhibit No.</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>99.1</td>
<td>Conference Call Presentation</td>
</tr>
<tr>
<td>99.2</td>
<td>Press Release issued April 12, 2019</td>
</tr>
</tbody>
</table>
Pursuant to the requirements of the Securities Exchange Act of 1934, the registrant has duly caused this report to be signed on its behalf by the undersigned hereunto duly authorized.

SPRING BANK PHARMACEUTICALS, INC.

Date: April 12, 2019

By: /s/ Martin Driscoll

Martin Driscoll
President and Chief Executive Officer
Forward Looking Statements

This presentation includes forward-looking statements within the meaning of the Private Securities Litigation Reform Act of 1995. Forward-looking statements include, among other things, statements other than historical facts, regarding the progress, scope, duration or results of clinical trials and preclinical studies of inargir, SB 9225, SB 11285 or any of our other product candidates or programs, such as the size, design, population, conduct, costs, objective or endpoints of any clinical trial, or the timing for initiation or completion of or availability of results from any clinical trial; the potential benefits that may be derived from any of our product candidates; our future operations, financial position, revenues, costs, expenses, uses of cash, capital requirements or our need for additional financing; or our strategies, goals, milestones, prospects, beliefs, intentions, plans, expectations, forecasts or objectives. Words such as “anticipates,” “believes,” “plans,” “expects,” “projects,” “future,” “intends,” “may,” “will,” “should,” “could,” “estimates,” “predicts,” “potential,” “continue,” “guidance,” and similar expressions sometimes identify forward-looking statements. Any forward-looking statement involves known and unknown risks, uncertainties and other factors that may cause our actual results, levels of activity, performance or achievements to differ materially from those expressed or implied by such forward-looking statement, and therefore, you are cautioned not to place undue reliance on any forward-looking statement. These factors include, but are not limited to, whether our cash resources will be sufficient to fund our continuing operations for the period anticipated, the components, timing, costs and results of our clinical trials, preclinical studies and other development activities involving our product candidates; whether certain top-line results from our clinical trials materially change as more information becomes available; whether results obtained in preclinical studies and clinical trials will be indicative of results obtained in future clinical trials; whether inargir, SB 9225, SB 11285 and any of our other product candidates will advance through the clinical trial process on a timely basis and receive approval from the United States Food and Drug Administration or equivalent foreign regulatory agencies; and whether, if inargir, SB 9225, SB 11285 or any of our other product candidates obtain regulatory approval, it will be successfully distributed and marketed. These and other risks and uncertainties that we face are described in our most recent Annual Report on Form 10-K, filed with the Securities and Exchange Commission (SEC) on March 11, 2019, and in other filings that we make with the SEC from time to time.

All forward-looking statements speak only as of April 12, 2019 and should not be relied upon as representing our views as of any other date. We specifically disclaim any obligation to update any forward-looking statement, except as required by applicable law. All trademarks, service marks, trade names, logos and brand names identified in this presentation are the properties of their respective owners.

This presentation also contains estimates and other statistical data generated by independent parties and by us relating to market size and statistics. These estimates involve a number of assumptions and limitations, and you are cautioned not to give undue weight to such estimates.
Background

- Achievement of “Functional Cure” in HBV requires:
  
  - Loss of HBsAg or sustained suppression of HBV DNA off all treatment after a finite duration of therapy
  
  - Safety and tolerability profile similar to NUCs
  
  - Immune control of cccDNA/ HBV replication - critical for achieving a sustained response

- Inarigivir (IRIG) is being developed as an oral immunomodulatory backbone agent for combination strategies to achieve HBV Functional Cure
Inarigivir Acts Through Modulation of the Innate Immune System Involving RIG-I

**Novel mechanism of action**

- Actively transported into hepatocytes via OATP-1 and OAT-1 with 30:1 liver to plasma ratio
- Binds to RIG-I and causes induction of IFN signaling
- Demonstrated activation of immune system in HCV patients and healthy volunteers at 400mg daily
- DAA effect to prevent interaction of HBV Pol and pgRNA in cell systems
- Active against polymerase and capsid resistant strains
- Activates "host" targets instead of viral targets – potential for higher barrier to viral resistance

**RIG-I is a sentinel protein involved in the body's innate defense system**

---

**Diagram:**

- **Viral infection**
- **RIG-I activation and dimerization**
- **CARD: CARD interaction between RIG-I and MIF-2**
- **Antiviral signal transduction**
- **IHF3 induction**
- **Protection of uninfected cells from viral infection**
- **Apoptosis of infected cells**

---

**springbank**
Healthy Volunteers Trial Design

Inarigivir 400 mg /Daily

12 days: 14 healthy volunteers

Day 1

Day 12

11 Days 400 mg

Samples

0

2h

6h

24h

0

4h

12h

Healthy Volunteers Trial Design

Inarigivir 400 mg /Daily

12 days: 14 healthy volunteers

Day 1

Day 12

11 Days 400 mg

Samples

0

2h

6h

24h

0

4h

12h

Cytokines in sera (IFN-a, IP-10, TNF-a, IFN-g, IL-6, IL-12p70)

PBMC for flow cytometry analysis (T, NK, myeloid cells activation)

PBMC for Nanostring analysis

Inarigivir, a RIG-I agonist, activates innate immunity in healthy volunteers. Nina Le Bert¹, Kamini Kunasegaran¹, Meiyin Lim¹, Kevin Leach², Radhakrishnan Iyer³, Antonio Homel⁴, Nezar Atrash⁵, ‘O’Connell-NUS Medical School, Emerging Infectious Diseases Program, Singapore, Singapore; Singapore; Spring Bank Pharmaceuticals, Hopkinton, United States

Inarigivir, a RIG-I agonist, activates innate immunity in healthy volunteers. Nina Le Bert¹, Kamini Kunasegaran¹, Meiyin Lim¹, Kevin Leach², Radhakrishnan Iyer³, Antonio Homel⁴, Nezar Atrash⁵, ‘O’Connell-NUS Medical School, Emerging Infectious Diseases Program, Singapore, Singapore; Singapore; Singapore; Spring Bank Pharmaceuticals, Hopkinton, United States
Evidence of Immune Activation without Systemic Cytokine Toxicity

- Serum cytokines levels of IFN-a, IFN-g, TNF-a, IL-6 and IL-12p70 were undetectable while IP-10 levels declined after inarigivir treatment
- As early as 2h post treatment, phenotypic analysis showed uniform up-regulation of activation markers on monocytes (CCR2, CD16, CD86) and dendritic cells (CD86)
- The frequency of peripheral NK and CD8+ T cells declined and was associated with reduction of activating receptor NKG2D (NK cells) and increase of activation markers CD39 and HLA-DR (T cells)
- PBMC gene induction of innate immune markers was demonstrated by nanostring analysis
- Measurements of immune cell activation before and after the first and final dose demonstrated a similar response with no evidence of tolerance
Inarigivir Cross-Resistance Studies

- Inarigivir effective against all known NUC resistant variants
- Inarigivir effective against pre-core mutation stop codon G1896A
- Inarigivir effective against all known capsid resistance variants

- Inarigivir will cover pre-existing NUC / CpAM variants and prevent emergent resistance variants
ACHIEVE Phase 2 Dose Escalation Study

Inarigivir monotherapy 12 weeks followed by switch to Tenofovir 300mg for 12 weeks

Up to 60 non-cirrhotic HBV subjects, randomized 4:1 between Inarigivir and placebo

<table>
<thead>
<tr>
<th>Dose</th>
<th>Cohort 1</th>
<th>Cohort 2</th>
<th>Cohort 3</th>
<th>Cohort 4</th>
</tr>
</thead>
<tbody>
<tr>
<td>Inarigivir - 25 mg</td>
<td>Inarigivir - 50 mg</td>
<td>Inarigivir - 100 mg</td>
<td>Inarigivir - 200 mg</td>
<td>Placebo</td>
</tr>
</tbody>
</table>

Cohorts:
- Cohort 1: Inarigivir - 25 mg
- Cohort 2: Inarigivir - 50 mg
- Cohort 3: Inarigivir - 100 mg
- Cohort 4: Inarigivir - 200 mg
- Placebo

All patients switch to tenofovir 300 mg monotherapy

**PRIMARY ENDPOINT**
- Safety and antiviral activity at 12 weeks

**SECONDARY ENDPOINT**
- PK, change in serum HBV DNA, HBsAg, HBV RNA and HBeAg from baseline to weeks 6, 12, 14, 16, and 24
<table>
<thead>
<tr>
<th></th>
<th>Pbo Epos</th>
<th>Pbo Eneg</th>
<th>E+ve 25mg</th>
<th>E-ve 25mg</th>
<th>E+ve 50mg</th>
<th>E-ve 50mg</th>
<th>E+ve 100 mg</th>
<th>E-ve 100 mg</th>
<th>E+ve 200 mg</th>
<th>E-ve 200 mg</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>n</strong></td>
<td>8</td>
<td>8</td>
<td>9</td>
<td>7</td>
<td>11</td>
<td>5</td>
<td>13</td>
<td>4</td>
<td>8</td>
<td>7</td>
</tr>
<tr>
<td><strong>Age</strong></td>
<td>35</td>
<td>48</td>
<td>37</td>
<td>43</td>
<td>36</td>
<td>47</td>
<td>34</td>
<td>46</td>
<td>42</td>
<td>52</td>
</tr>
<tr>
<td><strong>M:F</strong></td>
<td>7:1</td>
<td>5:3</td>
<td>5:5</td>
<td>3:3</td>
<td>9:2</td>
<td>5:0</td>
<td>7:6</td>
<td>3:1</td>
<td>4:4</td>
<td>2:5</td>
</tr>
<tr>
<td><strong>ALT</strong></td>
<td>85</td>
<td>53</td>
<td>82</td>
<td>75</td>
<td>75</td>
<td>65</td>
<td>75</td>
<td>90</td>
<td>54</td>
<td>73</td>
</tr>
<tr>
<td><strong>HBV DNA</strong></td>
<td>7.64</td>
<td>4.75</td>
<td>7.86</td>
<td>5.69</td>
<td>7.79</td>
<td>4.55</td>
<td>8.20</td>
<td>5.95</td>
<td>7.88</td>
<td>4.95</td>
</tr>
<tr>
<td><strong>HBV RNA</strong></td>
<td>6.44</td>
<td>2.23</td>
<td>6.36</td>
<td>4.2</td>
<td>6.58</td>
<td>1.54</td>
<td>7.23</td>
<td>2.77</td>
<td>6.68</td>
<td>2.86</td>
</tr>
<tr>
<td><strong>HBsAg</strong></td>
<td>4.17</td>
<td>2.79</td>
<td>4.32</td>
<td>3.17</td>
<td>4.13</td>
<td>2.96</td>
<td>4.38</td>
<td>2.68</td>
<td>4.15</td>
<td>2.72</td>
</tr>
<tr>
<td><strong>GT A</strong></td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>GT B</strong></td>
<td>2</td>
<td>6</td>
<td>4</td>
<td>3</td>
<td>3</td>
<td>4</td>
<td>4</td>
<td>3</td>
<td>2</td>
<td>5</td>
</tr>
<tr>
<td><strong>GT C</strong></td>
<td>6</td>
<td>1</td>
<td>5</td>
<td>7</td>
<td>1</td>
<td>8</td>
<td>1</td>
<td>6</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td><strong>GT D</strong></td>
<td>6</td>
<td>2</td>
<td>1</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*9 HBeAg negative patients had undetectable HBV RNA at baseline*
Primary Endpoint: Mean Change from Baseline in HBV DNA at Week 12 in Placebo (PL) and IRIG Cohorts
HBeAg-Positive Patients: Change from Baseline in HBV DNA at Week 12 and Week 24

- Log₁₀
- Week 12 PL or IRIG
- Week 24 TDF 300mg
- Mean change (per cohort)
- P< 0.01: IRIG 50, 100 and 200mg vs PL

WEEKS 0 – 12
PL 25mg 50mg 100mg 200mg

WEEKS 12 – 24
TDF 300mg switch
PL 25mg 50mg 100mg 200mg
HBeAg-Negative Patients: Change from Baseline in HBV DNA at Week 12 and Week 24

18 of 22 (82%) patients undetectable at week 24

P< 0.01: IRIG 100mg and 200mg versus PL
Secondary Endpoint: Mean Change from Baseline in HBV RNA at Week 12 in Placebo (PL) and IRIG Cohorts

- Log$_{10}$ values for placebo and IRIG doses:
  - Placebo (PL): -0.1
  - 25mg: -1.0
  - 50mg: -0.8
  - 100mg: -0.81
  - 200mg IRIG: -1.14
HBeAg-Positive Patients: Change from Baseline in HBV RNA at Week 12 and Week 24

Log_{10}

Week 12 PL or IRIG
Week 24 TDF 300mg
Mean change (per cohort)

P < 0.01: IRIG 50, 100 and 200mg vs PL
HBeAg-Negative Patients: Change from Baseline in HBV RNA at Week 12 and Week 24

P = 0.05: All cohorts combined versus PL at week 12

3 placebo and 6 IRIG undetectable HBV RNA at baseline. 1 placebo became replicative and detectable at week 12

Week 12 PL or IRIG
Week 24 TDF 300mg
Mean change (per cohort)

All IRIG patients at 50, 100 and 200mg became undetectable at week 12
Baseline HBsAg Cutoff of $4\log_{10}$ Predictor of HBV DNA and HBV RNA Response to IRIG at Week 12

Change from Baseline to Week 12 $\log_{10}$

- Black circles: HBV DNA
- Red triangles: HBV RNA
- Mean change

P < 0.001 for both HBV DNA and HBV RNA

24 HbeAg +ve and 1 HbeAg -ve > $4\log_{10}$
16 HbeAg +ve and 21 HbeAg -ve < $4\log_{10}$
Positive Predictors of Response for IRIG

- **HBV DNA and HBV RNA**
  - Baseline HBsAg < $4\log_{10}$
  - Baseline IP-10 > 310ng/L
  - Reduction in IP-10 > 110ng/L between baseline and week 12

- **HBsAg**
  - Genotype B > C
  - Good responses genotype A / D but numbers small
### Secondary Endpoint: Predefined Responders with HBsAg Reduction of $\geq 0.5 \log_{10}$

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Week 12 &gt;0.5log$_{10}$</th>
<th>Week 24 &gt;0.5log$_{10}$</th>
<th>Total Responders</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Placebo / TDF</td>
<td>1$^*$</td>
<td>2$^*$</td>
<td>2</td>
<td>*ALT flare &gt; 400 IU/ml</td>
</tr>
<tr>
<td>IRIG 25mg / TDF</td>
<td>4$^#$</td>
<td>6</td>
<td>8</td>
<td>*2 non sustained of which 1 dose reduced</td>
</tr>
<tr>
<td>IRIG 50mg / TDF</td>
<td>1$^5$</td>
<td>2</td>
<td>2</td>
<td>*1 non sustained and dose reduced</td>
</tr>
<tr>
<td>IRIG 100mg / TDF</td>
<td>1</td>
<td>2</td>
<td>2</td>
<td>1 non-sustained with a flare</td>
</tr>
<tr>
<td>IRIG 200mg / TDF</td>
<td>1</td>
<td>3</td>
<td>3</td>
<td>2 GT C patients</td>
</tr>
</tbody>
</table>

- 16 IRIG patients (26%) met predefined HBsAg loss criteria for response at week 12 or 24
- Response in 7 HBeAg negative (mean 0.7log$_{10}$) and 9 HBeAg positive (mean 0.9log$_{10}$)
- Overall mean responder reduction of 0.8log$_{10}$ (range 0.5 – 1.4log$_{10}$)
Quantitative HBsAg Responder Patients > 0.5log_{10} Reduction at Week 12 or Week 24 from Baseline

Mean Change in HBsAg
Week 12: 0.4log_{10}
Range 0.1 – 0.9log_{10}
Week 24: 0.72log_{10}
Range 0.15 – 1.4log_{10}
HBsAg Response (> 0.5 log\(_{10}\)) by Genotype

Percentage of responders within each Genotype

GT A 100%
GT B 33%
GT C 10%
GT D 75%

Genotype response data consistent with that seen with IFN therapy
Preliminary Safety Analysis

- Most common mild / moderate TEAE’s all groups
  - Headache / dizziness 13; fatigue 9; abd pain/GI upset 10;
  - Flu / flu like symptoms 5 URTI 10; ALT / AST elevation: 8
- 1 Grade 3 transient hypertriglyceridemia not sustained on retesting
- 1 SAE of hospitalization for knee pain, patient on placebo in cohort 4
- No investigator determined interferon-like side effects
- No difference between active Rx or placebo and no dose dependency
- 3 patients discontinued
  - 1 placebo after hospitalization for knee pain
  - 2 in 50mg group withdrew consent at Day 1 and Day 14 for patient preference
Conclusion

- **IRIG demonstrated dose dependent responses for HBV DNA and HBV RNA**
- **Baseline HBsAg major predictor of HBV DNA and HBV RNA response**
- **HBsAg response seen in 26% of patients at either 12 or 24 weeks**
  - Responders seen at every dose and may indicate response more dependent on host immunity
  - More common in Genotype B versus C
- **Safety profile excellent with good tolerability and no systemic interferon like effects**
- **IRIG can be considered as a backbone immunomodulator in combination studies with agents having different MOAs**
Spring Bank Announces Positive Results from the Recently Completed Phase 2 Chronic Hepatitis B ACHIEVE Trial and Additional Inarigivir Studies

Consistent reduction in HBV DNA demonstrated with higher inarigivir doses including in high viral burden HBeAg-positive patients

26% HBsAg responder population for all inarigivir doses showed mean HBsAg change of 0.8log\(_{10}\) with a range of 0.5log\(_{10}\) - 1.4log\(_{10}\)

Inarigivir 400mg rapidly and uniformly increased activation markers of innate immunity without evidence of tolerance in a new inarigivir study in healthy volunteers

Favorable safety and tolerability profile observed across all doses studied

Conference call to be held today, April 12 at 8:00am EDT

HOPKINTON, Mass., Apr. 12, 2019 – Spring Bank Pharmaceuticals, Inc. (Nasdaq: SBPH), a clinical-stage biopharmaceutical company developing novel therapeutics for the treatment of viral infections, inflammatory diseases and certain cancers, today announced at The International Liver Congress™ (ILC), the 2019 Annual Meeting of the European Association for the Study of the Liver (EASL) in Vienna, Austria, robust results from the recently completed inarigivir Phase 2 dose escalation ACHIEVE trial for patients with chronic hepatitis B virus (HBV). Spring Bank is developing inarigivir, an orally-administered hepatic-selective immunomodulator, as a potential backbone in a combinatorial treatment for chronic HBV with the goal to accelerate and substantially increase chronic HBV functional cure rates in a simple, safe and selective manner.

In an oral presentation during the General Session II Award Ceremony I at the ILC earlier today, Professor M.F. Yuen, Chief of Gastroenterology and Hepatology, University of Hong Kong, and Principal Investigator for the ACHIEVE trial, presented top-line results from all cohorts including the final 200mg dosing cohort. The data show that inarigivir continued to demonstrate a dose-dependent effect on HBV DNA and HBV RNA in the fourth cohort of the ACHIEVE trial over the 12-week dosing period. Inarigivir 200mg monotherapy dosing exhibited a mean decrease of 1.54log\(_{10}\) in HBV DNA with a range of 0.71log\(_{10}\) to 3.26log\(_{10}\) and a mean decrease of 1.14log\(_{10}\) in HBV RNA with a range of 0.08log\(_{10}\) to 4.88log\(_{10}\). Inarigivir 200mg monotherapy showed a uniform antiviral response (HBV DNA) in all patients, in particular the high viral burden HBeAg-positive patients who had not responded to the lower doses of inarigivir monotherapy in the earlier ACHIEVE cohorts. In addition, across all patients HBV DNA and HBV RNA responses strongly correlated with baseline HBsAg, baseline IP-10 and the decline of IP-10 at week 12, comparable to the clinical experience with interferon in chronic HBV.

Across all four dosing cohorts, 16 of the 62 (26%) evaluable patients treated with inarigivir had a 0.5log\(_{10}\) or greater reduction in HBsAg at week 12 or week 24 and were categorized as responders. The mean reduction of HBsAg in this responder population was 0.8log\(_{10}\) with a range of 0.5log\(_{10}\) to 1.4log\(_{10}\). Importantly, the HBsAg response to inarigivir in the ACHIEVE trial was genotype and host dependent. Although the number of patients was small, the HBsAg response seen in Genotype A and D was excellent with 4 of 5 patients responding. Genotype B and C were the most common genotypes and 33% of
genotype B responded compared to 10% of genotype C. It is important to note that 2 of the 3 genotype C patients that responded received 200mg inarigivir dose and belonged to the higher viral burden HBeAg-positive group. This amplified HBsAg response observed with inarigivir in genotype B compared to genotype C is comparable to the clinical experience of the immunomodulator interferon in the treatment of chronic HBV.

The overall data from the ACHIEVE trial also revealed evidence of a significant shutdown of viral transcription by inarigivir in HBeAg-negative patients where HBV RNA was undetectable at week 12 in all patients treated with inarigivir at the 50mg, 100mg, and 200mg doses with a concomitant undetectable HBcrAg in the majority of these patients. At week 24, the HBV RNA and HBcrAg response was sustained with the switch to tenofovir disoproxil fumarate 300mg and associated with 18 of the 22 (82%) HBeAg-negative patients also having undetectable HBV DNA.

Inarigivir was shown to be well tolerated at all doses in the ACHIEVE trial. Treatment-emergent adverse events ranged from mild to moderate in severity, with no investigator determined interferon-like side effects. The percentage of treatment-emergent adverse effects reported in the ACHIEVE trial was similar between the active and placebo groups. In the fourth cohort, one Grade 3 event (hypertriglyceridemia) was observed, but was not sustained on retesting, and there was one serious adverse event (SAE) for knee pain hospitalization reported, but this patient was dosed with placebo. There were no other clinical or biochemical events above Grade 3 during the ACHIEVE trial.

“The data from the ACHIEVE trial demonstrate a clear dose dependent antiviral response on HBV DNA and HBV RNA with inarigivir treatment,” said Professor M.F. Yuen. “Importantly, inarigivir treatment of HBeAg- negative patients in this study suggests a rapid down-regulation of cccDNA transcription.” Professor Yuen continued, “The HBsAg response of a 0.5log_{10} or greater reduction in the inarigivir-treated patients is the best reported to date with an oral agent in the treatment of chronic HBV patients, with a similar genotype distribution of responders to that reported for interferon, the only approved immunomodulator treatment for chronic HBV.”

Additional inarigivir data announced highlighting immune-activation with the 400mg dose

Spring Bank also announced today the results from a healthy volunteer study that examined the potential for the use of the 400mg dose of inarigivir in chronic HBV patients. The results from this study revealed that inarigivir 400mg rapidly and uniformly increased activation markers of innate immunity on circulating peripheral monocytes and dendritic cells which was sustained over a ten-day period of dosing without evidence of tolerance. There was an associated activation of CD8+ T-cells and down-regulation of NK cells resulting in a potentially favorable adaptive immune profile for an antiviral response. The results from this study also demonstrated a lack of systemic cytokine activation secondary to the intra-hepatic targeting of inarigivir resulting in a favorable tolerability profile. These compelling immune-activation data for inarigivir, together with the dose-dependent effects on HBV DNA and HBV RNA and favorable tolerability profile observed up to 200mg daily in the ACHIEVE trial, serve as the basis for the recent launch of the Spring Bank global CATALYST trials in both treatment-naïve and virally-suppressed chronic HBV patients. The CATALYST trials will focus on the examination of the 400mg dose of inarigivir.

“The increasing antiviral response seen at the 200mg dose, especially in high viral burden patients, and the clear evidence of innate immune activation at the 400mg dose in healthy volunteers with good
tolerability, has led Spring Bank to choose 400mg as the inarigivir dose to move forward into longer studies focused on HBV cure,” said Nezam Afdhal, M.D., D.Sc., chief medical officer of Spring Bank Pharmaceuticals. “We have developed a comprehensive strategic clinical trial program that takes into account both patient and viral heterogeneity that we anticipate will inform our Phase 3 strategy moving into 2020.”

**EASL 2019 ILC Poster Presentation demonstrating antiviral activity of inarigivir in nucleoside- and capsid-resistant HBV variants**

In a poster presentation at EASL 2019 ILC, Professor Stephen Locarnini, Head of Research & Molecular Development at the Victorian Infectious Diseases Reference Laboratory and Principal Investigator of the Virology Core for the ACHIEVE trial, described his in vitro study examining the antiviral activity of inarigivir in HBV clinical isolates consisting of capsid inhibitor (CpAM) and nucleoside (NUC) analog resistant variants. Looking forward to novel combinations with agents under investigation with differing mechanisms of action, Dr. Locarnini evaluated inarigivir in NUC- and CpAM-resistant clinical isolates from HBV patients. Professor Locarnini’s data indicate that inarigivir could be used as rescue or combination therapy for NUC/CpAM-resistant failures.

Professor Locarnini stated, “The sensitivity of both NUC-resistant and CpAM-resistant strains to inarigivir and the lack of cross-resistance is beneficial for combination studies since inarigivir will cover both pre-existing resistant variants and prevent treatment-emergent resistance to both NUCs and CpAM inhibitors,” said Dr. Locarnini.

**Inarigivir Clinical Development**

Spring Bank has launched two Phase 2 global trials (CATALYST 1 and CATALYST 2) examining the administration of inarigivir 400mg as monotherapy and co-administered with a NUC in naïve and virally-suppressed chronic HBV patients. The CATALYST trials include multiple patient cohorts with dosing periods to include 12 weeks, 24 weeks, and 48 weeks. In addition, Gilead Sciences, Inc. is conducting a Phase 2 trial examining (i) the co-administration of inarigivir 50mg and tenofovir alafenamide 25mg (marketed by Gilead as Vemlidy®) in naïve chronic HBV patients, (ii) the co-administration of inarigivir 200mg and Vemlidy® in naïve chronic HBV patients and (iii) the administration of inarigivir 100mg in virally-suppressed patients who currently are and continue to be treated with a NUC.

**Conference Call**

Spring Bank will host a conference call and webcast at 8:00 am EDT today, Friday, April 12, 2019. The conference call may be accessed by dialing (877) 407-0789 for U.S. callers and (201) 689-8562 for international callers and providing the conference ID 13689400. Additionally, a live, listen-only webcast of the conference call can be accessed by visiting the Investors & Media section of the company’s website at [www.springbankpharm.com](http://www.springbankpharm.com) or by clicking here.

A replay of the call may be accessed by visiting Spring Bank’s website.

Additional details, including presentation abstracts, can be found on the ILC website at [https://ilc-congress.eu/](https://ilc-congress.eu/). A copy of the presentation materials can be accessed by visiting the Presentations and Publications section of the Spring Bank Pharmaceuticals website.
About Spring Bank Pharmaceuticals
Spring Bank Pharmaceuticals, Inc. is a clinical-stage biopharmaceutical company engaged in the discovery and development of a novel class of therapeutics using its proprietary small molecule nucleotide platform. The company designs its compounds to selectively target and modulate the activity of specific proteins implicated in various disease states. The company’s lead product candidate, inarigivir, is being developed for the treatment of chronic hepatitis B virus (HBV). Inarigivir is designed to activate within hepatic cells retinoic acid-inducible gene 1 (RIG-I), which has been shown to inhibit HBV viral replication and induce the intracellular interferon signaling pathways for antiviral defense. The company is also developing its lead STING agonist product candidate, SB 11285, an immunotherapeutic agent for the treatment of selected cancers. For more information, please visit www.springbankpharm.com.

Forward-Looking Statements
Statements in this press release about Spring Bank’s future expectations, plans and prospects, as well as any other statements regarding matters that are not historical facts, may constitute forward-looking statements within the meaning of The Private Securities Litigation Reform Act of 1995. These statements include, but are not limited to, statements about the potential of inarigivir as a drug. The words “anticipate,” “believe,” “estimate,” “expect,” “intend,” “may,” “plan,” “predict,” “project,” “will,” “would,” “could,” “potential,” “possible,” “hope” and similar expressions are intended to identify forward-looking statements, although not all forward-looking statements contain these identifying words. Such statements are subject to numerous important factors, risks and uncertainties that may cause actual events or results to differ materially from current expectations and beliefs. For example, there can be no guarantee that any product candidate will be successfully developed or complete necessary preclinical and clinical phases, that the results of any clinical study will be predictive for other clinical studies of the same product candidate, or that development of any product candidates will successfully continue. Management’s expectations and, therefore, any forward-looking statements in this press release could also be affected by risks and uncertainties relating to a number of other important factors, including: whether preliminary data reported by Spring Bank changes following a more comprehensive review of the data related to the clinical trial and as more patient data become available or as additional analyses are conducted; whether Spring Bank’s product candidates will advance through the clinical trial process on a timely basis, or at all; whether Spring Bank’s cash resources will be sufficient to fund its continuing operations for the periods and/or trials anticipated; whether the results of such trials will warrant submission for approval from the United States Food and Drug Administration or equivalent foreign regulatory agencies; whether Spring Bank’s product candidates will receive approval from regulatory agencies on a timely basis or at all; whether, if product candidates obtain approval, they will be successfully distributed and marketed; and other factors discussed in the “Risk Factors” section of Spring Bank’s Annual Report on Form 10-K for the year ended December 31, 2018, which was filed with the Securities and Exchange Commission (SEC) on March 11, 2019 and in other filings Spring Bank makes with the SEC from time to time.

In addition, the forward-looking statements included in this press release represent Spring Bank’s views as of the date hereof. Spring Bank anticipates that subsequent events and developments will cause Spring Bank’s views to change. However, while Spring Bank may elect to update these forward-looking statements at some point in the future, Spring Bank specifically disclaims any obligation to do so. These forward-looking statements should not be relied upon as representing Spring Bank’s views as of any date after the date hereof.
Contacts
Spring Bank Pharmaceuticals, Inc.
Jonathan Freve
Chief Financial Officer
(508) 473-5993

LifeSci Advisors, LLC
Ashley R. Robinson
(617) 535-7742
Ashley@lifesciadvisors.com

Source: Spring Bank Pharmaceuticals, Inc.