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INSTITUTIONAL RESEARCH

Healthcare & Biotechnology

COVERAGE INITIATION

November 6, 2016

Spring Bank Pharmaceuticals, Inc.

(Nasdaq CM/SBPH)

BUY

Spring Bank is a clinical stage biotech company developing immune and inflammatory modulating small-chain nucleotide-based pharmaceuticals

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Summary and Investment Highlights

Spring Bank Pharmaceuticals has developed a platform immune-modulatory drug discovery and development engine based upon proprietary hybrid small molecule nucleotide prodrug chemistry expertise. The Company's core technology, **Small Molecule Nucleotide Hybrids (SMNH)**, together with associated chemistries, comprise a library of synthetic short sequence nucleic-acid and nucleotide molecules capable of interacting with target proteins, DNA or RNA to modulate the downstream activity of proteins following translation and replication. In doing so, these SMNH compounds interplay with innate immune sensors to reconstitute or stimulate immune response.

The Company's lead compound, SB 9200 is a novel, first-in-class, oral agonist of innate immunity which upregulates interferon (IFN)-responsive gene expression. Spring Bank initiated a Phase IIa clinical trial in chronic Hepatitis B (CHB) for SB 9200 in June 2016. Pre-clinical data support a mechanism of antiviral action of SB 9200 involving modulation of innate immunity through the induction of endogenous intracellular interferon, thus facilitating interferon induction but potentially by-passing known adverse events associated with exogenous immune-modulation. We are initiating coverage of Spring Bank Pharmaceuticals Inc. with a **BUY** rating based upon the Company's proprietary position in what we believe could become a 3rd generation oligo/nucleotide technology "wave" following direct nucleic acid/gene manipulation technologies such as RNAi and CRISPR.

Investment Thesis

Our **BUY** rating is supported by the following investment thesis. Many intractable diseases, such as certain cancers and viral infections, are proving resistant to legacy monotherapy treatments, thus contributing to a seeming "lack of progress" in increasing treatment cure rates. While recent medical advances

Current Price: $9.59 (11/4/16)

(SPRINGBANK LISTED ON NASDAQ MAY 2016)

FY Ended Dec 31 unless otherwise specified

Estimates (MM's)*	FY2015A	FY2016E	FY2017E
Revenues(000)	**$0.94 A**	**$0.35** A	**$0.00** E
1Q	$0.00	$0.28 A	E
2Q	$0.00	$0.07 A	E
3Q	$0.00	$0.00 A	E
4Q	$0.00	$0.00 E	E
2018 Preliminary Revenue Estimate			**$0.0** E
EPS(loss)	**($2.03)**	**($2.75)** E	**($3.11)** E
1Q		($1.11) A	NA
2Q		($0.62) A	NA
3Q		($0.53) A	NA
		($0.59) E	NA
P/E (x)	NA	**MA**	**NA**
2018 Preliminary EPS (loss)			($3.67)
REV/Share	NA	NA	NA
EV/EBITDA (x)			
Stock Data			
52-Week Range			$7.62-$13.25
Shares Outstanding (mil.)			7.76
Market Capitalization			$74.1MM
Enterprise Value			$63.59MM
Current Ratio (6/16)			4.74X
Book Value/Share (6/16)			$1.74
Price/Book			5.86.X
Average Trading Volume (3-Month)			6,300
Insider Ownership			46.1%
Institutional Ownership			NA
Short interest (Million shares)			0.99
Dividend / Yield			$0.00/0.0%

*Some numbers may not add due to rounding

have permitted some cancers and viral infections to be treated as chronic diseases, current long-term therapies have proven challenging for patients due to inherent toxicities and mutational drift to resistance that is associated with long-term use. While several anti-Hepatitis B (HBV) drugs are approved for clinical use, a significant unmet medical need still exists due to the emergence of antiviral resistance and tolerance, as well as dose-limiting toxicities that contribute to lack of a definitive cure and patient lack of compliance with long-term treatments.

New treatment approaches, led especially by the rapid development of CAR-T technology, have begun to acknowledge the role of immune response in disease progression and cure. As such, legacy monotherapeutic treatments are being replaced with combination therapies. In forming these combination treatments, Researchers and practitioners alike are gravitating towards adding immune modulation to the armament of treatment for both viral infection and cancer thanks to an increasing level of understanding of how eloquently both cancer and viruses manipulate and disable the body's immune response machinery.

Spring Bank Pharmaceuticals stands at the forefront of the development of an entirely new approach to harnessing the immune system which seeks to restore, through endogenous mechanisms, a competent and effective immune system. The Company's has developed a platform nucleotide/nucleic acid combinatorial approach that has now generated lead clinical candidates targeting Hepatitis and other viral infectious diseases where immune competency plays a significant role in long-term outcomes. In addition, the unique mechanism of action of the Company's core technology provides a platform by which its compound library might be deployed as a backbone to many other indications where immune response and stimulation would be appropriate. In short, we consider Spring Bank an attractive long term investment for the following reasons.

- Platform technology with novel mechanisms of action that can be used alone and in combination with other drugs. Multiple multibillion dollar market opportunities;
- Core technology induces immune stimulation independent of the genomic composition of the underlying viral infection or tumor type, does not interact with healthy cells;
- Strong intellectual property portfolio;
- Experienced execution-oriented management;
- Value relative to peers;
- Initial technology validation with Gilead Sciences and Arrowhead Pharmaceuticals partnerships. We expect other partnerships to follow over the next 12-18 months.

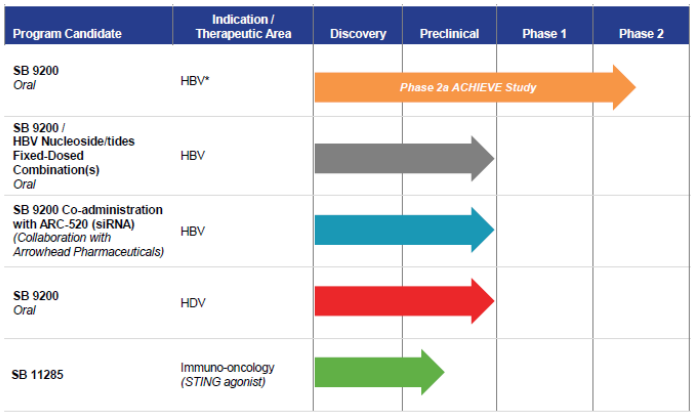
Company Background and Business Strategy

Spring Bank, headquartered in Hopkinton, MA, was founded in 2002 to exploit nucleotide chemistry technology developed by its Chief Science Officer and co-founder, Dr. R.P. (Kris) Iyer. Over the last 15 years, Dr. Iyer has published over 100 scientific articles and several books related to nucleic acid and nucleotide chemistry and on the antiviral agent development challenges associated with nucleotides. His work has resulted in the development of proprietary synthesis techniques, chemistries and a library of Small Molecule Nucleotide Hybrids (SMNHs). The Company brought a lead compound into the clinic in mid 2016 for the treatment of HBV. As of September 30, 2016, the Company reported having 18 full-time employees, of whom, ten hold Ph.D. or M.D. degrees. Spring Bank's executive offices are located at 86 South Street, Hopkinton, MA 01748. The Company's telephone number is (508) 473-5993 and its website address is *www.springbankpharm.com*.

Spring Bank's business is to operate as partially virtual, drug discovery research and development engine for its SMNH platform and related chemistries while outsourcing clinical and manufacturing functions, and to commercialize products through selective collaborations with leading biotechnology and pharmaceutical companies. The Company does not intend to become a fully integrated pharmaceutical business. Spring Bank's business strategy is to capitalize on its position as the leader in the development of SMNH therapeutics for the treatment of viral infections and to expand the use of its platform SMNH technology to non-viral indications such as immune-oncology and inflammatory disease. Spring

Bank intends to accomplish these goals with a two-pronged strategy. For HBV and other viral infections, Spring Bank is driving a rapid clinical process of SB 9200 in CHB infection, for which there is a substantial need for new treatments despite a number of FDA-approved drugs on the market, in order to partner the drug with a global pharmaceutical company for worldwide commercialization. Spring Bank intends to bring forward preclinical proof-of-concept research to support clinical studies for SB 9200 in other viral indications, such as Respiratory Syncytial virus (RSV), HIV latency and Hepatitis D (HDV). Concurrently, Spring Bank believes there is opportunity to leverage the SMNH platform to expand into non-viral therapeutic areas that could benefit from immune modulation or induction and is beginning proof-of-concept work on initial lead compounds. The following exhibit illustrates this strategy along with near-term milestones.

Exhibit 1. Spring Bank Pharmaceuticals Pipeline and Status



Source: Spring Bank Corporate Presentation, Oct. 2016

Lead Clinical Candidate SB 9200 Clinical Progress:

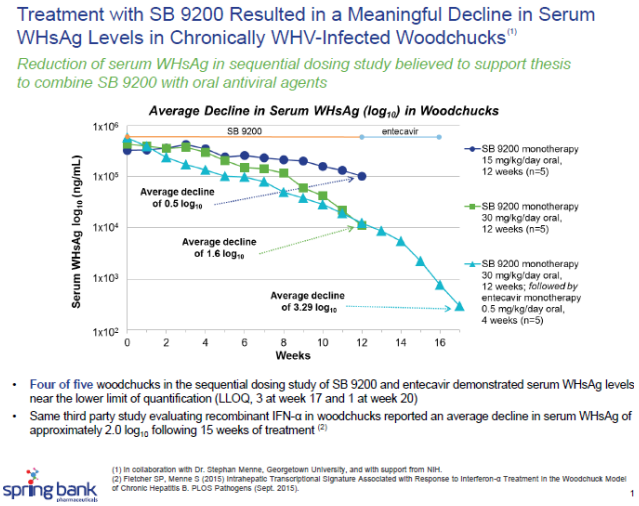
Spring Bank's lead clinical candidate is SB 9200, the first compound from the SMNH library to reach the clinic. SB 9200 is an alkyoxycarbonyloxy linear dinucleotide prodrug that has been optimized by Spring Bank to be an orally-available anti-hepatitis B agent. Bioavailability via oral administration without dose limiting toxicities is a significant challenge with nucleos(t)ide antiviral agents due to their inherent negative charge and relatively large molecular size. Furthermore, SB 9200's site of action is unique in that the molecule interacts with the cytosolic DNA "sensors" (pattern recognition receptors or PRRs) in the cell cytoplasm to circumvent the virus's ability to evade innate immune system activation, and in particular, interferon induction. By blocking the virus's ability to turn off the interferon induction pathway, SB 9200 facilitates normal innate immune system function and may facilitate eventual clearance of the virus. This site of action is independent of other antiviral nucleos(t)ides that seek to interrupt viral replication. Because SB 9200's primary site of action is targeted to the liver cell (hepatocyte) and is not specific to any viral genetic material, the compound is pan-viral and less likely to elicit virus resistance. In addition, it's mode of action is complementary to existing nucleos(t)ide therapeutics and could therefore, become an immune-stimulating backbone to antiviral therapy. We further discuss specific aspects of the SMNH chemistry and the mode of action of SB 9200 later in this report.

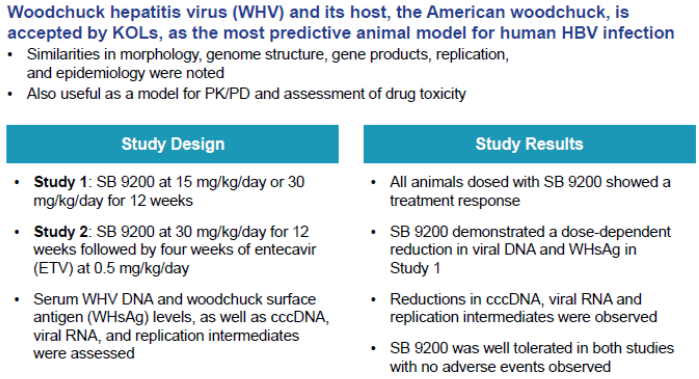
SB 9200 Differentiating Advantages

- Formulated synthetically through proprietary solution-phase organic chemistry that is scalable and reproducible, thus allowing for ease of manufacturing at potentially higher yields and lower cost of goods compared to recombinant proteins and biologicals.
- Less potential for "surprise" variable efficacy in late stage clinical trials or commercial sales due to straightforward and predictable organic chemistry transferable to scaled cGMP manufacturing.
- SMNH mechanism of action and oral delivery-based chemistry facilitates co-formulation and combination therapy opportunities.

Spring Bank conducted a number of preclinical studies in the American woodchuck model to demonstrate the potential efficacy achieved by SB 9200's mode of action prior to the compound entering Phase II clinical trials. The woodchuck model is accepted as the definite animal model for HBV. The following exhibit summarizes the key study endpoints, data derived from measurement of woodchuck hepatitis (WHV) DNA and woodchuck hepatitis surface antigen (WHsAg).

Exhibit 2: Spring Bank Woodchuck Model Pre-clinical Data





Source: Spring Bank Pharmaceuticals

As indicated by this data and other woodchuck data not shown, SB 9200 demonstrated a dose-dependent antiviral potency as measured by log order declines in both serum WHV (not shown) and in serum WHsAg over the 12 week study period. Further, when the woodchucks were treated sequentially with an FDA-approved nucleos(t)ide, the decline in both circulating viral RNA and surface antigen was accelerated. This could suggest that efficacy of the nucleos(t)ide was potentiated by SB 9200.

Following the encouraging animal model data, Spring Bank conducted an ascending dose Phase I trial to demonstrate oral bioavailability, safety, pharmacokinetics and pharmacodynamics of SB 9200. The study was conducted in Australia and New Zealand as a first-in-man, open-label, placebo-controlled, randomized trial in 2014. Thirty-eight non-cirrhotic hepatitis C (HCV), antiviral treatment naïve patients were treated in the ascending dose trial. The following summarizes key findings:

Exhibit 3. Key findings from SB 9200 Phase I HCV Ascending Dose Trial

Source: Spring Bank Pharmaceutical

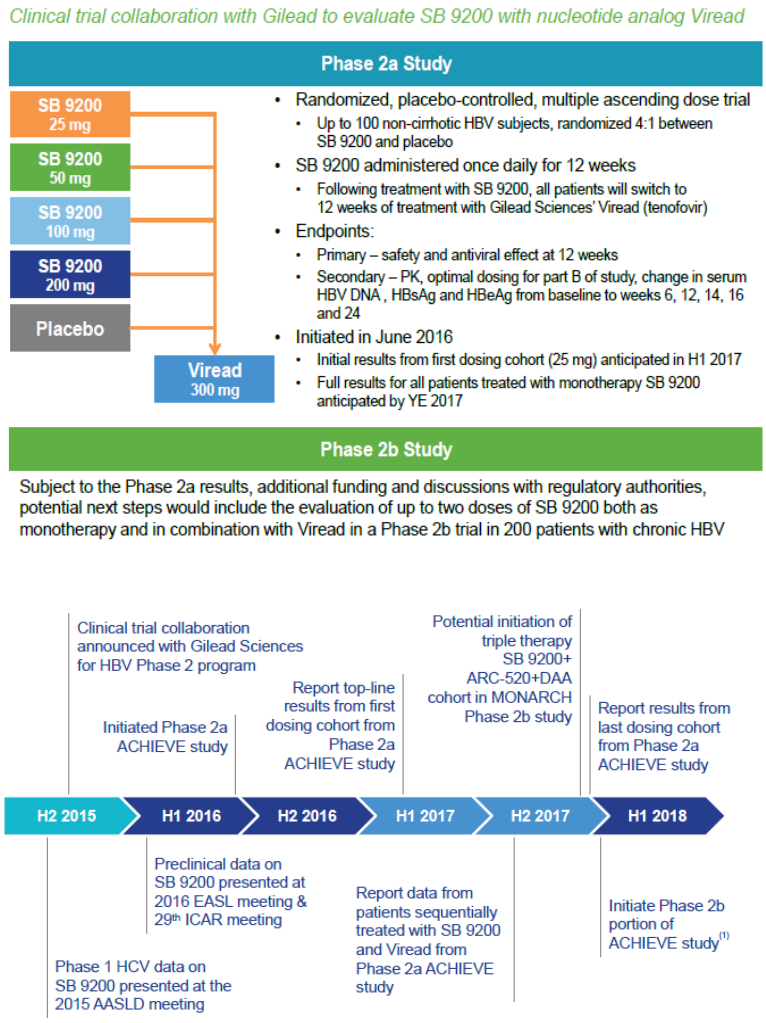
Phase II Clinical Progress

Following its public offering, Spring Bank initiated its **ACHIEVE** Phase IIa trial for SB 9200 in June 2016. The two part, multi-arm, dose-finding trial will evaluate SB 9200 as a monotherapy and as part of a sequential combination therapy with Gilead Sciences' **Viread®.** One hundred non-cirrhotic patients are being randomized on a 4:1 basis, and stratified according to HB envelope protein antigen (HBeAg +/-) status to receive one of four SB 9200 doses as a monotherapy or receive a placebo for the first 12 weeks of the study. Following the monotherapy, all patients will continue on to a 12 week treatment course with either Viread or TAF, Gilead's 2nd generation tenovofir. The primary endpoint of the study will be the reduction in serum HBV DNA from baseline, with secondary endpoints assessing safety and HBsAg and HBeAg levels. There are currently four sites enrolling in Canada and additional sites outside of Canada are expected to begin enrolling over the next several weeks. Read-out from the first cohorts is expected in the first half of 2017.

Following the conclusion of and subject to the results of the ACHIEVE Phase IIa (and assuming additional funding) Spring Bank will initiate a Phase IIb trial in 2017 with chronic HBV patients. This trial protocol will be similar to that of the Phase IIa trial. SB 9200 will be evaluated at two monotherapy doses and also in combination with Viread or TAF. This trial would enroll 200 patients.

It should be noted here that Gilead Sciences has been deeply involved with this clinical collaboration since its inception in 2015. Gilead has contributed expertise and insights into SB 9200's current and future clinical trial protocols as well as working with Spring Bank on developing a co-formulation of SB 9200 and Viread or TAF. Further, as part of this collaboration, Gilead is also contributing Viread and/or TAF at no cost to Spring Bank. Assuming favorable results from the ACHIEVE trial, Spring Bank would partner with a global pharmaceutical company to continue the clinical process towards a goal of global approval for SB 9200.

Exhibit 4. SB 9200 ACHIEVE Trial Phase lIa and Timeline



(1) Subject to Phase IIa results, regulatory discussion and additional funding
Source: Spring Bank Pharmaceuticals Corp. presentation, Oct. 2016

Multiple SB 9200 Analog Compound Pipeline: SB 9400, SB 9941 and SB 9946

SB 9400, SB 9941 and SB 9946 are next-generation analogs of SB 9200 that are being developed as antivirals targeting RIG-I and NOD2, cytosolic DNA sensors (CDSs), against various viruses. Spring Bank is currently conducting preclinical evaluation of these analogs against other viral diseases so as to expand the applications of its SMNH compounds. Subject to funding, other viral disease targets Spring Bank is interested in pursuing for the SB 9200 family include RSV, rhinovirus and HDV. SB 9200 or an analog may also be of interest in addressing HIV latency, for which the Company would seek to collaborate with major research centers and other third parties with significant expertise in HIV.

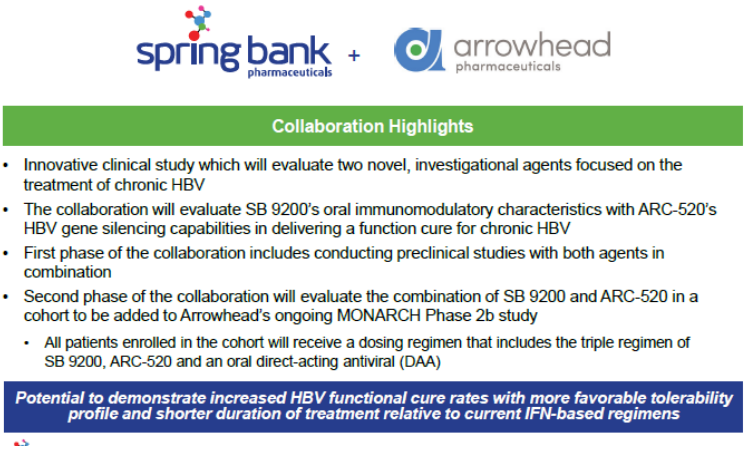
Recent Events:

Arrowhead Pharmaceuticals and Spring Bank Announce Novel Collaboration

Arrowhead Pharmaceuticals and Spring Bank announced on October 6th the initiation of an innovative clinical collaboration that seeks to bring together the companies' respective lead clinical assets in a novel HBV combination therapy. The collaboration will initially evaluate the combination of SB 9200 and ARC-520, Arrowhead's ARC-520 Phase II RNAi compound, in preclinical models before the SB 9200/ARC-520 combination is added as an additional cohort in Arrowhead's currently enrolling **MONARCH** Phase IIb trial. SB 9200 will add an immuno-modulatory component to the RNAi gene silencing mechanism of ARC-520. The combination will be further evaluated as part of a regimen incorporating already approved nucleotide(side) polymerase inhibitor agents (NUCs) such as tenofovir and entecavir. The companies hope that by applying a multi-disciplinary, triple combination approach to HBV treatment, HBV functional cure rates can be increased with better treatment tolerability, and perhaps a shorter treatment duration compared to current standard of care with interferon-based therapies.

The ARC-520 technology uses RNAi machinery to direct specific cleavage of HBV RNA transcripts, thereby reducing the levels of HBV proteins and the RNA template used to produce viral DNA. Reducing the load of circulating and non-circulating viral proteins and viral RNA will hopefully allow for re-constitution of an effective host immune response and ultimately increase the likelihood of HBsAg seroconversion. In this setting, SB 9200 functions as an immune-stimulatory adjuvant which may provide a "one plus one equals more than two" augmentation of the patient's own immune system. We consider this an important first step for Spring Bank in terms of validating the Company's technology as an immuno-modulatory platform that may be broadly applicable across a number of indication settings.

Exhibit 5. Triple Combination of SB 9200, ARC-520 and Direct-acting Antiviral Study Plan MONARCH Phase 2b Study



Source: Spring Bank Pharmaceuticals

Unveiling of SB 11285-First STING (STimulator of INterferon Genes) Agonist Candidate

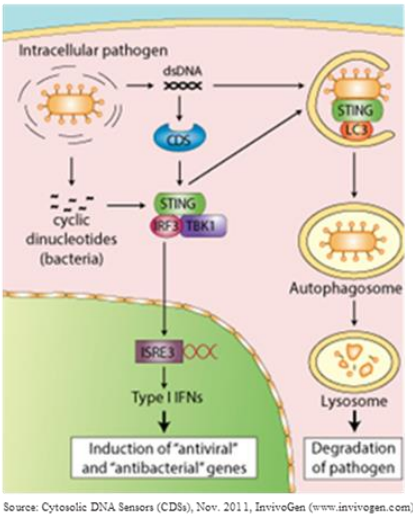
Concurrently with the development of SB 9200 and its analogs for viral diseases, Spring Bank has been researching other pathways in the immune system where its novel SMNH nucleic acid chemistries might offer unique treatment solutions and provide potentially competitive advantages due to their small molecule-like synthetic chemistries, ease of manufacturing and oral bioavailability. One such opportunity lies in the mediation of interferon-stimulating genes and NF-κB induction in tumor immune response and auto inflammatory diseases. With the exception of RNA viruses, which are directly recognized by the cytosolic sensors/pattern recognition receptors (PRRs), virtually all other foreign material is recognized by DNA in the cytoplasm, which the cell knows is an abnormal location. Similar to the cell's surveillance of viral material, tumor associated DNA also triggers activity by cytosolic sensors/pattern recognition receptors, including RIG-1, and other apoptosis-related immune responders. STING plays a central role in facilitating immune cell activity and apoptosis through the direct and indirect mediation of interferons that lead to immune cell priming.

It is being recognized that induction of type I interferons (IFN) and interferon-stimulated genes (ISGs) in tumor cells and within the tumor microenvironment (TME) is essential for modulating the host immune response to tumors. In the last two years, there has been increasing evidence of STING's role (and opportunity as a therapeutic target) in the mediation of immune response as evidenced by the following authors' conclusions in a journal article published in 2014. "*In the tumor microenvironment in vivo, tumor cell DNA was detected within host antigen-presenting cells, which correlated with STING pathway activation and IFN-b production. Our results demonstrate that a major mechanism for innate immune sensing of cancer occurs via the host STING pathway, with major implications for cancer immunotherapy* (Woo et al, **Immunity** 41, 830–842, November 20, 2014 ©2014 Elsevier Inc). STING gained substantial validation in 2015 when Aduro BioTech Inc., a leader in the development of cyclic dinucleotide compounds that target STING, signed a worldwide collaborative research, development and commercialization agreement with Novartis AG. Novartis paid Aduro a $200 million upfront payment and took an equity stake in Aduro. ***Aside from Aduro and Spring Bank, we note that at this juncture, we can only identify three other companies specifically developing STING or PRRs technologies****:* Kineta, Rigontec and Multicell Immunotherapeutics, all of which are private companies.

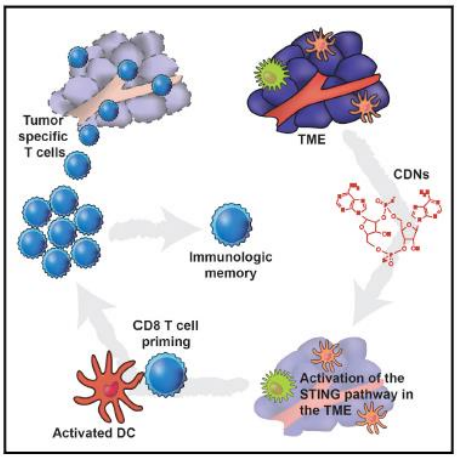
Spring Bank's researchers have found that SB 9200 will bind to the STING receptor in addition to RIG-1 and NOD-2, thus making the family of SMNH compounds potentially applicable to treating cancerous tumors. Spring Bank has conducted a number of *in vitro* proof-of-concept experiments with SB 11285, a cyclic purine dinucleotide, and has begun to present encouraging early proof-of-concept data at academic meetings, most recent of which were poster presentations at the American Association for Cancer Research (AACR) Special Conference on Tumor Immunology and Oncology held in Boston on October 20-23, 2016. For readers' information, we've attached links to these posters at the end of this report.

Exhibit 6. General Mechanism of STING in Pathogen and Tumor Immune Response

In the left panel, STING is activated by pathogenic nucleotides that leads to interferon induction and STING promotion of the initiation of phagocytosis, the process by which pathogenic cellular remains are degraded and eliminated from the cell. The right panel depicts an illustration of the interaction of STING and tumor and necrotic DNA in the tumor microenvironment. The foreign DNA debris triggers STING to activate CD8+ T-cells that prime dendritic cells.



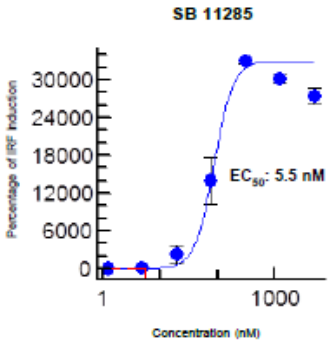
Source: Cytosolic DNA Sensors (CDSs), Nov. 2011, InvivoGen (www.invivogen.com)



Source: Corrales et al., 2015, **Cell Reports** 11, 1018–1030, May 19, 2015 http://dx.doi.org/10.1016/j.celrep.2015.04.031

Exhibit 7. Selected SB 11285 *in vitro* Studies

To the right, we have selected representative *in vitro* data from Spring Bank's Oct. 2016 corporate presentation that illustrates two key points with regard to SB 11285's activity. The left panel demonstrates SB 11285's ability to promote a high rate interferon induction, thus confirming the interferon pathway's response to SB 11285, while the right panel demonstrates SB 11285's ability to amplify immune response via mediation of interferon genes, pattern recognition receptors and checkpoint inhibitors which may assist in T-cell trafficking into the tumor microenvironment. The combination of these two mechanisms may make SB 11285 particularly attractive in an immune-oncology combination therapy component.



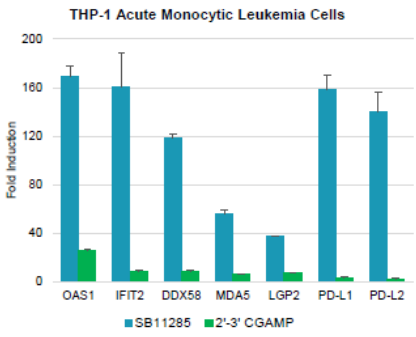
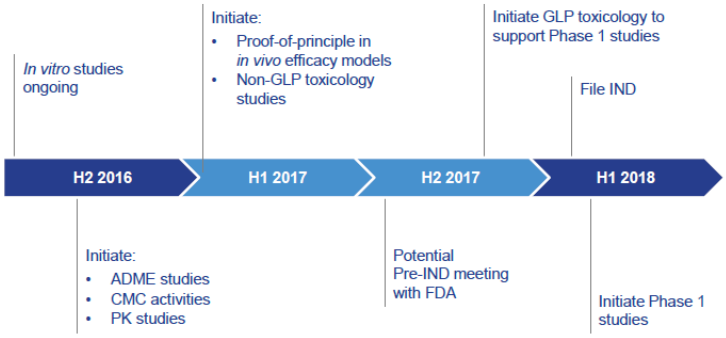


Exhibit 8. SB 11285 Development Plan



Source: Spring Bank Pharmaceuticals

Technology Background:

Viral Interaction with Cells and the Rationale behind SMNH Compounds

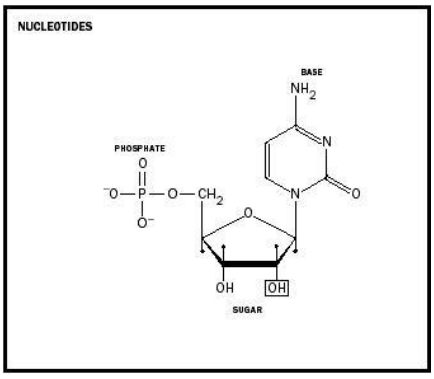
Pathogenic organisms possess nucleic acid sequences of RNA, or ribonucleic acid, and DNA, deoxyribonucleic acid, that are unfamiliar to the host. Mammalian cell immune mechanisms have become highly evolved to recognize these foreign nucleic acids in the cytoplasm as a danger signal. By activating host pattern recognition receptors (PRRs) known as cytosolic DNA sensors (CDS) that are akin to intracellular "sentries", foreign nucleic acids stimulate the innate immune response. Through several pathways, cytosolic DNA sensors activate the production of interferons (IFNs) by the induction of IFN-responsive genes and proinflammatory chemokines targeted against the invading pathogen and together contribute to mounting the innate immune response. The production of this initial set of signaling and proinflammatory proteins is critical to the body's ability to ultimately destroy the pathogenic agent. Furthermore, the innate immune system sets the stage for the adaptive immune cascade by educating host adaptive immune cells about the pathogen. The adaptive immune system is taught to recognize the pathogen through complementary sensor pathways and process its elimination. Without a properly functioning innate immune system, the "education" of the adaptive immune system is inadequate and the body is unable to clear future pathogenic assaults.

Microbial proteins targeting cytosolic sensing have been repeatedly shown to be virulence factors important for pathogenesis. In addition, because they rely on the host for their metabolism, replication and propagation by integrating in part or in whole with host genetic machinery, viruses are adept at mutation, particularly under the selective pressure (a few viral particles survive) of the typical antiviral therapy. The inherent viral capability to take over host genetic machinery makes focusing on the virus's interaction with host cellular DNA and RNA an apparent ideal target for antiviral treatment. Break the virus's ability to use the host genetic material for replication and propagation, and the infection would be cleared.

This concept led to early HIV and other antiviral drugs. Since the approval of those early HIV monotherapies, scientists have learned that viruses employ multiple mechanisms in commandeering host genetic machinery and there is also significant heterogeneity among those mechanisms, depending upon viral family, particular serotype and its genomic subtypes. Hepatitis is a perfect case in point, where each family, such as HBV, may have numerous genotypic subtypes (A, B, C, D, etc.) and these genomic subtypes employ different infection/persistence and immune system evasion mechanisms. As such, antiviral treatments directed against a single viral mechanism may not be effective against another, and partial effectiveness can quickly lead to resistance. This fact has transformed antiviral treatment from monotherapies to combination therapies such as AZT for HIV/AIDS.

In a general sense, early antiviral drugs were targeted against viral nucleotides that were largely involved with usurping the host cells genetic machinery for viral replication and proliferation. Nucleotides are subunit molecules that are linked to form the base nucleic acids of life, DNA and RNA, which are the cell's storehouse and translator of genetic information, respectively. Nucleotides may also exist outside of replicative DNA and RNA structures. Free nucleotides are among the most important ligands for a number of intracellular and extracellular proteins. Nucleic acid-protein interactions play critical roles in cell signaling and metabolism by serving as universal carriers of metabolic energy and high-energy electrons.

Exhibit 9. General structure of a Nucleotide



All nucleotides are composed of three parts: a five-carbon sugar, a phosphate, and a nitrogen-rich structure called a nitrogenous base. In the associated drawing, the sugar is ribose, which is found in ribonucleotides and RNA, or deoxyribose, which is found in deoxyribonucleotides and DNA. There are five nitrogenous bases belonging to two groups: pyrimidines (cytosine, thymine, and uracil) that are relatively small and have only one ring structure. The larger purines (adenine and guanine) are comprised of two rings. Depicted here is a pyrimidine base.

The phosphate group is bonded to the #5 carbon of the sugar, and when nucleotides are joined to form RNA or DNA, the phosphate of one nucleotide is joined to the sugar of the next nucleotide at its 3 carbon, to form the sugar-phosphate backbone of the nucleic acid. In a free nucleotide, there may be one, two, or three phosphate groups attached to the sugar, in a chain of phosphates emanating at the #5 carbon. Adenosine triphosphate (ATP) is an example of a tri-phosphate nucleotide and is critical to producing the cell's energy.

Source: http://www.biologyreference.com/Mo-Nu/Nucleotides.html

Nucleotides perform diverse biological functions as an energy pool in catalyzing di- and triphosphates, and as secondary hormonal messengers in the form of cyclic nucleotides that in turn act as coenzymes and cofactors that are critical for enzymatic functions related to metabolism and cell signaling. Analogs of nucleotides (nucleosides) that interfere with a virus's ability to control/integrate with host genetic information form the basis of most of today's antiviral therapeutics.

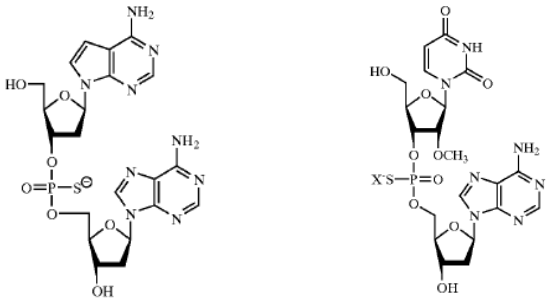
Spring Bank's Platform Technology: Nucleotide Combinatorial Target-guided Ligand Assembly

However, one of the major stumbling blocks in current antiviral drug development is the still quite limited information concerning how viruses interact structurally, mechanistically and functionally with their virus-specific molecular targets. As a result, the design of new target-specific compounds that interfere with key aspects of viral life cycles has proven to be a significant challenge. There continues to be a substantial unmet clinical need for antiviral drugs with novel viral molecular-targeting structures and unique mechanisms of action, beyond the conventional and currently approved nucleos(t)ide analogs. Furthermore, there is a need to develop nucleos(t)ides that act independently of viral genomic make-up and thus circumvent viral propensity for mutation.

Spring Bank's SMNH compounds mimic naturally occurring human nucleotides and nucleic acids in order to modulate the interaction between target nucleotides (oligonucleotides) or nucleic acids and their associated proteins that occurs in response to microbial pathogens. By optimizing target SMNH compounds, these synthetic nucleotides can be engineered to modulate in either an agonist and antagonist manner to upregulate or downregulate a targeted interaction. Thus Spring Bank's SMNH library potentially affords the Company broad therapeutic potential as to the modulation of disease pathways that remain challenging with current modalities.

Spring Bank's core technology has evolved from over 15 years of nucleic acid combinatorial chemistry discovery and experimentation. Many structural and functional proteins, viral and non-viral, are known to contain nucleotide-binding domains within specific binding sites (pockets). Nucleotides within binding pockets can act as substrates or ligands using a number of different interactions, including hydrogen bonding, hydrophobic, ionic, and van der Waals interactions. Over time, the Company's scientists established a library of more than 600 two to six nucleotide long di- and trinucleotide compounds synthesized using a combinatorial strategy and optimized to respond to these various types of intra- and extracellular interactions. Candidates were screened for potential anti-HBV replication inhibition using a standard HBV-producing cell line. Through an iterative process of synthesis, lead screening and optimization, a number of specific analogs were identified as potent inhibitors of HBV replication through analysis in *in vitro* assays.

Exhibit 10. Schematic of Variation in Spring Bank's Small Molecule Nucleic Acid Hybrid (SMNH) Structures



Examples of two different SMNH dinucleotide molecules structures. In these examples, both SMNH molecules share a similar nucleotide, but then have had different internucleotide linkers used between the sugars and different side groups attached as the second nucleotide. Spring Bank's chemistry platform uses combinations of from one to six nucleotides. In addition, these molecules are enantiomers (left or right handed) and can be made raecemers or isomers. Other topographical differences can be modified depending upon the specific interaction being targeted. Source: Spring Bank Pharmacueticals, US Patent#6,881,831

This strategy has been variously referred to as "**diversity-oriented organic synthesis for therapeutic target validation**" or "**combinatorial target-guided ligand assembly**" and its goal was to design a library of small molecules that can intercede and mimic the repertoire of interactions that occur among nucleic acids and proteins. Importantly, Spring Bank's scientists used the nucleic acid-based (NAB) scaffold (backbone) as the template to incorporate "**diversity attributes**" that could potentially target different and specific protein-protein and protein-nucleic acid interactions. The flexibility of this approach, and hence value in the technology platform, is demonstrated by the diversity of both rigid and flexible NAB scaffolds used to create a variable array of hydrogen bonding, degree of hydrophobicity, charge transfer, electrostatic state, and other noncovalent interactions. Also, by linking the individual scaffolds together, certain shape-defining motifs such as circles, pseudoknots, bulges, and stem-loops can also be incorporated into libraries. Many protein-protein interactions are shape and/or topographically-oriented dependent.

There are significant advantages to this approach. From a synthesis perspective, Spring Bank's NAB libraries can be easily assembled using well-developed solid-phase or solution-phase organic chemistry methods. By exploiting a variety of key structural features, such as modifications in phosphorothioate (an oxygen atom is replaced with a sulfur atom) and phosphoramidate (the phosphate group has OH substitutions) internucleotide connections, combinations of these modifications are employed in the creation of linear and cyclic di- or trinucleotides. Thus, Spring Bank's compounds can be engineered to provide specific metabolic stability, a key obstacle to the use of RNA interference in antiviral and other therapeutics. Phosphorothioates may also be added to potentially participate in electrostatic interactions, while the nonionic phosphoramidates can facilitate hydrophobic and hydrogen-bonding interactions with the target receptor, thereby facilitating delivery into cells.

Spring Bank's flexibility in phosphoramidite, H-phosphonate chemistry, or methylphos phoramidite nucleotide chemistry has created a disciplined structure-based drug design, that uses combinational chemistry, structural biology and phenotypic screening approaches to develop compounds with specific attractive and desirable attributes. Compounds are further optimized for high selective binding to protein targets. The extensive library provides a broad array of template modifications from which to improve or assign druggable characteristics, such as oral administration and bioavailability.

Through this discovery approach, Spring Bank's technology addresses several of the major technical challenges that have heretofore vexed antiviral oligonucleotide therapeutics, including target cell specificity, cell permeability and bioavailability issues that result in toxicity and viral escape:

- Spring Bank's nucleotides are small, less than 8mers, compared to more typical 18-30mer oligo-nucleotides, or antiviral aptamers, antisense, ribozymes or RNA interference sequences currently in development. In addition, at around 7,000 daltons in size, more standard oligonucleotides have been found to be too large to effectively pass through the pores in the intestine and traverse the cellular lipid bilayer to eventually reach intended targets. By contrast SMNH compounds are around only 700 daltons in size;

- Spring Bank's charge-modified nucleotide backbone technology overcomes very significant charge-related stability and bioavailability issues such as GI tract acid/base-related premature nucleotide degradation and suppression of cell permeation though the intestinal mucosal barrier. Charge in particular, plays a key role in cell permeation;

- Further, Spring Bank's compounds do not directly interfere with the transcription of HBV RNA as their first site of action, and thus may be less prone to causing viral mutational resistance as well as other "escape" mechanisms that can occur with specific antisense oligonucleotide sequences.

Lastly, SMNH compounds have an advantage over RNAi or other direct nucleic acid-based approaches, such as siRNA or miRNA and the like, that act by inhibiting specific protein expression through downregulation of messenger RNA. SMNH compounds act directly on proteins and therefore can be used to either upregulate or downregulate the activities of these proteins that play a role in disease processes, independently of specific genomic make-up of the virus and/or can by-pass certain types of off-targeting, such as that recently experienced by Alnylam in its Revusiran failed trial.

SB 9200 Chemistry Affords Distinct and Unique Advantages

SB 9200 is a linear alkoxycarbonyloxy dinucleotide prodrug that has been designed to be metabolized by human liver microsomes into the active form, dinucleoside phosphorothioate, without processing by cytochrome p450- mediated oxidation or conjugation. Cytochrome p450 is one of the body's primary metabolic enzymes and drug interference with the enzyme by many hydrophobic small molecule compounds is a major contributor to adverse drug interactions. SB 9200's phosphorothioate backbone contributes to both its own and the activated dinucleoside's preferential distribution to the liver where the conversion of the prodrug to active form is accomplished by liver esterases. (Coughlin, et al, **Drug Metabolism and Disposition,** May 2012.) Bypassing cytochrome p450 metabolism is a major and unique advantage of SB 9200 and the SMNH library of compounds in that these compounds are not subject to the "first pass effect" and so can be combined with other classes of drugs at a reduced potential for adverse drug-drug interactions.

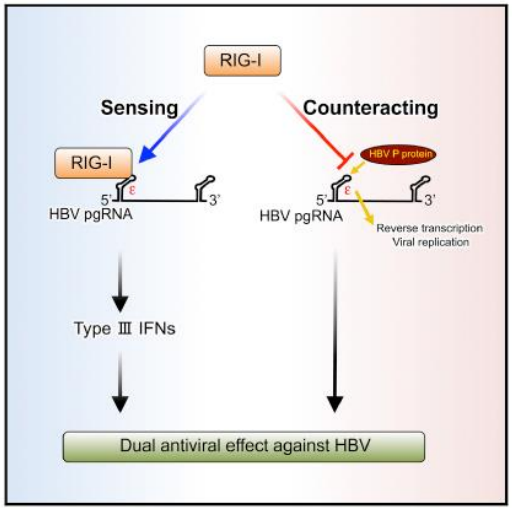
SB 9200 chemistry also avoids typical antiviral toxicity issues. Toxicity issues with exogenous interferon opened the door for the current generation of antiviral therapeutics, nucleoside analogs, that seek to interrupt viral nucleotide interaction with host genetic replication machinery at the level of transcription and replication. A major obstacle with these antiviral nucleosides in general, and with HBV nucleos(t)ides, in particular, is they primarily interact with viral polymerase in the viral transcription process. Unfortunately, in eukaryotic cells, mitochondrial DNA is replicated by DNA polymerase gamma, a catalytic enzyme very similar to the virus's own polymerase. Human polymerase gamma is critical to normal mitochondrial activity. Interference by viral polymerase targeting antiviral drugs has resulted in dose limiting toxicities that in turn can lead to viral escape and resistance. Because SB 9200 targets cytosolic DNA pattern recognition receptors, it does not interfere with liver mitochondrial function.

Unique Targeting at Immune Evasion, the Central Problem in Antiviral Treatment

Viruses in particular are adept at immune evasion by employing a variety of stealth mechanisms, including disabling normal immune response pathway activation. The interference with and inactivation of the host cell's cytosolic DNA sensors is implicated in a number of chronic viral infections. Viral genomes encode proteins that can block interferon synthesis and signaling, inhibit the function of interferon-induced antiviral proteins and produce interferon receptor decoy molecules to prevent activation of the interferon signaling pathway. Thus, by turning off interferon production and function, viruses nullify the immune response, which enables them to rapidly multiply in cells.

The activation of the IFN pathway requires the interaction (binding) of CDSs with their active sites on various proteins. These proteins can bind either directly to nucleic acids or indirectly through an alternative nucleic acid or nucleotide. The proper activation of this pathway leads to the transcription factor IFN regulatory factor 3 (IRF3) properly go through a phosphorylation mechanism in order for the cell to produce IFN. HBV genome or genomic products are actively sensed by the one or more cytosolic DNA sensors in the HBV-containing cell, including RIG-I and cGAS. Research has shown that with regard to HBV, RIG-1 and mitochondrial antiviral signaling (MAVS) expression are the primary cytosolic DNA sensors for hepatoma cells.

Exhibit 11.
RNA Sensor RIG-I Dually Functions as an Innate Sensor and Direct Antiviral Factor for HBV Virus



Highlights

- Type III IFNs are predominantly induced in human hepatocytes during HBV infection
- RIG-I senses the HBV genotype A, B, and C for the induction of type III IFNs
- The 5′-ε region of HBV pgRNA is a key element for the RIG-I-mediated recognition
- RIG-I counteracts the interaction of HBV P with pgRNA to suppress viral replication

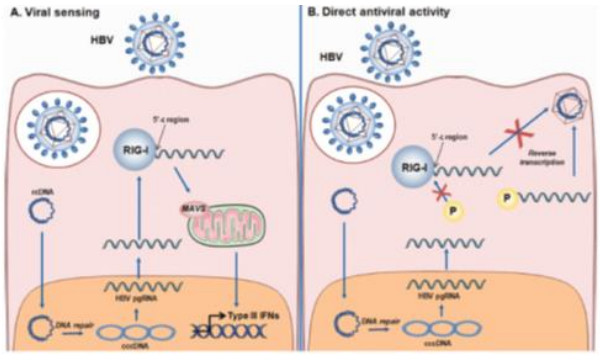


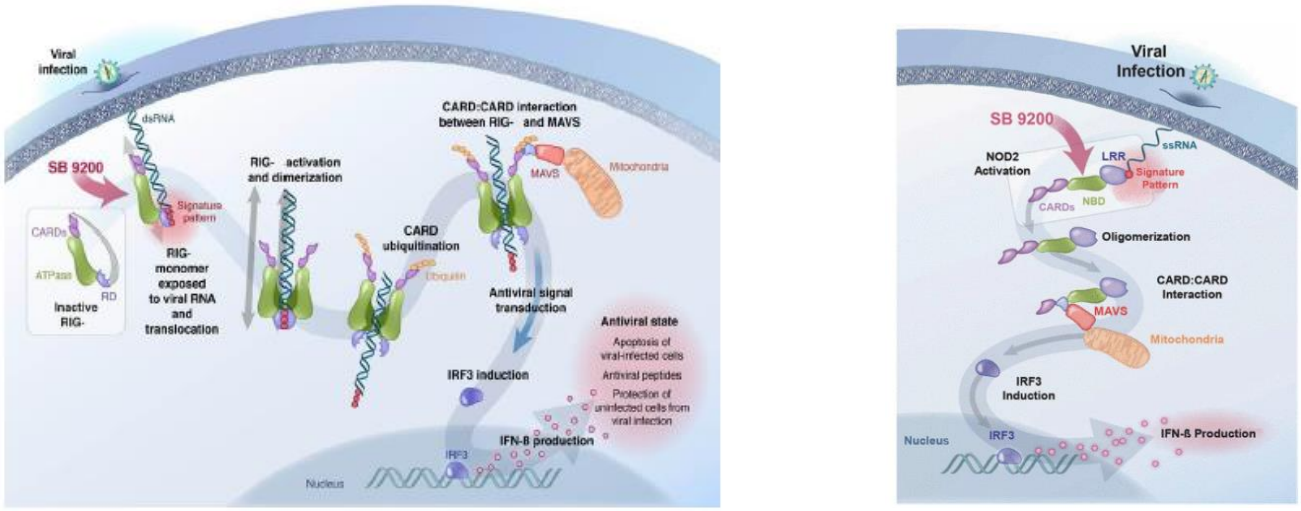
Fig. 1. Model of HBV-RIG-I interactions according to Sato et al. During HBV infection, viral genomic relaxed circular (rc)DNA is imported into the nucleus. rcDNA is then converted into a covalently closed circular (ccc)DNA that serves as a template for the transcription of viral RNA, including pgRNA. (A) HBV sensing by RIG-I. Sato et al. report that after its export into the cytoplasm, the 5′-ε region of HBV pgRNA is recognized by RIG-I. Sensing of RIG-I induces activation of type III IFN expression. (B) The direct antiviral effect of RIG-I reported by Sato et al. involves binding of RIG-I to HBV pgRNA, which inhibits the interaction between viral polymerase (P) and HBV pgRNA, resulting in impairment of reverse transcription.

Several 'sensors' of RNA, including Toll-like receptors (TLRs), the RNA helicases retinoic acid-inducible gene 1(RIG-I) and Mda5, have been shown to be essential for innate immune responses to many types of pathogens. RIG-I and nucleotide-binding oligomerization domain-2 (NOD2), another DNA sensor, are inactive in uninfected cells, but become activated when a cell is virally infected. The activation of RIG-I and NOD2 results in the production of interferon within the cells through a well-regulated signaling pathway. Viruses invade cells and are able to replicate and spread because they have evolved mechanisms to block the protective effects of antiviral signaling proteins such as RIG-I and NOD2. Source: Verrier et al **HEPATOLOGY**, Vol. 62, No. 3, 2015

RIG-I sensing of viral RNA is a crucial element of the process of the activation of antiviral innate immune response in order to limit viral replication and of triggering the cascade leading to adaptive immunity (Takeuchi and Akira, 2009). Interaction with HBV-derived pregenomic(pg) RNA, which mediates encapsidation of the pgRNA into viral capsids, triggers RIG-1 mediated type III IFN expression. Additionally, it has now been shown that RIG-1 can interact with a specific region of the HBV pgRNA that serves as the binding site of the viral polymerase P protein required for reverse transcription of the viral genome by unwinding viral RNA duplexes and translocating itself along base-paired RNA (Leung and Amarasinghe, 2012). Thus these observations suggest that RIG-I is a both a sensor of HBV-inducing antiviral IFN type III immune responses in infected hepatocytes and also a direct-acting host natural antiviral agent.

SB 9200 has been designed to bind selectively to and upregulate RIG-I and nucleotide-binding oligomerization domain-containing protein 2, or NOD2, each of which is a cytosolic sensor shown to be targets of microbial proteins. SB 9200 selectively activates these sensors ***within, and only within, infected*** cells to inhibit viral replication and enable in interferon (IFN) mediated antiviral immune responses in virus-infected cells. Additionally, the binding of SB 9200 to these sensor proteins could also sterically block the ability of the viral polymerase to access pre-genomic RNA for nucleic acid synthesis. Uniquely, SB 9200 does not interact with non-infected cells.

Exhibit 12. SB 9200 Site of Action: RIG1 (left panel), NOD-2 (right panel)



Source: Spring Bank Pharmaceuticals

Other viruses that also downregulate the RIG-I and NOD2 signaling pathway include para-influenza, respiratory syncytial virus, noroviruses, rhinoviruses, HCV and HDV. The similarity in immune evasion tactics of these viruses to that employed by HBV provides the basis for Spring Bank's development pipeline for SB 9200 and its analogs.

Technology Background:

STING (Stimulator of INterferon Genes)

The innate arm of the immune system is predominantly responsible for an initial inflammatory response to pathogens or injury via a number of soluble factors, including complement system and the chemokine/cytokine system factors and through a number of specialized cell types including mast cells, macrophages, dendritic cells (DCs), and natural killer cells. Cyclic-dinucleotide (CDN) immune stimulators have been identified that activate DCs via the cytoplasmic receptor, STING. STING is an adaptor protein residing in the cytoplasm of host mammalian cells that activates the TANK binding kinase (TBK1)—IRF3 signaling axis, resulting in the induction of IFN-β and other IRF-3 dependent gene products that strongly activate innate immunity. It is now recognized that STING is a component of the host cytosolic surveillance pathway, contributing to the sensing of foreign nuclear material inherent from infection or necrotic cells. In response, STING induces the production of IFN-β, leading to the development of an adaptive-protective pathogen-specific immune response consisting of both antigen-specific CD4+ and CD8+ T cells and antibodies. It is STING's activation of dendritic cell priming and downstream activation of CD4+ and CD8+ cells that holds promise in cancer therapy.

When viral or cellular DNA is present in mammalian cell cytoplasm, upon binding to the foreign DNA, the protein, cyclic GMP-AMP Synthase (cGAS), generates a cyclic dinucleotide that triggers dimerization of AMP and GMP to form a cyclic dinucleotide, GMP-AMP (cGAMP). STING is activated by binding to this cGAMP ligand which triggers phosphorylation of the IRF3 protein. IRF3 translocates to the nucleus to trigger transcription of inflammatory genes that then induce the production of interferon and NF- κB . Activation of STING also results in the production of cytokines and chemokines which, along with interferon, enables cells to launch a potent innate and adaptive immune response directed towards cancer cells. Like viruses, cancerous tumors can produce immuno-inhibitory molecules that prevent dendritic cells from initiating an immune response. In this manner, tumors are thought to be are able to downregulate STING and avoid the action of the immune system.

Exhibit 13. STING Innate Immune Response Signaling

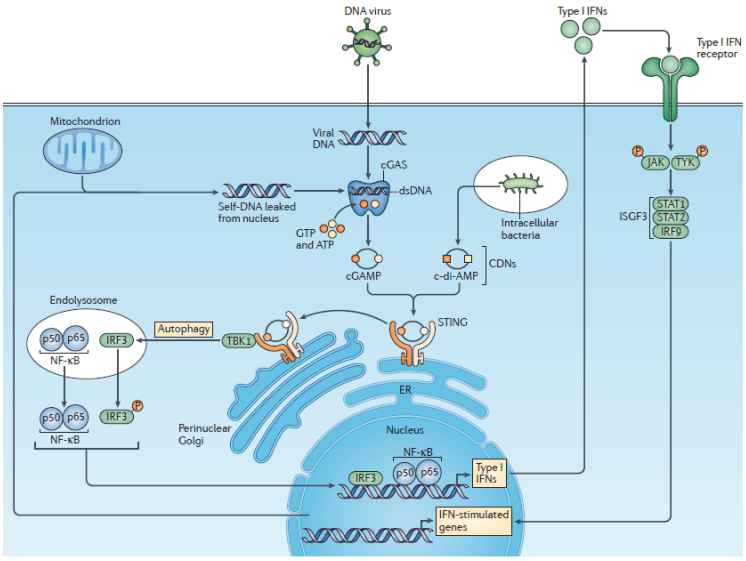
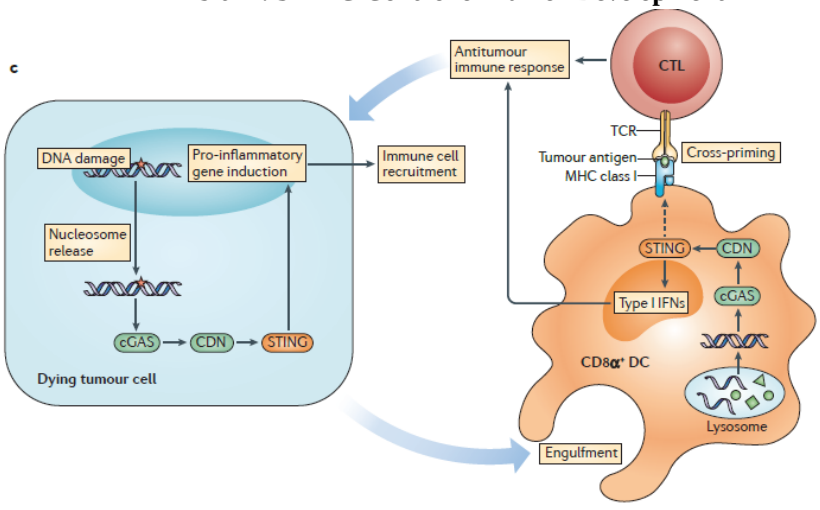


Exhibit 14. STING Control of Tumor Development



Source: Adapted from Berber et al; STING: Infection, inflammation and cancer; **Nature Reviews Immunology**; Vol.15, Dec. 2015

Spring Bank's Target Indications:

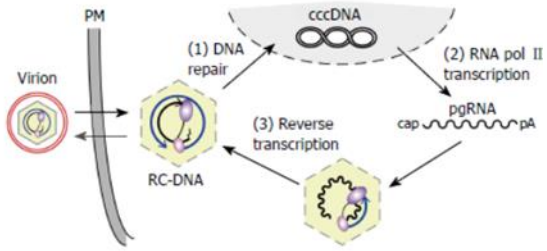
Hepatitis B (HBV)

Hepatitis B is a DNA virus belonging to the Hepadnaviridae family of viruses that infects both man and birds. It is a partially double-stranded DNA virus with several serological markers, including surface and envelope antigens, HBsAg

and HBeAg. HBV is unusual as it is one of the smallest enveloped DNA viruses to infect man, and it replicates by reverse transcription (RT) of a pre-genomic RNA (pgRNA). The biggest challenge in curing HBV infection is to eradicate the virus's ability to produce covalently closed circular DNA (cccDNA) within the host hepatocyte, which is responsible for viral persistence.

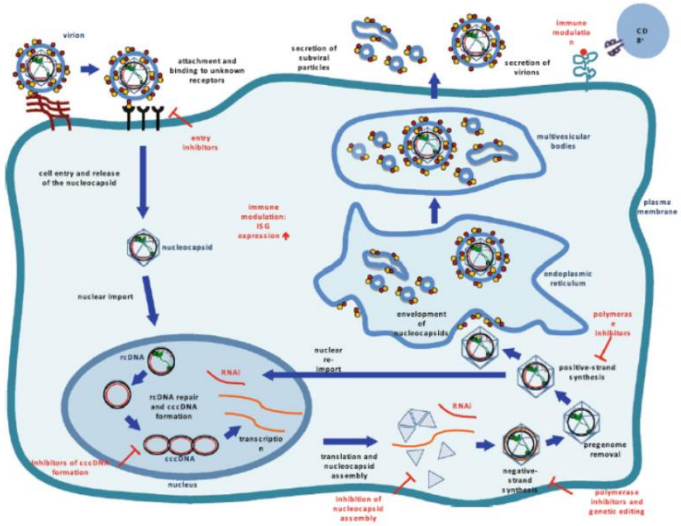
HBV is highly evolved for adaptation to differentiated hepatocytes of its host and has become extremely dependent upon the hepatocyte in a classic symbiotic fashion. Despite its very limited genomic coding capacity, HBV is able to effectively evade the immune system of the host on several levels and persist lifelong within infected hepatocytes of the host. During active replication, HBV viral DNA segments integrate with human liver cell DNA forming a reservoir of cccDNA in the nuclei of hepatocytes which can produce as many as 10^{11} virons per day. This produces enormous viral loads and a massive surplus of subviral surface antigen particles that are released into the blood of infected patients, but without actually killing the hepatocyte the virus used to self-replicate. Its unique replication process provides an enormous genetic flexibility for selection of viral mutants when selectively pressured, for example, by attack from the immune system or from antiviral therapy intervention. In addition, viral wild-type and mutated viral genomes are quite stable and can readily remain "archived" in the nucleus of the infected hepatocyte in an episomal DNA form that provides independence from cellular replication or integration within the host genome.

Exhibit15. Replication cycle of the HBV genome



Enveloped virions infect the cell, releasing RC-DNA containing nucleocapsids into the cytoplasm. RCDNA is transported to the nucleus, and repaired to form cccDNA (1). Transcription of cccDNA by RNA polymerase II (2) produces, amongst other transcripts (not shown), pgRNA. pgRNA is encapsidated, together with P protein, and reverse transcribed inside the nucleocapsid (3). (+)-DNA synthesis from the (-)-DNA template generates new RC-DNA. New cycles lead to intracellular cccDNA amplification; alternatively, the RC-DNA containing nucleocapsids are enveloped and released as virons. PM, plasma membrane.

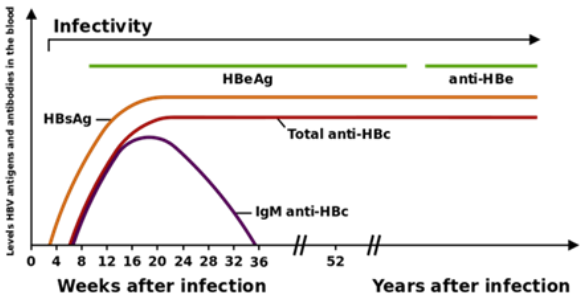
Exhibit 16. HBV Infection in the Liver Hepatocyte



Source: left panel: Adapted from: Beck, et al, Hepatitis B Virus Replication, **World J Gastroenterol.,** 2007 January 7; 13(1): p48-64
Right panel: Grimm et al., **Hepatol. Int.,** (2011) 5:644–653 HBV life cycle and novel drug targets

HBV does not structurally damage liver cells in order to replicate. Instead both liver damage and viral control depend on a complex interplay between viral replication and host immune response, thus making the virus a difficult therapeutic target. It is primarily transmitted by blood and bodily fluids and is 100X more infective than HIV. Patient outcomes are highly dependent upon age at which the virus is contracted and whether the patient is able to mount a sufficient immune response. Overall, as much as 40% of men and 15% of women with perinatally acquired HBV infection will die of liver cirrhosis or hepatocellular carcinoma. In addition to decreasing hepatic inflammation, long-term (life-long) antiviral treatment can reverse cirrhosis and reduce hepatocellular carcinoma.

Exhibit 17. HBV Infectivity Cycle



Source: Creative Commons; Chronic_HBV_v2.png

Typically, the incubation period for HBV is 90 days (range, 60–150 days). Typical signs and symptoms of HBV include malaise, fatigue, anorexia, nausea, vomiting, abdominal pain, and jaundice. In some cases skin rashes, joint pain, and arthritis may occur. In children under 5 years and immunocompromised adults, HBV infection is typically asymptomatic. Around 90% of infants infected perinatally become chronic carriers, unless vaccinated at birth. Above the age of 5, only 30%–50% of infected children will develop typical HBV symptoms. The overall case-fatality ratio of acute HBV is approximately 1%. Acute HBV progresses to chronic HBV infection in 30%–90% of people infected as infants or young children, and in about 5% of people infected during adolescence or adulthood. Chronic infection with HBV leads to chronic liver disease, including cirrhosis, liver cancer, and death.

HBV is a major public health problem worldwide, but especially in Asia and the developing world. Even though HBV is but one of the five hepatitis virus major serotypes (Hep. A, B, C, D and E), the World Health Organization (WHO) estimates *roughly 30% of the world's population shows serological evidence of current or past HBV infection*. The most recent WHO data reports approximately 240 million people worldwide are currently infected with HBV and an estimated 786,000 deaths occur each year worldwide as a result of infection. According to the CDC's *Facts for the Public*, there were approximately 19,400 new HBV infections reported in the US (2014) and about 850,000-2.2 million chronic HBV-infected people living in the US, of which as many as 70% (studies range from 47-72%) were born outside the US. This number may be under-reported as the majority of chronic HBV cases in the US are among foreigners coming to live here.

Exhibit 18. HBV Leads to Cirrhosis of the Liver and Liver Cancer

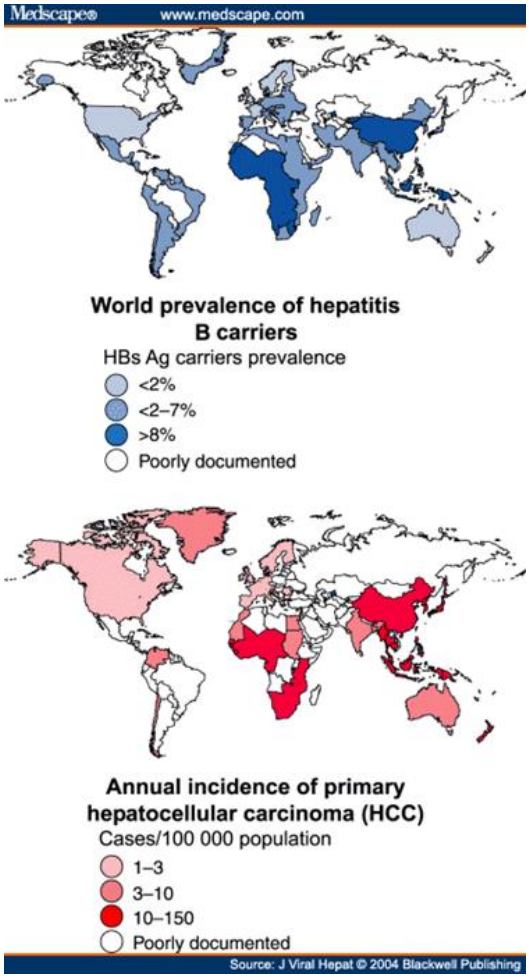
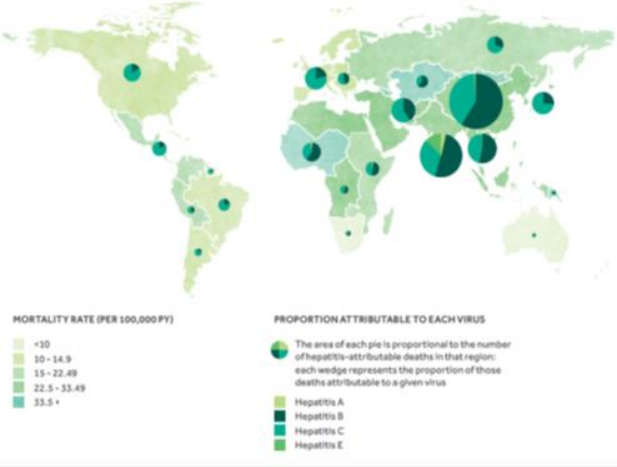
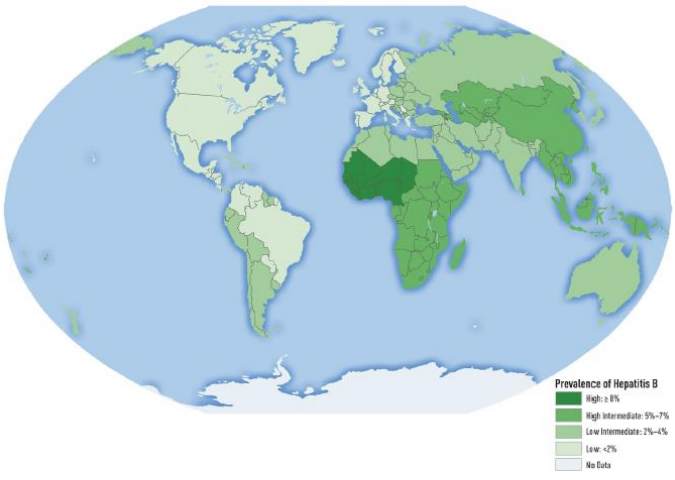


Exhibit 19. Global Distribution of Hepatitis

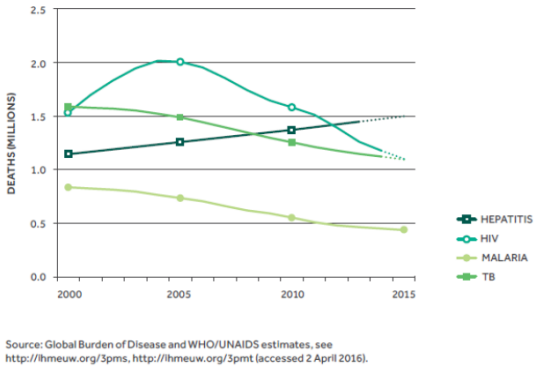
Distribution of All Hepatitis Genotypes



Prevalence of Hepatitis B

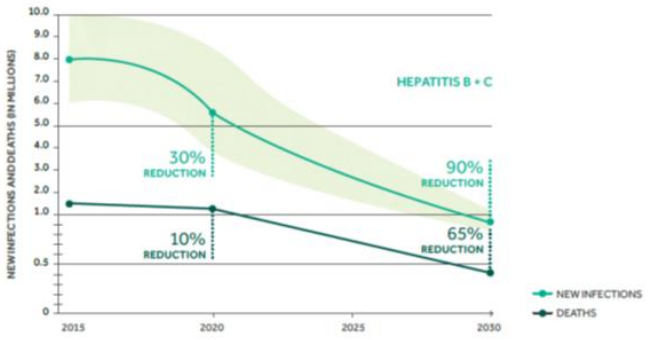


Hepatitis Deaths vs. Other Major Infections
Note that only Hepatitis has a rising death rate



Source: Global Burden of Disease and WHO/UNAIDS estimates, see http://ihmeuw.org/3pms, http://ihmeuw.org/3pmt (accessed 2 April 2016).

WHO Analysis of the Eradication of Hepatitis



Source:left panels, lower right: WHO Global Health Sector Strategy on Viral Hepatitis 2016-2021, p11, 12, 23
Source: top right panel: Ott JJ, Stevens GA, Groeger J, Wiersma ST. Global epidemiology of hepatitis B virus infection: new estimates of age-specific HBsAg seroprevalence and endemicity. **Vaccine** 2012; 30(12): 2212-2219.

Like HCV and HDV, HBV is transmitted primarily through contact with infected blood and semen. An effective vaccine has been available since 1981, and the implementation of universal vaccination in a growing number of countries has resulted in a sharp decline in prevalence. WHO's 2030 Agenda for Sustainable Development calls for combating hepatitis and in 2014, WHO set 2030 as the goal for the eradication of the virus. WHO has defined the elimination of viral hepatitis as a public health threat as achieving a 90% reduction in new chronic infections and a 65% reduction in mortality.

The major treatment strategies include interferons and nucleos(t)ide analogs that interfere with viral replication. In most patients, these agents fail to completely achieve virus eradication as they only suppress the virus during treatment without providing significant levels of HBsAg loss or clearance. As a result, patients need to take these oral antiviral drugs potentially for the rest of their lives. However, prolonged treatment with nucleos(t)ide analogs also typically leads to serious side effects in addition to promoting increasing resistance by the virus. Exogenous interferons (IFNs), the body's natural defense again infections, cannot be used for long term therapy also because of dose and treatment length limiting toxicities. Even if tolerated, the rate of durable HBV surface antigen loss is higher with interferons than with nucleos(t)ide analogs, although still only occurs in 10% or less of patients.

In the absence of a true cure for HBV, achieving HBV antigen seroconversion has become the target of a "functional" cure, whereby antiviral therapy can be terminated because the patient's own immune system takes over to eradicate the virus. Seroconversion is defined as the loss of threshold levels of HBV surface antigen presence in the patient's blood on two occasions at least 6 months apart. In the absence of a direct HBV cure, development of new therapies that can improve HBsAg clearance and lead to an eventual virological cure are most urgently needed.

Other Viral Indications Provides a Pipeline for SB 9200 and its Analogs
Because of its unique mechanism of action that allows for the reconstitution and/or potentiation of the host immune system at the host level and not at the viral level, SB 9200 may be applicable as a pan-viral immune-stimulatory agent in the treatment of other diseases caused by RNA viruses, including Hepatitis D (HDV), HIV latency and certain respiratory viral diseases such as respiratory syncytial virus (RSV) and rhinovirus (RV).

RSV
RSV is a viral disease of the very young and very old. It is the leading cause of pediatric viral respiratory tract infections and infant hospitalizations. It is also the second leading cause of death among the elderly. Premature newborns, immune-compromised infants and adults such as transplant patients and individuals with COPD or asthma, are most susceptible to RSV. RSV tends to be a seasonal virus in North America, but is ubiquitous in terms of disease exposure. By the age of 18 months, approximately 87% of children have already developed RSV-specific antibodies. Nearly all children are

exposed to the virus by two or three years of age. RSV infection typically presents as a mild upper respiratory tract illness, but in high-risk infants and young children, it can cause bronchiolitis, a lower respiratory tract infection (LRTI) characterized by wheezing, coughing, and respiratory distress. A few key US statistics from the CDC:

- 2.1 million hospital outpatient visits among children under five years old annually;
- Hospitalizations of over 57,000 children under two and about 177,000 adults over 65 years old
- Approximately 14,000 RSV-related deaths of older adults.
- Worldwide, the virus is estimated to be attributable to over one million infants and children deaths under the age of five each year (Polak et al, 2004).

Structurally RSV, a member of the paramyxovirus family of viruses, is a relative simple virus comprised of a negative sense, single-stranded, enveloped RNA encoding nine proteins. Yet, while there has been significant preclinical research and numerous clinical trial studies conducted targeting various of the RSV proteins, there still remains no FDA-approved therapies to cure RSV. Currently, supportive care, with the use of corticosteroids and inhaled beta agonists, is the most common treatment for RSV infection. A monoclonal antibody, palivizumab (**Synagis®),** marketed by Medimmune LLC., is approved for prophylactic treatment in high-risk infants. Ribavirin is approved to treat adults with RSV.

It is thought a central obstacle to an effective RSV treatment lies with the immune system. RSV has the capacity to evade the immune system, and take advantage of the immaturity of the infant's immune system or the waning immunity in the adult, thus contributing to an infected individual's inability to mount an enduring immune response. A journal article just published (Cheung et al, **PLoS**, Oct. 3 2016) gives further evidence of RSV's influence over the immune system via the disruption of the development of a protective immunity. In the article, Cheung reports that RSV infects mesenchymal stem cells (MSCs) in addition to bronchial epithelial cells and in doing so, RSV infection of MSCs alters their immune regulatory function by upregulating IFN-β and IDO (indoleamine 2, 3-dioxygnease), a key enzyme in the immune pathway. The upregulation of IDO is thought to impede normal immune cell proliferation. Lackluster immune cell proliferation may be responsible for a lack of protective RSV immunity and for the chronicity of RSV-associated lung diseases such as asthma and COPD. "*Important for survival, the regulation of IDO biosynthesis and its activities in cells of the immune system can critically alter their responses to immunological insults, such as infection, autoimmunity and cancer."* (Source: Mbongue et al, **Vaccines (Basel)** 2015 Sep; 3(3) 703-709). Links to these two journal publications are provided at the end of this report.

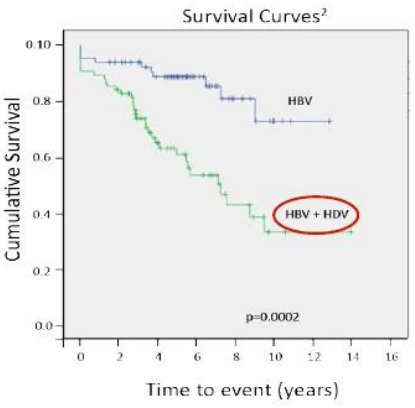
There is likely a large underdiagnosed RSV-infected population as rapid RSV diagnostics have only become available in 2013. Thus, Spring Bank sees a significant opportunity for an effective treatment, especially in pediatric and at-risk adult populations, including the immunocompromised, the elderly and those with respiratory co-morbidities such as chronic obstructive pulmonary disease and emphysema. Spring Bank has conducted preclinical experiments of SB 9200 in an RSV murine model. A SB 9200 antiviral effect was quantified by measurement in the reduction of lung inflammation in the mouse model. If Spring Bank elects to proceed with development of SB 9200 for RSV, depending upon funding, the Company would initiate a Phase II RSV challenge clinical trial in otherwise healthy adult volunteers.

Hepatitis D (HDV)

Spring Bank sees a market opportunity in HDV-infected patients, an orphan indication and subset of chronic HBV carriers. Therapies such as SB 9200, which can reduce or enhance the clearance of HBsAg, may be a necessary component for treatment of HDV co-infected HBV patients. Currently, the only treatment for HDV is long-term (12-18 months) administration of PEG-IFN-α products. However, response rates are marginal, and long-term IFN treatment is associated with significant toxicity and poor patient tolerability.

Only associated with HBV, HDV is a highly virulent form of hepatitis that leads to a greater severity of liver disease characterized by accelerated liver fibrosis, liver cancer and eventual failure than is typical for HBV. It is estimated by the WHO that approximately 15 million HBV-infected individuals worldwide are also infected with HDV.

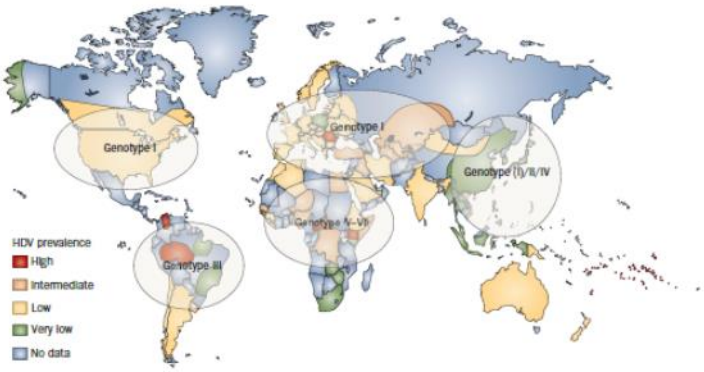
Exhibit 20. HBV/HDV Co-infection



²Serrano et al, EASL 2011.

WHO puts HDV prevalence of about 5-6% among chronic HBV carriers (WHO Hepatitis D Fact Sheet July 2016). Prevalence varies among countries greatly with China, Central Asia, Russia, Turkey, Africa and South America having evidence of HDV prevalence orders of magnitude higher than the worldwide average and in some cases, is reported as high as 60% of HBV carriers. Based on an average of estimates of approximately 1.5 million chronic HBV individuals in the United States, about 100,000 may be co-infected with HDV. As such, HDV has been designated an Orphan Drug indication by the FDA.

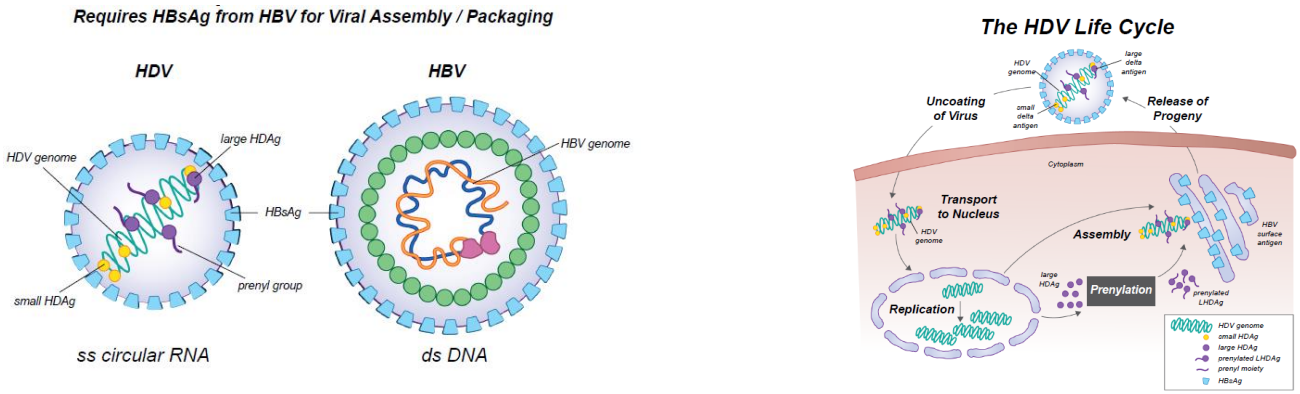
Exhibit 21 . Worldwide Prevalence of HDV



Source: www.eigerbio.com/resources/Wedemeyer-Epidemiology-Pathogenesis-Management-of-HepatitisD.pdf

HDV is the smallest single stranded RNA virus that infects man and further unusual in that it possesses a novel circular RNA genome. It shares the HBV surface antigen envelope protein (HBsAg) which is thought to provide protection for the HDV nucleocapsid antigen and the means by which HDV enters and exits the hepatocyte. At present only PEG-interferon has shown any sustained virological response. Clinical trials using nucleos(t)ide analogs have been widely attempted, but have all failed to show any meaningful virological response similar to that of HBV, and now are largely considered ineffective in HDV. Since HDV is dependent upon the HBV surface antigen and HBV pathogenesis, it is logical that SB 9200 or an SB 9200 analog may have clinical utility in HDV through similar mechanisms by which SB 9200 mediates the immune system and viral replication in HBV.

Exhibit 22. HDV/HBV Mechanism and HDV Life Cycle



Source: Overview of Hepatitis Delta, Jeffrey Glenn MD, PhD, Eiger Biopharmaceuticals HDV Analyst Day, May 2016

Disease progression is similar to HBV but comes in two forms: Acute and Superinfection. In an acute infection, there is simultaneous infection with HBV and HDV that can lead to a mild-to-severe hepatitis, but recovery is usually complete and development of chronic hepatitis D is rare (less than 5% of acute hepatitis). In a superinfection, however, HDV infects a person already chronically infected with HBV. The superinfection of HDV in chronic hepatitis B patients accelerates progression to a more severe disease in all ages in 70–90% of cases, most of whom progress to cirrhosis of the liver within 5-10 years, despite HDV's ability to naturally suppress HBV replication. The mechanism in which HDV causes more severe hepatitis and a faster progression of fibrosis than HBV alone remains unclear.

HIV Latency

HIV latency occurs when an individual no longer appears to be infected during or just after treatment, but at a later date, the virus becomes reactivated and re-exerts itself. Herpes simplex and chicken pox/shingles are two familiar viruses capable of latency. Current HIV treatments, like HBV, are effective in *suppressing* viral replication, but the HIV virus hides in stores of host T-cells, there remaining dormant until it is reactivated and overcomes the immune system. It is for this reason AIDS patients remain on their viral suppression medication and are not "cured". Research at Rockefeller University, Johns Hopkins University and other institutions is pointing to the need to "kick-start" and augment the

immune system to reach and treat these latent reservoirs of HIV-infected cells. A treatment strategy combining the elimination of HIV's invisibility to the immune system with viral nucleosides will most likely be necessary to effect the eradication of the virus and real cure.

There are currently no FDA-approved drugs for HIV latency. However, researchers are exploring a wide range of possible solutions, including anti-cancer drugs, in addition to non-traditional antivirals. HIV is thought to be able to replicate in cells by evading innate immune mechanisms involving sensory proteins such as RIG-I. Thus an immune activator will also be required to drive viral eradication. The dual action of Spring Bank's SB 9200 compound potentially may provide a combination therapy in a single drug. Under this potential scenario, a patient would likely halt current viral suppression medication, thus allowing the virus to become reactivated sufficiently to stimulate the production of RIG-I and NOD2. SB 9200 would be administered, upregulating the RIG-I and NOD2 proteins and further stimulating the innate immune system. In addition, SB 9200 could also interfere with directly with viral replication. Spring Bank intends to pursue further preclinical development for HIV latency depending upon interest from potential collaborators such as major research centers and third parties with significant expertise in HIV.

HBV Market and Competition:

Worldwide revenues for HBV market leaders, **Baraclude**® (entecavir) and **Viread®** (tenofovir) were reported at approximately $2.5 billion in 2014 (Spring Bank SEC filings). In recent market analyses, **MarketResearch** in Sept. 2015 **GlobalData** in Jan 2016 each pegged the CAGR growth rate for HBV treatments at 2.3-2.4% over the next 5-8 years in eight major geographic territories, defined as China, US, France, Germany, UK, Italy and Spain, thus implying a potential market of $3-3.5 billion in the years 2021-2024. Much of this growth is forecasted to originate from expanded treatment in China, which currently accounts for about 38% of the market and is expected to grow to better than 45% of the market. However, the actual dollar growth in the market, in our view, will be more difficult to predict as it will likely be impacted by the number and timing of new and novel treatments offset by the rapidity at which current branded drugs are converted to generics and drive treatment pricing lower and the expansion of vaccination. Currently, the average wholesale price for generic lamivudine **(Epivir**®) is in the range of $285 for a month's treatment, while branded Viread sells in the range of $1,200/mo. Pricing data is from the NIH's website, www.aidsinfo.nih.gov updated July 2016, average monthly wholesale prices provided by to the NIH by the Micromedexsolutions division of Truven Health Analytics, April 2016.

There are a number of approved HBV treatments on the market. FDA-approved treatments for patients with chronic HBV include injection pegylated interferon-a, or PEG-a, products, including **Pegasys®,** marketed by Roche (Genentech, Inc.) and **PEG-Intron** (PEG-IFN a-2b), marketed by Merck & Co., Inc., and oral antiviral agents such as the nucleoside analog **Baraclude®** (entecavir), marketed by Bristol-Myers Squibb, **Tyzeka®** (telbivudine) marketed by Novartis, **Epivir HBV®** (lamivudine) marketed by GlaxoSmithKline and the nucleotide analog **Viread®** (tenofovir), marketed by Gilead Sciences. Market-leading Viread will be coming off patent in 2017. Gilead is expected to receive FDA approval very shortly for its second generation tenofovir, TAF. GSK, under a bi-directional license agreement, markets Gilead's first generation hepatitis drug, adefovie dipivoxil as **Hepsera®** in certain Asian countries.

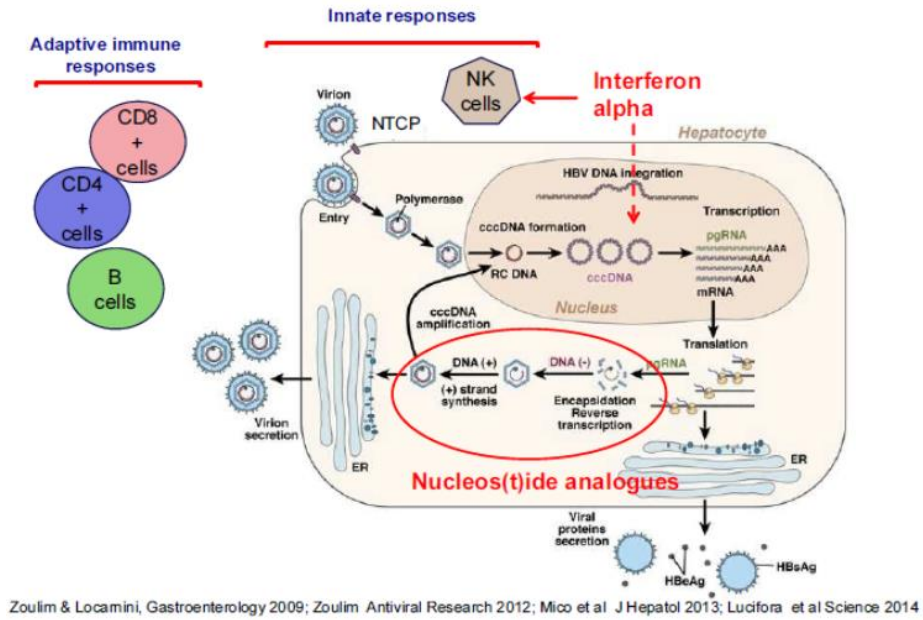
As mentioned, WHO has put into place a worldwide effort to vaccinate newborns to eventually eradicate the disease and FDA-approved vaccinations are available for children and high-risk adults that protect well against HBV. These vaccines are primarily manufactured and sold by Merck & Co., Inc. and GlaxoSmithKline Plc. However, vaccines are also marketed by Sanofi-Pasteur (**HBVAXPRO**®), Grifols USA (**Hyer HEP B™**) and Roche (Hoffman-LaRoche). The vaccines are widely available in the United States and becoming more available in the developing world in order to meet the WHO's goal of eliminating HBV by 2030. The prophylactic vaccines have limited side effects.

Companies with approved anti-HBV products	**Mechanism**
Gilead Sciences	Reverse transcription (RT) inhibitor nucleoside prodrug
GlaxoSmithKline (2 products)	HBV RT inhibitor
	Newborn HBsAg recombinant DNA vaccine (Energix-B)
Merck Sharpe Dohme (Merck) (2 products)	PEGylated interferon
	HBsAg recombinant vaccine (Recombivax-B)
Bristol-Myers Squibb Co.	HBV cccDNA RT inhibitor nucleoside

Company	Mechanism
Sanofi (Sanofi Pasteur)	HBsAg antigen recombinant DNA vaccine
Hoffman-LaRoche (Roche)	PEGylated interferon alfa 2a
Novartis AG	Thymidine β-L HBV DNA nucleoside
Grifols (Grifols Biologicals)	Human plasma derived immune globulin

Although the vaccines are effective against HBV in non-infected individuals, they do not reverse or cure the disease in people who have already contracted the virus. Several pharmaceutical and biotechnology companies are developing mono and combination drugs through therapeutic strategies incorporating nucleos(t)ides that are aimed at achieving "functional" cures in order to decrease the risk of liver damage and eventually to allow the patient to terminate antiviral treatment. These companies include Arbutus Biopharma Corp., Alnylam Pharmaceuticals, Inc., Arrowhead Pharmaceuticals, Inc., Assembly Biosciences, Inc., Gilead Sciences and Janssen (Crucell) Pharmaceuticals, Inc., to name a few. They are also exploring a variety of other mechanisms of action to reach this goal including RNAi, non-nucleotide antivirals and non-interferon immune enhancers and various combinations thereof. Another group of companies, including ContraVir, are seeking to make lower cost/better side effect profile analogs of tenofovir and other now generic nucleos(t)ides. We believe that instead of competing with certain of these therapies, SB 9200 has the potential to be used as a complementary therapy.

Exhibit 23. HBV Infection and Sites of Therapeutic Intervention



Zoulim & Locarnini, Gastroenterology 2009; Zoulim Antiviral Research 2012; Mico et al J Hepatol 2013; Lucifora et al Science 2014

In addition, there are now a number of Asian companies who are currently marketing antiviral drugs or are conducting FDA-registered clinical trials seeking to enter the HBV treatment market. These include: Green Cross Corporation, Beijing Bio-Fortune Ltd., Beijing Tiantan Biologicals Products Ltd., Biotest Pharmaceuticals Corp., Chung Kun Dang Pharmaceuticals and Ark Biosciences.

There are no FDA-approved products for Hepatitis D. Eiger Biopharmaceuticals has received Fast Track status for its drug, lonafarnib, and is in Phase II clinical trials. Alnylam is conducting preclinical development in HDV.

Intellectual Property:

Spring Bank has developed and maintains a strong globally protected intellectual property portfolio, covering 18 patent families, that combines both patent and trade-secret knowhow. Spring Bank also holds two exclusively licensed patents. These patent families include more than 65 patents and patent applications worldwide, including 10 patents and 19 patent applications in the United States and 13 patents and 28 patent applications outside the United States. Of these patents, four

are subject to a license with BioHEP Technologies Ltd., or BioHEP. The licensed families include the active ingredient in SB 9200 and methods of assembling libraries of SMNH compounds. The following are Spring Bank's issued US patents:

US Patent No.	Title
9,138,442	Compositions and methods for treating viral infections
9,040,234	Oligonucleotide analogs as therapeutic agents
8,691,787	Nucleotide and oligonucleotide prodrugs
8,404,651	Compositions and methods for treating viral infections
8,076,303	Nucleotide and oligonucleotide prodrugs
	Patent Applications
20160060287	NUCLEOTIDE AND OLIGONUCLEOTIDE PRODRUGS
20160038527	COMPOSITIONS AND METHODS FOR TREATING VIRAL INFECTIONS
20150374816	DESIGN OF SHORT OLIGONUCLEOTIDES AS VACCINE ADJUVANTS AND THERAPEUTIC AGENTS
20150329864	DESIGN OF OLIGONUCLEOTIDE ANALOGS AS THERAPEUTIC AGENTS
20140323554	NUCLEOTIDE AND OLIGONUCLEOTIDE PRODRUGS
20130197066	COMPOSITIONS AND METHODS FOR TREATING VIRAL INFECTIONS
20130078390	METHODS AND PROCESSES FOR ATTACHING COMPOUNDS TO MATRICES
20120264709	NUCLEOTIDE AND OLIGONUCLEOTIDE PRODRUGS
20120053226	DESIGN OF OLIGONUCLEOTIDE ANALOGS AS THERAPEUTIC AGENTS
20110207690	COMPOSITIONS AND METHODS FOR TREATING VIRAL INFECTIONS

Source: US Patent and Trade Office

The core SB 9200 US patents' claims cover the composition of matter of SB 9200 specifically and generically, as well as alternative pharmaceutical compositions, methods of treating HBV and the methods of formulation of SB 9200. The earliest SB 9200 US patent expires December 2026, if not extended with additional intellectual property. Spring Bank has been issued a European patent and multiple other foreign patents with similar claims covering the composition of matter of SB 9200 that expire in December 2026 in each case, without considering potential patent term extensions. Importantly, Spring Bank has been granted patents in China, Japan and Korea with claims covering the active ingredient in SB 9200, and methods of using this compound. These patents are expected to expire in 2022. Other patent applications cover methods of using SB 9200 for other viral infections such as HCV and RSV. These patent applications, if issued, will expire in 2036 without potential patent term extension.

BioHEP Technologies, Ltd. License

Spring Bank is a party to a license agreement with BioHEP Technologies Ltd. (formerly known as Micrologix Biotech, Inc.) covering patents and know-how of certain product candidates, including SB 9200 and related compounds, which include SB 9400, SB 9941 and SB 9946, for the diagnosis and/or treatment of all viral diseases and conditions. In January 2016, the Company entered into an amended and restated license agreement with BioHEP, which amended and restated a 2003 license agreement with BioHEP. Under the terms of the original license agreement, Spring Bank issued to BioHEP 1,000,000 shares of Series A preferred stock and 12,500 shares of common stock. In connection with the amendment and restated license agreement, Spring Bank issued BioHep 125,000 shares of common stock and granted BioHEP a warrant to purchase an additional 125,000 shares of common stock at a purchase price of $16.00 per share, which warrant will expire on August 1, 2018. The amended license agreement became effective on February 1, 2016.

Under the amended and restated license agreement, Spring Bank will pay BioHEP up to $3.5 million in development and regulatory milestone payments for disease(s) caused by each distinct virus for which the Company develops one of the licensed product(s). BioHEP is also eligible to receive tiered royalties in the low-to-mid single-digits on net product sales

of licensed products sold by the Spring Bank, its affiliates and sublicensees, and to receive a specified share of non-royalty sublicensing revenues, capped at a maximum aggregate of $2 million, that Spring Bank might receive from any and all sublicensees.

Financial Overview; Q3 2016 Results and Outlook:

Spring Bank is a mid-clinical stage biotech company. The Company has not generated any revenue to date and has primarily relied upon grant funding from the NIH and the sale of equity and debt to meet its financial needs. NIH grants provided approximately $6.8 million from October 2003 to April 2016. Spring Bank does not anticipate any further grant funding. The Company expects that the current cash on hand is sufficient to fund the Phase IIa HBV trial for SB 9200 and operations until the third quarter of 2017. Spring Bank intends to raise additional capital in 2017 to fund other clinical trials and pipeline development.

Spring Bank recently reported results for the quarter ending September 30, 2016. Research and development expenses were reported as $2.7 million for the 2016 period compared to $2.1 million for the 2015 period. For the nine months 2016, research and development expenses rose to $11.2 million from $4.8 million, reflective of the Company's initiation of a Phase IIa clinical trial for SB 9200. We expect that research and development expense will continue to rise moderately as the current trial progresses. Once the Company has secured additional funding, likely in 2017, we expect research and development expenses to accelerate as Spring Bank advances clinical activities for SB 9200 and brings other potential clinical candidates forward. General and administrative expenses for the September 30th quarter were reported at $1.4 million, down slightly quarter over quarter, despite the Company's now public status. Spring Bank's net loss for the 2016 period was $4.1 million and for the nine months ended September 30th, $14.9 million. Spring Bank ended the quarter with approximately $16.6 million in cash and marketable securities. The Company's accumulated deficit rose to $49.1 million.

For the time horizon of this report, we do not expect Spring Bank to generate any meaningful revenue and thus, the Company will rely primarily upon the equity markets for funding. We're pegging 2016 cash burn at about $20 million and cash burn in 2017 at about $25 million. As the Company's technology gains validation both in the academic community and among potential corporate partners such as Gilead, Arrowhead or others in the HBV arena who might seek to add an immunotherapy component to their own products, the opportunity for contract revenue and/or partnership milestone payments will increase. We believe Spring Bank will secure one or more such partnerships over the next 12-18 months.

Exhibit 24.
Spring Bank Financial Results and Estimates, Balance Sheet

			USD$ in Thousands (except for shares outstanding)						
	2014	2015	Year End 12/2016				2016E	2017E	2018E
			Q1 2016A	Q2 2016A	Q3 2016A	Q42016E			
Revenue									
Product Sales									
Contract Revenue/Milestone Payments							$0	$100	$500
Grant revenue	$738	$946	$ 280	$ 72	$0	$0	$352		
Total Revenue	$738	$946	$ 280	$ 72	**$0**	**$0**	**$352**	**$100**	**$500**
Operating expenses:									
Research and development	6,132	7,539	5,589	2,936	2,723	3,213	14,461	18,655	24,811
General and administrative	2,412	5,003	1,226	1,458	1,452	1,583	5,719	6,519	7,432
Total operating expenses	8,544	12,542	6,815	4,394	**4,175**	**4,796**	**20,180**	**25,174**	**32,243**
Loss from operations	($7,806)	(11,596)	(6,535)	(4,322)	(4,175)	(4,796)	(19,828)	(25,074)	(31,743)
Other income (expense)									
Interest income (expense)	(1,906)	32	17	21	27	5	58	35	15
Pre-tax income (loss)	**(9,712)**	**(11,564)**	**(6,518)**	**(4,301)**	**(4,148)**	**(4,791)**	**(19,770)**	**(25,039)**	**(31,728)**
Income tax expense (income)									
Net loss	(9,712)	(11,564)	(6,518)	(4,301)	(4,148)	(4,791)	(19,770)	(25,039)	(31,728)
Unrealized gain (loss) on marketable securities			(1)	4	(3)		0		
Comprehensive loss			**$ (6,519)**	**$ (4,297)**	**$ (4,151)**	**$ (4,791)**	**(19,758)**	**(25,039)**	**(31,728)**
Net loss per common share - basic and diluted	**(0.78)**	**(2.03)**	**$ (1.11)**	**$ (0.62)**	**$ (0.53)**	**$ (0.59)**	**(2.75)**	**(3.11)**	**(3.67)**
Weighted-average number of shares outstanding - basic and diluted	3,118,344	5,932,799	5,877,135	6,923,941	7,759,630	8,147,612	7,177,079	8,055,362	8,644,504

Spring Bank Pharmaceuticals Balance Sheet

USD$ in Thousands	Sep. 30, 2016	Dec. 31, 2015
ASSETS		
Current assets:		
Cash and cash equivalents	$ 5,332	$ 4,347
Marketable securities	10,313	5,335
Prepaid expenses and other current assets	1,059	313
Total current assets	**$16,704**	**$9,995**
Marketable securities		3,189
Property and equipment, net	496	427
Other assets	35	966
Total Assets	$17,235	$14,577
LIABILITIES AND STOCKHOLDERS' EQUITY		
Current liabilities:		
Accounts payable	$2,340	$2,183
Accrued expenses and other current liabilities	1,360	1,369
Total Liabilities	$3,700	$3,552
Commitments (See Note 7 of Sep. 30 10Q)		
Stockholders' equity:		
Convertible preferred stock, $0.0001 par value—authorized, no shares and 5,000,000 shares at June 30, 2016 and December 31, 2015, respectively; no shares and 1,000,000 shares issued and outstanding at June 30, 2016 and December 31, 2015, respectively		
Preferred stock, $0.0001 par value—authorized, 10,000,000 and no shares at June 30, 2016 and December 31, 2015, respectively; no shares issued or outstanding at June 30, 2016 and December 31, 2015		
Common stock, $0.0001 par value—authorized, 200,000,000 and 50,000,000 shares at June 30, 2016 and December 31, 2015, respectively; 7,767,981 and 5,796,091 shares issued and outstanding at June 30, 2016 and December 31, 2015, respectively	1	1
Additional paid-in capital	62,669	45,211
Accumulated deficit	(44,135)	(34,169)
Other comprehensive income (loss)		(18)
Total stockholders' equity	13,535	11,025
Total	**$17,235**	**$14,577**

Source: Company SEC filings, press releases, DJ estimates

Peer Group and Valuation:

We note that none of the companies we have analyzed as Spring Bank peer group members are generating revenue, nor, with the exception of Dynavax's HBV candidate, have any of these peer group companies' product candidates yet entered Phase III trials with non-traditional nucleos(t)ide candidates or other novel antiviral drugs. Therefore, we are electing to use peer group and other valuation measures, rather than the commonly use discounted cash flow method, in assessing Spring Bank's valuation. We have chosen to develop peer groups based upon infectious diseases with lead HBV or RSV programs or with nucleic acid/nucleotide-based platforms. From our analysis, we draw the primary conclusion that investors are according higher relative valuations to companies with "platforms". Having name brand partners is an important component to moving market valuations from the $100 million or so range into the several hundreds of millions, whereas clinical stage, Phase I or Phase II, seems to be less of a valuation inflection driver. Our peer group average market value as 11/4/2016 for the HBV group is $204.11 million, for the RSV companies, $244.58 million, while our nucleic acid platform companies, command an average market value of $356.01 million.

When measured against either peer group, Spring Bank is significantly undervalued considering the maturity of its technology and the fact the company has a phase II asset in the clinic. We believe investors are under-valuing the "validation" of SB 9200 as evidenced by the Company's relationships with Gilead and now Arrowhead Pharmaceuticals. As such, we consider the stock a **BUY. We further believe that the potential of the SMNH platform as an immune-modulation technology is substantially unrecognized by investors and thus, there is potential for long-term shareholder appreciation that could lead the stock to trade more in line with members of our nucleic acid platform universe.**

Exhibit 25. Peer Group Comparables

Symbol	Company Name		Price ($) 11/4/2016	Mkt Cap $MM's
	Infectious Disease-HBV	**Comment**		
GILD	Gilead Sciences	Nucleos(t)ides	72.82	96,001
ABUS	Arbutus Biopharma, Inc.	RNAi Phase IIa	3.05	167.20
ARWR	Arrowhead Pharmaceuticals	RNAi Recent partnership with Amgen-Phase IIb	5.72	347.90
ASMB	Assembly Biosciences	Core protein allosteric modifiers-IND enabling	12.72	213.60
CTRV	ContraVir Pharmaceuticals-PIIa (TDF+)	Intends to compete on price- Phase Ib/Iia	2.01	108.40
DVAX	DynaVax Technology Corp.	Immune mediation-Phase III	9.81	377.60
EIGR	Eiger Biopharma	Focus on Hep D-Phase IIa	11.45	95.68
TRGNF	TransGene SA	Immunotherapy focuses, Phase Ib	3.07	118.42
	Group Average ex GILD			**204.11**
SBPH	**Spring Bank Pharmaceuticals**	**Phase IIa**	**9.74**	**75.34**
	Nucleotide/Nucleic Acid Platform			
pvt	Rigontec	RIG-1 targeted therapeutics IND-enabling studies		
pvt	CyTuVax	Immunostimulatory vaccines-AI20 adjuvant + HBA120 HBV non responders-Phase I		
pvt	Novira Therapeutics (owned by JNJ)	Small molecule allosteric modulator of capsid -NVR 3-778 Phase Ib-Iia		
pvt	Replicor Inc.	HBV HDV/amphipathic protein-nucleic acid polymers, Phase II with NUCs, PII with PEG-I		
pvt	CaroGen Corporation (PSC)	Immunostimulation with synthetic viral antigen vaccine particles p/c		
pvt	Crucell/Janssen	Direct-acting antivirals, vaccines		
quasi-pvt	MultiCell Immunotherapeutics	Monoclonal antibodies targeting TLRs, RIG-1, RLR and MDA-5 signaling		
ALNY	Alynlam Pharmaceuticals	RNAi directed technology, Phase I HBV	37.50	3,200
ADRO	Aduro BioTech Inc.	STING Partnership with Novartis	10.88	703.50
ARWR	Arrowhead Pharmaceuticals	Broad RNAi chemstry platform	5.72	347.90
INO	Inovio	Multiantigen DNA vaccines +IL-12 immune activator Phase I	6.28	462.15
PRQR	ProQR	RNAi directed technology towards orphan diseases	4.15	96.30
PTCT	PCT Therapeutics	Small molecule nucleotide modifiers acting at RNA level	4.17	170.20
MCET	MultiCell Therapeutics	RIG-1 directed technology	NA	NA
	Group Average ex ALNY			**356.01**
	Infectious Disease -RSV			
AVIR	Aviragen Therapeutics (Biota Pharma Europe)	Oral RSVfushion inhibitor	1.27	49.85
NVAX	Novavax	Viral-lkie clones-surface protein fusion RSV-F	1.89	439.30
REGN	Regeneron Pharmaceuticals		355.83	38,100
	Group Average ex REGN			**244.58**

Source: Company websites, Clinical trials.gov, Cap IQ

Conclusion:

We believe Spring Bank is a potential leader in an emerging class of 3rd generation nucleotide/nucleic acid-based therapeutics that hold significant promise in overcoming many of the challenges inherent with direct DNA and RNA-targeted therapies. A key element to our view in this regard is the fact that the Company's technology is more mature and more deeply characterized than a number of competitors. In addition, the flexibility to control unique sites of activity of Spring Bank's nucleotides in orally-available formulations sets the Company's technology apart from others and positions the technology as a potential immune-modulating backbone that can be applied not only in infectious disease, but potentially in cancer and autoimmune disorders. In addition to the points we list below, we believe Spring Bank's management's razor focus on technology and clinical "drivers" and demonstrated execution will be instrumental in further setting the company apart from its peers.

- Novel small molecule nucleotide/nucleic acid chemistry that can address multiple billion dollar markets, as well as orphan diseases such as HDV.
- Phase II program well under way, numerous clinical events over the next 12 months
- Validation of technology established through Gilead and Arrowhead relationships
- Limited competition in pipeline indications
- Market value substantially under peers
- Proven management with track record of execution

As the Company executes on its clinical milestones and SB 11285 gains visibility, we expect SBPH shares to more properly reflect the depth and maturity of its SMNH technology, and be recognized as a nucleic acid platform company. *SG*

Appendix

Key Management

Martin Driscoll

President, Chief Executive Officer, Chairman of the Board of Directors. Mr. Driscoll joined Spring Bank as President and CEO in August 2015 and in September 2015, was appointed Chairman of the Board of Directors. From October 2010 until July 2015, he served as CEO of Asmacure Ltée a venture-backed clinical-stage biopharmaceutical company, which was acquired by a privately held Canadian life sciences company in July 2015. Prior to Asmacure, from March 2008 until July 2010, Mr. Driscoll was the Chief Executive Officer and a director of Javelin Pharmaceuticals, Inc., a publicly traded developer of acute care pain products that was acquired in July 2010 by Hospira, Inc. He serves on the board of directors of MetaStat, Inc., a publicly traded company, and Asmacure Inc., a privately held company, Mr. Driscoll has previously served on the board of directors of Javelin Pharmaceuticals, Inc. and Genta, Inc., both public companies. Mr. Driscoll holds a B.Sc. in communications from the University of Texas at Austin.

RP (Kris) Iyer, PhD.

Chief Scientific Officer, Director, and a Spring Bank founder, since the Company's inception in 2002. Prior to Spring Bank, Dr. Iyer was co-founder and VP of Discovery at Origenix Technologies, Inc., a clinical-stage biotech company, from 1998 to 2002 and was a Senior Scientist and Associate Director of the Discovery Group at Hybridon, Inc. (now known as Idera Pharmaceuticals, Inc.) from 1993-1998. Previously, Dr. Iyer was a Professor of Medicinal Chemistry at the University of Bombay, a Visiting Scientist at the University of Texas, M. D. Anderson Cancer Center and a Visiting Scientist at the Center for Biologics Evaluation and Research at FDA/NIH. Dr. Iyer received his BSc, with honors, and his MSc degrees from the University of Bombay. He received a PhD degree in Pharmaceutical Sciences from the University of the Pacific in Stockton, California and carried out postdoctoral work at the Oak Ridge National Laboratory and at Johns Hopkins University.

Nezam H. Afdhal, MD

Chief Medical Officer. Dr. Afdhal became Chief Medical Officer in November 2015 having served previously as a consultant to the Company from early 2011 to November 2015. Dr. Afdhal is a Senior Physician in Hepatology at the Beth Israel Deaconess Medical Center and has served as the hospital's Chief of Hepatology from January 2000 to December 2014. Dr. Afdhal is also currently a Professor of Medicine at Harvard Medical School. Dr. Afdhal serves on the scientific advisory board of multiple pharmaceutical companies, including Gilead Sciences, Inc., GlaxoSmithKline Plc, Bristol Myers Squibb Company and Novartis Pharmaceuticals. Dr. Afdhal received his MB Bch degree in 1981 from the Royal College of Surgeons in Ireland and did fellowship training at University College in Dublin and at Boston University School of Medicine.

Jonathan Freve, CPA,

Chief Financial Officer, Treasurer and Secretary. Mr Mr.Freve became CFO in January 2015. Prior to joining Spring Bank, Mr. Freve served as the Senior Director of Finance of Santaris Pharma A/S from March 2014 to November 2014, when it was acquired by F. Hoffmann-LaRoche Ltd. Prior to Santaris, Mr. Freve was the Controller of Brookfield Renewable Energy Partners, L.P., a renewable power generation facilities owner/operator company from April 2011 to March 2014. Mr. Freve served as Corporate Controller of Virtusa Corporation, an information technology consulting company, from October 2007 to April 2011. Mr. Freve began his career at FASB and PricewaterhouseCoopers. Mr. Freve is a certified public accountant in the Commonwealth of Massachusetts and holds a BBA in accounting from the University of Massachusetts, Amherst.

For further reading:

Spring Bank's recent AACR I/O meeting poster presentations

http://springbankpharm.com/wp-content/uploads/2013/07/Challa_AACR-Poster-1.pdf

http://springbankpharm.com/wp-content/uploads/2013/07/SZ-AACR-SB-9003-6-and-11285-1023-1.pdf

The immune system and RSV

http://journals.plos.org/plosone/article/asset?id=10.1371/journal.pone.0163709.PDF

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4586474/

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4008286/pdf/ceor-6-217.pdf

Public Companies mentioned in this report; prices as of 11/4/16

Aduro BioTech, Inc.-- ADRO-NASDAQ-$11.05-Not rated
Arbutus Biopharma Corp.--ABUS-NASDAQ-$2.95-Not rated
Alnylam Pharmaceuticals, Inc.-- ALNY-NASDAQ- $33.75-Not Rated
Arrowhead Research Corporation -- ARWR-NASDAQ-$5.68-Not rated
Assembly Biosciences, Inc.-- ASMB-NASDAQ-$12.80-Not rated
Bristol-Myers Squibb -- BMY --NYSE-$51.01Not rated
ContrVir Pharmaceuticals -- CTRV-NASDAQ-$1.97-Not rated
Eiger Biopharmaceuticals -- EIGR-NASDAQ-$10.90-Not rated
Gilead Sciences -- GILD-NASDAQ-$72.48-Not rated
GlaxoSmithKline -- GSK-NYSE-$38.63-Not rated
Merck & Co. -- MRK-NYSE-$58.82 -Not rated
Novartis AG -- NVS-NYSE--$70.25- Not rated
Roche Holdings AG --RHHVF-NASDAQ-$229.12-Not rated
Sanofi -- SNY-NYSE-$39.94-Not rated

Risk Factors

In addition to normal economic and market risk factors that impact most equities and the common risks shared by Spring Bank Pharmaceuticals Holdings with other companies in the industry, we believe an investment in SBPH involves the following risks:

- **FDA and regulatory risks** -- Spring Bank is subject to regulatory review for its ongoing research and development activities, commercial marketing approval as well as laboratory facilities, principally with the US Food and Drug Administration, but also potentially with the EMA and other international regulatory agencies if the Company undertakes clinical trials or sells its products in the future outside the US.
- **Need to defend patents, trade secrets and other intellectual property** -- At present, the Company holds a limited number of patents relating to its products, methods and manufacturing and depends in part on trade secrets. The Company may need to defend its intellectual property in the US and overseas in the future. Further, the Company currently has limited patent protection for some of its pipeline product candidates. The Company, or its licensing partners, have made various applications which may never result in effective patents, as there is already an existing array of prior art that may preclude granting of patents.
- **Dependence on Key License** -- The Company is dependent upon license agreements covering its core technology. The licensors may have the right to terminate these license agreements under certain conditions. Such termination would materially adversely affect the value of the product candidate being developed under the license agreement and may result in our having to negotiate new or reinstated licenses with less favorable terms.
- **Need to raise additional capital** -- Although the Company has historically successfully raised funds in the public markets, there can be no guarantee of such success in the future. Currently, the Company has limited cash on hand to fund ongoing research and development programs, ongoing clinical trials and product commercialization and launch activities. Until such time as cash flows from product sales surmount R&D, clinical and operational activities, the Company will need to seek additional funding. Unforeseen events including potential delays in product sales, clinical programs and regulatory approvals could require the Company to raise additional capital through the sale of equity, therefore potentially diluting current shareholders.
- **Limited stock liquidity** -- Trading volume in Spring Bank has been comparatively light compared to other stocks in its industry, and as such, news regarding Spring Bank, its target markets, partners and/or competitors could lead to significant volatility in the stock price.

- **Competitive Markets** -- The Company competes in prescription drug markets with a number of other manufacturers, marketers and service companies, many of whom represent much larger companies with substantial resources. There can be no assurance that the Company will be able to successfully launch new products into these competitive markets in the future. Further, advances in prevention and vaccination rates may limit future market potential of some of the Company's products.
- **CRO and Contract Manufacturer Risk** -- Spring Bank is pursuing a semi-virtual operating business model and is therefore reliant upon outsourced services for certain key functions, including managing clinical trials and manufacturing. Outsourcing may have associated risks as to CRO expertise in clinical trial enrollments, for project management and clinical monitoring services. Spring Bank is working with the largest of the public CROs, Quintiles (NYSE: Q) which is a relatively low risk provider given the size and experience of the CRO. In addition to CROs, Spring Bank relies of third party manufacturers for sourcing API and encapsulating the product. These manufacturers may change over time, which may add risk in terms of product availability, quality control and successfully maintaining FDA and/or EMA certifications.
- **Risks of poor manufacturing processes** -- Quality control issues and product delays may postpone ultimate production of the drug. Additionally, the company intends to work with a partner to conduct Phase 3 trials, take the product through the regulatory process and ultimately market it worldwide. The partner may lack the desire or skill to successfully steer the product(s) through the regulatory process and the partner may have other competing products.
- **Pricing risk** -- The Company could encounter several types of pricing risk. First, at the current high annual costs for antivirals, the drugs may be unaffordable for a broad segment of the population, thus reducing the market size below present expectation of potential and forecast. Price increases may attract new legislation and implement of regulations that limit drug profitability. Governments may impose additional non-price related regulation and disclosure that can increase costs for the for the Company's target indications and industry. With the intent to partner for product commercialization, the Company may not have control over the pricing decision and so revenue expectations may not be met.

Readers are referred to the Company's SEC filings for additional risk disclosures.

Price Chart:

Price target and ratings changes over the past 3 years:
Initiated – November 6, 2016– Price Target- No price target published.

Important Disclosures:

Dawson James Securities, Inc. (the "Firm") is a member of the Financial Industry Regulatory Authority ("FINRA") and the Securities Investor Protection Corporation ("SIPC").

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Ratings Definitions:

1) **Buy**: the analyst believes the price of the stock will appreciate and produce a total return of at least 20% over the next 12-18 months;
2) **Neutra**l: the analyst believes the price of the stock is fairly valued for the next 12-18 months;
3) **Sel**l: the analyst believes the price of the stock will decline by at least 20% over the next 12-18 months and should be sold.

The following chart reflects the range of current research report ratings for all companies followed by the analysts of the Firm. The chart also reflects the research report ratings relating to those companies for which the Firm has performed investment banking services.

	Company Coverage		Investment Banking	
Ratings Distribution	# of Companies	% of Total	# of Companies	% of Totals
Market Outperform (Buy)	2	22%	0	0%
Market Perform (Neutral)	1	11%	1	100%
Market Underperform (Sell)	0	0%	0	0%
Rating Suspensions*	6	67%	5	83%
Total	9	100%	6	11%

*Suspensions are ratings under review for possible change due to unusual market-moving news, and/or analyst departure/change

Analyst Certification:

The analyst(s) whose name appears on this research report certifies that 1) all of the views expressed in this report accurately reflect his (their) personal views about any and all of the subject securities or issuers discussed; and 2) no part of the research analyst's compensation was, is, or will be directly or indirectly related to the specific recommendations or views expressed by the research analyst in this research report; and 3) all Dawson James employees, including the analyst(s) responsible for preparing this research report, may be eligible to receive non-product or service specific monetary bonus compensation that is based upon various factors, including total revenues of Dawson James and its affiliates as well as a portion of the proceeds from a broad pool of investment vehicles consisting of components of the compensation generated by investment banking activities, including but not limited to shares of stock and/or warrants, which may or may not include the securities referenced in this report.