AVI BIOPHARMA INC
Form 10-K
March 13, 2012
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UNITED STATES

SECURITIES AND EXCHANGE COMMISSION

Washington, D.C. 20549

FORM 10-K

(Mark One)

x **ANNUAL REPORT PURSUANT TO SECTION 13 OR 15(d) OF THE SECURITIES EXCHANGE ACT OF 1934**
For the fiscal year ended December 31, 2011

Or

¨ **TRANSITION REPORT PURSUANT TO SECTION 13 Or 15(d) OF THE SECURITIES EXCHANGE ACT OF 1934**
For the transition period from to

Commission file number: 001-14895

AVI BioPharma, Inc.

(Exact name of registrant as specified in its charter)

Oregon	93-0797222
(State or other jurisdiction of incorporation or organization)	(I.R.S. Employer Identification Number)

3450 Monte Villa Parkway, Suite 101

Bothell, Washington	98021
(Address of principal executive offices)	(Zip Code)

Registrant s telephone number, including area code: (425) 354-5038

Securities registered pursuant to Section 12(b) of the Act:

Title of Each Class	Name of Exchange on Which Registered
Common Stock, $0.0001 par value	**The NASDAQ Stock Market LLC**
	(The NASDAQ Global Market)

Securities registered pursuant to Section 12(g) of the Act:

None

Indicate by check mark if the registrant is a well-known seasoned issuer, as defined in Rule 405 of the Securities Act. Yes ¨ No x

Indicate by check mark if the registrant is not required to file reports pursuant to Section 13 or Section 15(d) of the Act. Yes ¨ No x

Indicate by check mark whether the registrant (1) has filed all reports required to be filed by Section 13 or 15(d) of the Securities Exchange Act of 1934 during the preceding 12 months (or for such shorter period that the registrant was required to file such reports), and (2) has been subject to such filing requirements for the past 90 days. Yes x No ¨

Indicate by check mark whether the registrant has submitted electronically and posted on its corporate Website, if any, every Interactive Data File required to be submitted and posted pursuant to Rule 405 of Regulation S-T (§ 232.405 of this chapter) during the preceding 12 months (or for such shorter period that the registrant was required to submit and post such files). Yes x No ¨

Indicate by check mark if disclosure of delinquent filers pursuant to Item 405 of Regulation S-K (§ 229.405 of this chapter) is not contained herein, and will not be contained, to the best of the registrant s knowledge, in definitive proxy or information statements incorporated by reference in Part III of this Form 10-K or any amendment to this Form 10-K. x

Indicate by check mark whether the registrant is a large accelerated filer, an accelerated filer, a non-accelerated filer, or a smaller reporting company. See the definitions of large accelerated filer, accelerated filer and smaller reporting company in Rule 12b-2 of the Exchange Act.

Large accelerated filer	¨	Accelerated filer	x
Non-accelerated filer	¨ (Do not check if a smaller reporting company)	Smaller reporting company	¨

Indicate by check mark whether the registrant is a shell company (as defined in Rule 12b-2 of the Act). Yes ¨ No x

The aggregate market value of the voting and non-voting common equity held by non-affiliates of the registrant as of June 30, 2011 was approximately $192,635,782.

The number of outstanding shares of the registrant s common stock as of the close of business on February 29, 2012 was 135,743,120.

DOCUMENTS INCORPORATED BY REFERENCE

The issuer has incorporated into Part III of this Annual Report on Form 10-K, by reference, portions of its definitive Proxy Statement for its 2012 annual meeting.

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AVI BioPharma, Inc.

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PART I

Item 1. Business.

Forward-Looking Information

This Annual Report on Form 10-K, including the Management s Discussion and Analysis of Financial Condition and Results of Operation section in Item 7, and other materials accompanying this Annual Report on Form 10-K contain forward-looking statements or incorporate by reference forward-looking statements. The statements contained in this Annual Report on Form 10-K that are not purely historical are forward-looking statements within the meaning of Section 27A of the Securities Act of 1933, as amended, and Section 21E of the Securities Exchange Act of 1934, as amended. Forward-looking statements are identified by words such as believe, anticipate, expect, intend, plan, will, may, and other similar expressions. You should read these statements carefully because they discuss future expectations, contain projections of future results of operations or financial condition, or state other forward-looking information. These statements relate to our future plans, objectives, expectations, intentions and financial performance and the assumptions that underlie these statements. These forward-looking statements include, but are not limited to:

our expectations regarding the development and clinical benefits of our product candidates;

the results of our research and development efforts and the efficacy of our PMO-based chemistries and other RNA-based technology;

our expectations regarding our ability to become a leading developer and marketer of RNA-based therapeutics;

our expectations regarding the results of preclinical and clinical testing of our product candidates;

our ability to release results by the end of April 2012 from our Phase IIb clinical trial for eteplirsen and initiate a pivotal Phase III clinical trial for eteplirsen by the end of 2012;

our ability to initiate Phase I multiple ascending dose studies for AVI-7288 and AVI-6002 in the second half of 2012;

the receipt of any required approval from the U.S. Food and Drug Administration, or FDA, or other regulatory approval for our products;

the effect of regulation by FDA and other agencies;

our intention to introduce new products;

our expectations regarding the markets for our products;

acceptance of our products, if introduced, in the marketplace;

the impact of competitive products, product development, commercialization and technological difficulties;

our expectations regarding our ability to commercialize eteplirsen with a relatively small sales force, if eteplirsen is approved for commercial sale;

our expectations regarding partnering opportunities and other strategic transactions;

the extent of protection that our patents provide and our pending patent applications may provide, if patents issue from such applications, to our technologies and programs;

our plans to file additional patent applications to enhance and protect our existing intellectual property portfolio;

our ability to invalidate some or all of the claims covered by patents issued to competitors;

our estimates regarding our future revenues, research and development expenses, other expenses, payments to third parties and changes in staffing levels;

our estimate regarding how long our existing cash and cash equivalents, exclusive of receipt of future proceeds pursuant to our contracts with the U.S. government, will be sufficient to finance our operations;

our expectations about funding from the government and other sources; and

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the adequacy of funds to support our future operations and our future capital needs.

All forward-looking statements are based on information available to us on the date of this Annual Report on Form 10-K and we will not update any of the forward-looking statements after the date of this Annual Report on Form 10-K, except as required by law. Our actual results could differ materially from those discussed in this Annual Report on Form 10-K. The forward-looking statements contained in this Annual Report on Form 10-K, and other written and oral forward-looking statements made by us from time to time, are subject to certain risks and uncertainties that could cause actual results to differ materially from those anticipated in the forward-looking statements. Factors that might cause such a difference include, but are not limited to, those discussed in the following discussion and within Part I, Item 1A Risk Factors of this Annual Report on Form 10-K.

Overview

We are a biopharmaceutical company focused on the discovery and development of unique RNA-based therapeutics for the treatment of rare and infectious diseases. Applying our proprietary, highly-differentiated and innovative platform technologies, we are able to target a broad range of diseases and disorders through distinct RNA-based mechanisms of action. We are primarily focused on rapidly advancing the development of our potentially disease-modifying Duchenne muscular dystrophy drug candidates, including our lead product candidate, eteplirsen, which is currently in a Phase IIb trial. We are also focused on developing therapeutics for the treatment of infectious diseases, including our lead infectious disease programs aimed at the development of drug candidates for the Ebola and Marburg hemorrhagic fever viruses. By building our infectious disease programs funded by the U.S. government and leveraging our highly-differentiated, proprietary technology platforms, we are seeking to further develop our research and development competencies and identify additional product candidates.

Our highly-differentiated RNA-based technologies work at the most fundamental level of biology and potentially could have a meaningful impact across a broad range of human diseases and disorders. Our lead program focuses on the development of disease-modifying therapeutic candidates for Duchenne muscular dystrophy, or DMD, a rare genetic muscle-wasting disease caused by the absence of dystrophin, a protein necessary for muscle function. Currently, there are no disease-modifying therapies available for DMD. Eteplirsen is our lead therapeutic candidate for DMD. If we are successful in our development efforts, eteplirsen will address a severe unmet medical need. Data from 17 of the 19 individuals enrolled in our Phase Ib/IIa trial in the United Kingdom who were treated systemically with eteplirsen demonstrated some generation of novel dystrophin, and one participant exhibited the first ever reported increase in dystrophin positive muscle fibers to 55% of normal. Restoration of dystrophin expression and dystrophin positive fibers is believed to be critical for successful disease-modifying treatment of individuals with DMD. We initiated a Phase IIb trial for eteplirsen in August 2011 with an objective of initiating a pivotal trial by the end of 2012. We anticipate releasing results from our current Phase IIb trial by the end of April 2012.

We are also leveraging the capabilities of our RNA-based technology platforms to develop therapeutics for the treatment of infectious diseases. The U.S. Department of Defense, or DoD, has provided significant financial support for the development of therapeutics against Ebola, Marburg, Dengue and influenza viruses. In 2010, we were awarded contracts totaling more than $300 million for the research of select therapeutic candidates. We have attracted DoD s support based in part on our ability to rapidly respond to pathogenic threats by quickly identifying, manufacturing and evaluating novel therapeutic candidates, as discussed in greater detail in the section captioned Development Programs Anti-Viral Programs Influenza Program below.

The basis for our novel RNA-based therapeutics is our phosphorodiamidate-linked morpholino oligomer, or PMO, chemistries. Unlike other RNA-based therapeutics, which are often used to down-regulate gene expression, our technologies can be used to selectively up-regulate or down-regulate the production of a target protein, or direct the expression of novel proteins involved in human diseases and disorders. Further, we believe the charge-neutral nature of our PMO-based molecules may have the potential to reduce off-target effects, such

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as immune stimulatory effects often seen in alternative RNA-based technologies. We believe that our highly-differentiated, proprietary and innovative RNA-based technology platforms, based on charge neutral morpholino oligomers, may represent a significant improvement over traditional RNA-based technologies.

We were incorporated in the State of Oregon on July 22, 1980. Our executive office is located at 3450 Monte Villa Parkway, Suite 101, Bothell, Washington 98021 and our telephone number is (425) 354-5038. Our common stock trades on The NASDAQ Global Market under the symbol AVII.

This Annual Report on Form 10-K includes our trademarks and registered trademarks, including PMO*plus*®, PMO-X , AVI BioPharma®, Cytoporter® and NeuGene®. Each other trademark, trade name or service mark appearing in this Annual Report on Form 10-K belongs to its holder.

Where You Can Find Additional Information

We make available free of charge through our investor relations website, www.avibio.com, our annual reports, quarterly reports, current reports, proxy statements and all amendments to those reports as soon as reasonably practicable after such material is electronically filed or furnished with the SEC. These reports may also be obtained without charge by contacting Investor Relations, AVI BioPharma, Inc., 3450 Monte Villa Parkway, Suite 101, Bothell, Washington 98021, e-mail: investorrelations@avibio.com. Our Internet website and the information contained therein or incorporated therein are not intended to be incorporated into this Annual Report on Form 10-K. In addition, the public may read and copy any materials we file or furnish with the SEC at the SEC s Public Reference Room at 100 F Street, N.E., Washington, D.C. 20549 or may obtain information on the operation of the Public Reference Room by calling the SEC at 1-800-SEC-0330. Moreover, the SEC maintains an Internet site that contains reports, proxy and information statements, and other information regarding reports that we file or furnish electronically with them at www.sec.gov.

Objectives and Business Strategy

We believe that our highly-differentiated RNA-based technology platforms can be used to develop novel pharmaceutical products to treat a broad range of diseases and address key unmet medical needs. We intend to leverage our RNA-based technology platforms, organizational capabilities and resources to become a leading developer and marketer of RNA-based therapeutics, including for the treatment of rare and infectious diseases, with a diversified portfolio of product candidates and approved products. In pursuit of this objective, we intend to engage in the following activities:

- advancing the development of eteplirsen and our other drug candidates for the treatment of DMD to realize the product opportunities of such candidates and provide significant clinical benefits;

- successfully executing our government funded infectious disease therapeutic programs and building on and leveraging our experience with such programs to further develop our research and development capabilities and garner additional external funding; and

- leveraging our highly-differentiated, proprietary RNA-based technology platforms to identify additional product candidates and explore various strategic opportunities, including potential partnering, licensing or collaboration arrangements with industry partners.

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Development Programs

Our RNA-based drug programs are being clinically evaluated for the treatment of DMD and have also demonstrated promising anti-viral activity in infectious diseases such as Ebola, Marburg and H1N1 influenza in certain animal models. Our lead product candidates are at various stages of development summarized below.

Program	Indication	Mechanism	Chemistry	Development Stage	Developer / Collaborator
Eteplirsen	DMD (exon 51)	Exon Skipping	PMO	Phase IIb	Proprietary
AVI-6002	Ebola virus	Translation Suppression	PMO*plus*®	Phase I	Proprietary/ U.S. Government
AVI-6003*	Marburg virus	Translation Suppression	PMO*plus*®	Phase I	Proprietary/ U.S. Government
AVI-7100	H1N1 influenza virus	Translation Suppression	PMO*plus*®	Phase I	Proprietary/ U.S. Government

* As announced in February 2012, we intend to pursue development of AVI-7288, one of the two component oligomers in AVI-6003.
In the table above, under the heading Development Stage, Phase IIb indicates clinical safety and efficacy testing in a small patient population, and Phase I indicates initial clinical safety testing in healthy volunteers or a limited patient population, or trials directed toward understanding the mechanisms or metabolism of the drug. For purposes of the table, Development Stage indicates the most advanced stage of development that has been completed or is ongoing.

Duchenne Muscular Dystrophy Program

Duchenne muscular dystrophy, or DMD, is one of the most common fatal genetic disorders affecting children (primarily boys) around the world. DMD is a devastating and incurable muscle-wasting disease associated with specific mutations in the gene that codes for dystrophin, a protein that plays a key structural role in muscle fiber function. The absence of dystrophin in muscle cells leads to significant cell damage and ultimately causes muscle cell death and fibrotic replacement. The disease occurs in approximately one in every 3,500 male births worldwide. Females are rarely affected by the disorder. Initial symptoms, which usually appear between the ages of three and five, include progressive muscle weakness of the legs and pelvis, manifested as difficulty walking, running or climbing stairs, which eventually spreads to the arms, neck, and other areas. By age ten, braces may be required for walking, and many individuals require full-time use of a wheelchair before age 12. Eventually muscular degeneration progresses to the point of complete paralysis. Disease progression is also typically associated with respiratory muscle dysfunction and a corresponding difficulty in breathing, which may require ventilatory support, and cardiac muscle dysfunction which may lead to heart failure. DMD is ultimately fatal and death usually occurs before the age of 30. There is currently no disease modifying treatment or cure for DMD.

The yearly cost of care for individuals with DMD is high and increases with disease progression. Although DMD is a rare disease, it represents a substantial product opportunity due to the severity and inexorable progression of the symptoms.

Our lead program is designed to address specific gene mutations that result in DMD by forcing the genetic machinery to skip over an adjacent contiguous piece (i.e., one or more exons) of RNA and, thus, restore the ability of the cell to express a new, truncated but functional, dystrophin protein. We believe that the expression of this truncated dystrophin protein may restore, prevent or slow deterioration of muscle function, as exemplified by the less severe muscular dystrophy phenotype, called Becker muscular dystrophy.

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Eteplirsen. Eteplirsen is an antisense PMO-based therapeutic in clinical development for the treatment of individuals with DMD who have an error in the gene coding for dystrophin that can be treated by skipping exon 51. Eteplirsen targets the most frequent series of mutations that cause DMD. Eteplirsen has been granted orphan drug designation in the United States and European Union. See Government Regulation Orphan Drug Designation and Exclusivity for additional information.

In October 2010, we announced results from the most recently completed clinical trial of eteplirsen, AVI Study 28. Data from this study were published in *The Lancet* in July 2011. AVI Study 28 was a Phase Ib/IIa open label, dose-ranging, clinical trial assessing the safety, tolerability, pharmacokinetics and exploratory efficacy of eteplirsen in ambulatory individuals with DMD. Participants in AVI Study 28 were between the ages of five and 15 with errors in the gene coding for dystrophin, which were amenable to treatment by skipping exon 51. Participants were dosed once per week for 12 weeks. A total of 19 participants were enrolled and these individuals were assigned to one of six dose cohorts of 0.5, 1.0, 2.0, 4.0, 10.0 or 20.0 mg/kg. Of the 19 participants enrolled, 18 received at least ten of the 12 doses planned in this trial. After completion of dosing, participants were followed for an additional 14 weeks. Muscle biopsies were taken before treatment and 17 participants had a second biopsy at week 14, two weeks after administration of the final dose. The primary objective of the trial was to assess the safety of eteplirsen at these doses over the 26-week duration of the trial. Secondary trial objectives included assessment of plasma pharmacokinetics, urinary elimination and exploratory endpoints evaluating biological activity and clinical performance. This trial was conducted by investigators in the United Kingdom at the University College London Institute of Child Health / Great Ormond Street Hospital in London and at the Royal Victoria Infirmary in Newcastle-Upon-Tyne. Based on the AVI Study 28 results, we determined that:

- eteplirsen was well-tolerated in all participants;
- no drug-related serious adverse events or severe adverse events were detected, except that one participant exhibited deteriorating cardiac function, which was considered probably disease related;
- overall, adverse events were characteristic of the pediatric patient population and the underlying disease, with headache, upper-respiratory tract infection, back pain, rhinitis and myalgia being the most common. These adverse events were mostly mild or moderate in intensity, not dose-related, and none were considered probably or definitely related to eteplirsen;
- eteplirsen induced exon 51-skipping in all cohorts and new dystrophin protein expression in a significant, dose dependent (p=0.0203), but variable, manner in participants from cohort 3 (dose of 2.0 mg/kg) onwards;
- seven participants responded to treatment, in whom mean dystrophin fluorescence intensity increased from 8.9% to 16.4% of normal control after treatment (p=0·0287);
- the three participants with the greatest biochemical responses to treatment had 21%, 15%, and 55% dystrophin-positive fibers after treatment and these findings were confirmed with western blot, which showed an increase after treatment of protein levels from 2% to 18%, from 0.9% to 17%, and from 0% to 7.7% of normal muscle, respectively;
- new dystrophin expression was correctly localized in the membrane of muscle cells and was accompanied by restoration of the dystrophin-associated glycoprotein complex, or DGC, a protein complex, and neuronal nitrous oxide synthetase, or nNOS, both necessary for the proper function of muscle cells;
- reductions in key inflammatory markers, including reduced presence of inflammatory cells found in tissues, potentially suggest a favorable alteration in the underlying degenerative disease process;

no immune response to newly made dystrophin was detected; and

there was general stability in exploratory markers of participant clinical performance, including cardiac, pulmonary and muscle functional assessments.

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We initiated a Phase IIb trial for eteplirsen in August 2011, AVI 4658-us-201, or Study 201, at Nationwide Children s Hospital in Columbus, Ohio. This is a randomized, double-blind, placebo-controlled study to assess the efficacy, safety, tolerability and pharmacokinetics of eteplirsen administered intravenously in two different doses over 24 weeks for the treatment of ambulant boys with DMD. Exploratory clinical measures of ambulation, muscle function and strength will also be captured and evaluated during the course of the trial. Study 201 is fully enrolled with 12 participants and muscle biopsies of all participants were performed prior to initiation of treatment. The 12 participants with a genotypically-confirmed appropriate genetic mutation were randomized into one of three treatment groups with four participants in each group. The first treatment group received a weekly intravenous administration of eteplirsen at a dose of 50.0 mg/kg. The second treatment group received a weekly intravenous administration of eteplirsen at a dose of 30.0 mg/kg. The third and final treatment group received a weekly administration of placebo. Participants receiving the 50.0 mg/kg dose received a second biopsy at 12 weeks after initiation of treatment, and participants receiving the 30.0 mg/kg dose received a second biopsy at 24 weeks after initiation of treatment. We anticipate releasing results from this trial by the end of April 2012 and intend to initiate a pivotal trial by the end of 2012.

All participants were enrolled in an open-label extension study (AVI 4658-us-202) following the completion of Study 201 and all participants, including those from the placebo group in Study 201, will receive either 30.0 mg/kg or 50.0 mg/kg for the duration of the extension study. Clinical efficacy measures similar to what was obtained in Study 201 will be collected.

Pan-Exon Strategy. In addition to our lead product candidate, eteplirsen, we are actively pursuing development of a product candidate that skips exon 45 through an IND-enabling collaboration. We are also finalizing the terms of a second IND-enabling collaboration for the development of a product candidate that skips exon 50. The active and proposed collaborations and our eteplirsen program are part of our larger pan-exon strategy for the development of drug candidates to address the most prevalent exon deletions in the DMD population. Because the majority of DMD patients have exon deletions that cluster together, a small number of exon-skipping therapies will potentially be disease-modifying for a relatively large percentage of DMD patients. Approximately 83% of the total DMD population is potentially treatable with exon-skipping therapeutics. Of this 83%, exon 51 skipping is applicable to the largest sub-group, equal to approximately 16%, and skipping of exons 50 and 45 is applicable to approximately 5% and 10%, respectively.

Anti-Viral Programs

We are implementing our RNA-based technology platforms in our anti-viral programs for the development of therapeutics to treat viruses, such as Ebola, Marburg and influenza. Our arrangement with DoD supporting the development of our Ebola and Marburg virus drug candidates provides funding for all clinical and licensure activities necessary to obtain approval of a New Drug Application, or NDA, by the U.S. Food and Drug Administration, or FDA, if DoD exercises all of its options under the arrangement. Under a prior arrangement, DoD similarly provided funding to advance the development of our H1N1 influenza drug candidate through an Investigational New Drug, or IND, application with the FDA and to preclinically evaluate its therapeutic potential against H5N1 (avian flu), Tamiflu® resistant H1N1 (pandemic flu) and H3N2 (seasonal flu). Without continued government support of these programs we may be unable to continue our development efforts. Future funding is subject to availability of budgeted funds from DoD or potentially the Department of Health and Human Services, or DHHS. For example, the period of performance for our June 2010 H1N1 influenza contract expired in June 2011 and our subsequent submissions to a DoD request for proposal, or RFP, for funding of the full clinical development of our influenza drug candidate, AVI-7100, were unsuccessful. Currently, we have paused our clinical development efforts on AVI-7100 and are exploring funding opportunities or partnerships with DHHS and industry collaborators to advance its development.

In the periods presented, substantially all of our revenues were derived from research and development contracts with and grants from the U.S. government. As of December 31, 2011, we had substantially completed

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all of our contracts with the U.S. government except for the July 2010 agreement for the development of therapeutics against Ebola and Marburg. Pursuant to this agreement, as of December 31, 2011, we are currently entitled to receive up to $126.5 million of which $52.7 million has been recognized as revenue. In addition, if the U.S. government elects to exercise all its options under the agreement, an additional $161.5 million in funding is available. For a more detailed description of our contracts with the U.S. government, see Management s Discussion and Analysis of Financial Condition and Results of Operation U.S. Government Contracts below and Note 7 U.S. Government Contracts of the financial statements included elsewhere in this Annual Report on Form 10-K.

Hemorrhagic Fever Virus Programs. Our anti-viral therapeutic programs use our translation suppression technology and apply our proprietary PMO*plus*® chemistry backbone, an advanced generation of our base PMO chemistry backbone that selectively introduces positive backbone charges to improve selective interaction between the drug and its target. Our translation suppressing technology is based on Translation Suppressing Oligomers, or TSOs, which are PMO-based compounds that stop or suppress the translation of a specific protein by binding to their specific target sequence in mRNA. We are pursuing development and regulatory approval of our Ebola and Marburg hemorrhagic fever virus product candidates under the FDA s Animal Rule. The Animal Rule provides that under certain circumstances, where it is unethical or not feasible to conduct human efficacy studies, the FDA may grant marketing approval based on adequate and well-controlled animal studies when the results of those studies establish that the drug or biological product is reasonably likely to produce clinical benefit in humans. Demonstration of the product s safety in humans is still required. See Government Regulation Animal Rule for additional information. Our lead product candidate against the Ebola virus infection is AVI-6002. For Marburg virus infection, our lead product candidate has been AVI-6003. In February 2012, we announced that we received approval from the FDA to remove one of the two oligomers composing AVI-6003 and proceed with a single oligomer approach, AVI-7288, given that efficacy in non-human primates has been demonstrated to be attributable to this single oligomer. We are exploring the feasibility of alternate routes of administration of our Ebola and Marburg drug candidates, and at DoD s invitation, we are developing a proposal to be submitted for a study to demonstrate feasibility of the intramuscular route.

Ebola virus. AVI-6002, which is a combination of AVI-7537 and AVI-7539, is designed for post-exposure prophylaxis after documented or suspected exposure to the Ebola virus. The hemorrhagic fever caused by the Ebola virus is severe and often fatal in humans. The disease was first recognized in 1976 and is one of two members of a family of RNA viruses called Filoviridae. The disease is generally understood to be endemic to parts of Africa. The Ebola virus is classified as a Category A bioterrorism agent by the Centers for Disease Control and Prevention, or CDC, and was determined to be a material threat to national security by the Secretary of Homeland Security in 2006. Onset of illness from Ebola virus is abrupt and symptoms include fever, headache, muscle ache, vomiting and stomach pain. Internal and external bleeding may also be observed in some individuals. There are currently no treatments for Ebola virus infection beyond supportive care and the mortality rate is very high. We are currently evaluating the feasibility of developing AVI-7537 as a single agent for the post-exposure prophylaxis after documented or suspected exposure to Ebola virus.

Marburg virus. AVI-6003, which is a combination of AVI-7287 and AVI-7288, is designed for post-exposure prophylaxis after documented or suspected exposure to Marburg virus. Marburg hemorrhagic fever is another severe and often fatal disease in humans that was first recognized in 1967. It is also caused by an RNA virus of the Filoviridae family and is understood to be endemic to Africa. The Marburg virus is classified as a Category A bioterrorism agent by the CDC and was determined to be a material threat to national security by the Secretary of Homeland Security in 2006. Onset of the disease is often sudden and the symptoms include fever, chills, nausea, vomiting, chest pain and diarrhea. Increasingly severe symptoms may also include massive hemorrhaging and multiple organ dysfunction. There are currently no treatments for Marburg virus infection beyond supportive care and the mortality rate is even higher than that of Ebola infection. In February 2012, we announced that we received approval from the FDA to proceed with AVI-7288 as a single agent against Marburg virus infection. Studies conducted to date have shown that efficacy in non-human primates could be attributed to

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AVI-7288, while AVI-7287 did not appear to contribute to efficacy. We intend to proceed with dosing AVI-7288 in the Phase I multiple ascending dose studies described below and in non-human primate studies to continue to evaluate efficacy.

Primates infected with Ebola virus and treated with AVI-6002 achieved 80% survival and primates infected with Marburg virus and treated with AVI-6003 achieved 100% survival, compared to universal lethality in both control groups. In addition to survival, primates treated with AVI-6002 and AVI-6003 demonstrated decreases in levels of viremia, in harmful inflammatory indicators and in virus induced liver damage. Additional data have also demonstrated that the surviving animals were resistant to viral infection after subsequent injection with the virus. Further studies are planned to evaluate the rapidity of onset of disease to determine the window of opportunity for effective therapy post-viral exposure.

In February 2012, we announced positive safety results from all six cohorts of our Phase I single ascending dose trials of AVI-6002 and AVI-6003. For each group, safety, clinical laboratory and renal biomarker results through five days after treatment were reviewed by an independent Data and Safety Monitoring Board, or DSMB, which issued recommendations for both studies to progress as planned to multiple ascending dose studies after no safety concerns were identified. The Phase I single ascending dose trials were designed to characterize the safety, tolerability and pharmacokinetics of each therapeutic candidate in healthy adult volunteers. In the two studies, a total of 60 healthy human subjects (five per group) were enrolled into six sequential dose groups (0.01, 0.1, 1.0, 3.0, 6.0 or 9.0 mg/kg). Within each group, four subjects received the indicated dose of the therapeutic and one subject received placebo. Final, unblinded safety and pharmacokinetic results for all subjects will be available upon full completion of the analyses. We anticipate initiating the Phase I multiple ascending dose studies in the second half of 2012, which are planned to characterize the safety, tolerability and pharmacokinetics of multiple doses of AVI-6002 and AVI-7288 in healthy adult volunteers. The randomized, double-blind placebo controlled studies will be overseen by the DSMB, who will review safety and clinical laboratory data after each dose cohort prior to enrolling the next higher dose cohort.

Influenza Program.

Our anti-viral therapeutic programs are also focused on the development of our product candidates designed to treat pandemic influenza viruses. AVI-7100 is our lead product candidate for the treatment of influenza and employs our PMO*plus*® technology. In June 2010, we were awarded a contract under DoD s Transformational Medical Technologies, or TMT, program, which funded our activities to develop AVI-7100 as a medical countermeasure against the pandemic H1N1 influenza virus. The period of performance for this contract ended in June 2011. See Management s Discussion and Analysis of Financial Condition and Results of Operations U.S. Government Contracts for additional information.

Symptoms of H1N1 influenza include fever, cough, runny nose, headache, chills and fatigue. Many people infected with H1N1 also have respiratory symptoms without a fever. Severe illness and deaths have also occurred. The CDC estimated that between April 2009 and April 2010 there were up to 89 million cases of H1N1 infection in the United States. The CDC also estimated that there were up to 403,000 H1N1-related hospitalizations in the U.S. during the same time period.

The TMT program established a contract with us to conduct a rapid response exercise against a real-world emerging threat like the pandemic H1N1 virus. The intent of the exercise was to demonstrate our capability to efficiently respond to a real-world emerging viral threat by rapidly designing and producing multiple therapeutic candidates and evaluating preclinical efficacy. Initially the exercise involved identifying target sequences against H1N1, designing several drug candidates utilizing proprietary derivatives of our PMO chemistry, and then manufacturing the candidates in sufficient quantity for limited preclinical testing. We successfully accomplished these steps in approximately one week, demonstrating our ability to rapidly respond to a real-world viral threat utilizing our RNA-based technology platforms.

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Subsequently, we evaluated the preclinical activity of AVI-7100 and found that it showed a favorable safety profile in ferrets, rats and monkeys. In separate ferret studies, AVI-7100 demonstrated activity as a potentiator of Tamiflu and activity towards preventing transmission of Tamiflu-resistant H1N1.

In June 2011, we initiated dosing of AVI-7100 via intravenous infusion in single-ascending doses in up to 48 healthy adult volunteers. The first dose cohort in this Phase I, randomized, double-blind, placebo-controlled study was completed and received a favorable review from the DSMB to proceed to the next dose escalation. Currently, we have paused our clinical development efforts on AVI-7100 and are exploring funding opportunities or partnerships to advance its development.

Discovery Stage Program Overview

Our PMO-chemistries are highly-differentiated from other RNA technologies, including antisense, siRNA and RNAi. Unlike these technologies, which are often used for down-regulation of gene expression, ours can be used to selectively up-regulate or down-regulate the expression of proteins involved in human diseases and disorders, or direct the production of novel proteins with clinically relevant properties.

In addition to our pan-exon strategy for DMD, our preclinical research efforts are focused on the creation of product candidates for the treatment of other neuromuscular, infectious and rare diseases.

AVI Chemistry Technology

Our core chemistry is based on phosphorodiamidate-linked morpholino oligomers, or PMOs. PMOs are synthetic molecules based on a fundamental redesign of the natural nucleic acid structure of DNA and RNA. PMOs bind to complementary sequences of RNA by standard Watson-Crick nucleic acid base-pairing and control gene expression by steric blockade of targeted RNA. Structurally, the key difference between PMOs and naturally occurring DNA and RNA is that while PMOs, like DNA and RNA, have nucleic acid bases, those bases are bound to synthetic morpholine rings instead of deoxyribose (in DNA) or ribose (in RNA) rings, and they are linked through phosphorodiamidate groups instead of phosphate groups. Replacement of anionic phosphates with the charge-neutral phosphorodiamidate groups eliminates ionization in the usual physiological pH range, thus PMOs in organisms or cells are uncharged molecules. Because of these modifications, PMOs are especially resistant to degradation by plasma and intracellular enzymes. Unlike some other RNA-based technologies, including siRNAs and other types of antisense, PMOs rely on steric blocking rather than cellular enzymatic activity for their biological effects. In this way, PMOs operate fundamentally differently from other well-known RNA-based technologies.

We have developed three new PMO-based chemistry platforms in addition to our original PMO-based technology. We believe that the novel, favorable characteristics intrinsic in these new platforms will allow for the development of drug candidates with superior delivery specificity, therapeutic windows and drug-like properties.

PPMO. The first of these novel chemistries is based on peptide conjugated PMOs, or PPMOs, in which cellular uptake of the PMO component, as well as its potency and specificity of tissue targeting, may be significantly enhanced.

PMOplus®. The second of these chemistries, PMO*plus®*, includes the addition of selectively introduced positive charges to the PMO backbone. We believe that while PMO*plus®* has potentially broad therapeutic applications, it has thus far shown to be particularly effective in increasing the potency of PMO-based oligomers.

PMO-X . The third of these chemistries, PMO-X , involves novel, selective, and proprietary backbone chemistry modifications. We believe PMO-X may provide enhanced in vivo potency for our drug candidates, as well as greater flexibility in modulation of their tissue targeting, cellular delivery and uptake.

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We intend to continue to support our internal research and development efforts in order to advance our proprietary chemistries and to develop new analogues that may provide additional benefits in key characteristics of drug performance.

AVI Mechanisms

Humans have far fewer genes than the number of unique proteins expressed in the human proteome. The genetic information stored in human DNA is not contiguous. Short DNA stretches, called exons that code for fragments of the protein are separated by long non-coding pieces of DNA called introns. During processing of precursor or pre-mRNA, which is copied from the DNA template, introns are removed and exons spliced together to create the mature mRNA, from which a functional protein can be made. Pre-mRNA copied from a gene can be spliced through alternative paths, such that different exons are combined, creating multiple mRNAs and, hence, generate multiple proteins from a single gene.

Our PMO-based molecules are designed to sterically block the access of cellular machinery to pre-mRNA and mRNA without degrading the RNA. Through this selective targeting, two distinct biologic mechanisms of action can be initiated: (1) modulation of pre-mRNA splicing (also commonly described as splice switching, exon skipping or directed alternative splicing) and (2) inhibition of mRNA translation (also commonly described as translation suppression). Through these mechanisms, steric-blocking oligonucleotides can repair defective RNA, up or down-regulate the production of selected proteins, or produce novel or remodeled proteins.

Material Agreements and Strategic Alliances

We believe that our RNA-based technology could be broadly applicable for the potential development of pharmaceutical products in many therapeutic areas. To further exploit our core technology, we have and may continue to enter into research, development or commercialization alliances with universities, hospitals, independent research centers, non-profit organizations and pharmaceutical and biotechnology companies for specific molecular targets or selected disease indications. We may also selectively pursue opportunities to access certain intellectual property rights that complement our internal portfolio through license agreements or other arrangements.

U.S. Department of Defense Agreements

We currently have contracts with the U.S. Department of Defense, or DoD, and its agencies funding our programs. For a more detailed description of our contracts with the U.S. government, see Management s Discussion and Analysis of Financial Condition and Results of Operation U.S. Government Contracts below and Note 7 U.S. Government Contracts of the financial statements included elsewhere in this Annual Report on Form 10-K.

University of Western Australia

In November 2008, we entered into an exclusive license with the University of Western Australia, or UWA, for certain patents and technical information relating to the use of certain antisense sequences for the treatment of DMD. The license grants us specific rights to the treatment of DMD by inducing the skipping of certain exons. Unless earlier terminated in accordance with the terms of the agreement, such agreement will expire on the expiration date of the last to expire patent within the patents licensed to us under the agreement. Our clinical candidate, eteplirsen, falls under the scope of this agreement. Any future drug candidates developed for the treatment of DMD by exon skipping may or may not fall under the scope of this agreement.

Under the agreement, we are required to meet certain performance diligence obligations related to development and commercialization of products developed under license. We believe we are currently in compliance with these obligations. We made an initial upfront payment to UWA on execution of the license. We

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may be required to make additional payments to UWA of up to $150,000 based on successful achievement of certain regulatory-related milestones and also may be required to pay royalties ranging from a fraction of a percent to the low single digits on net sales of products covered by issued patents licensed from UWA during the term of the agreement. As of December 31, 2011, we have made milestone payments to UWA totaling $10,000, but have not made, and are not under any current obligation to make, any royalty payments to UWA until a product candidate is approved for commercial sale. We believe, however, that a milestone payment obligation of $15,000 to UWA may be triggered in 2012 upon initiation of our Phase III pivotal trial for eteplirsen.

Strategic Alliances

Isis Ercole Agreement

In May 2003, Ercole Biotechnology, Inc., or Ercole, and Isis Pharmaceuticals, or Isis, entered into a collaboration and license agreement related to RNA splicing. In March 2008, we acquired all of the stock of Ercole in exchange for 5,811,721 shares of our common stock, which was valued at approximately $8.4 million, and the assumption of approximately $1.8 million in liabilities of Ercole. We also issued warrants to purchase our common stock (also classified as equity), which were valued at $437,000, in exchange for certain outstanding warrants issued by Ercole. In connection with the March 2008 acquisition, we assumed Ercole s obligations under the Isis agreement. This agreement contains several cross-licenses between the parties granting each party certain exclusive and nonexclusive rights under a selected set of the other parties patents and patent applications for the research, development, and commercialization of antisense therapeutics using RNA splicing with respect to certain gene targets.

Subject to the satisfaction of certain milestones triggering the obligation to make any such payments, we may be obligated to make milestone payments to Isis of up to $23.4 million in the aggregate for each product developed under a licensed patent under this agreement.

As of December 31, 2011, we have not made, and are not under any current obligation to make, any such milestone payments, as the conditions triggering any such milestone payment obligations have not been satisfied. The range of percentage royalty payments required to be made by us under the terms of this agreement is from a fraction of a percent to mid single digits. We believe that our DMD, Ebola, Marburg and influenza programs will not fall under the scope of this agreement and therefore will not be subject to milestone or royalty obligations under its provisions.

Subject to the satisfaction of certain milestones triggering the obligation to make any such payments, Isis may be obligated to make milestone payments to us of up to $21.1 million in the aggregate for each product developed under a licensed patent under this agreement. As of December 31, 2011, Isis has not made, and is not under any current obligation to make, any such milestone payments, as the conditions triggering any such milestone payment obligations have not been satisfied. The percentage royalty payments required to be made by Isis under the terms of this agreement is a fraction of a percent. As to any product commercialized under the agreement, the agreement will terminate on the expiration date of the last to expire licensed patent covering such product. Research collaboration activity defined in the agreement expired in 2006.

Charley s Fund Agreement

In October 2007, Charley s Fund, Inc., or Charley s Fund, a nonprofit organization that funds drug development and discovery initiatives specific to DMD, awarded us a $2.45 million research grant and, in May 2009, the grant authorization was increased to a total of $5.0 million. Pursuant to the related sponsored research agreement, the grant was provided to support the development of product candidates related to exon 50 skipping using our proprietary exon skipping technologies. As of December 31, 2011, Charley s Fund has made payments of approximately $3.4 million to us. Revenue associated with this research and development arrangement is recognized based on the proportional performance method, using the payment received method. To date, we have

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recognized $60,000 as revenue, but did not recognize any revenue for the years ended December 31, 2011, 2010 and 2009. We do not expect to receive any incremental funding under the grant and have deferred $3.3 million of previous receipts which are anticipated to be recognized as revenue once we complete the remaining milestones and they are agreed to by Charley s Fund.

Under the terms of the sponsored research agreement, as amended, if we and any of our strategic partners elect to discontinue the development and commercialization of any product containing any molecular candidate arising or derived from the research sponsored by Charley s Fund for reasons other than safety or efficacy, we must grant to Charley s Fund an exclusive, royalty-bearing, fully-paid, worldwide license, with right of sublicense, to any such product. Depending on whether and when Charley s Fund obtains a license to any such product, percentage royalty payments on net sales required to be made by Charley s Fund to us under the terms of the sponsored research agreement, as amended, would be in the mid single digits. Under the terms of the sponsored research agreement, as amended, if we are able to successfully commercialize any molecular candidate arising or derived from the research sponsored by Charley s Fund either through sales of products or through licensing or partnership arrangements with a third party that include rights for such third party to sell, distribute, promote or market such products or the underlying intellectual property, then we are obligated to repay the research funds paid to us by Charley s Fund, up to an amount equal to the total amount of funds provided by Charley s Fund to us. In connection with this repayment obligation, we agreed that we would pay a mid range single-digit percentage royalty on net sales of products containing any molecular candidate arising or derived from the research sponsored by Charley s Fund and a mid-teens amount of any upfront cash and/or milestone payments received from a licensing or partnership arrangement with a third party with respect to such products (in each case, up to an amount equal to the total amount of funds provided by Charley s Fund to us). This agreement will terminate by its own terms at the completion of the research being sponsored by Charley s Fund. The AVI technology upon which the agreement is based is covered by certain patents, the last of which expires following the termination of the agreement.

Previously, we noted unexpected toxicology findings in the kidney as part of our series of preclinical studies for AVI-5038, our PPMO-based candidate designed for the treatment of individuals with DMD who have an error in the gene coding for dystrophin that can be treated by skipping exon 50. We have conducted additional preclinical studies and have not alleviated the toxicity problem. Pursuant to the terms of our agreement with Charley s Fund, the receipt of additional funds is tied to the satisfaction of certain clinical milestones. Because of the toxicity issues with AVI-5038, satisfaction of the additional milestones under the agreement is unlikely and we do not expect to receive any additional funds from Charley s Fund. We are currently evaluating alternatives regarding the development of AVI-5038, but in parallel, PMO-based therapeutics, which lack the conjugated peptide, are being considered for further development options.

Manufacturing

We believe we have developed proprietary manufacturing techniques that allow synthesis and purification of our product candidates to support clinical development. We have entered into certain manufacturing and supply arrangements with third party suppliers which will in part utilize these techniques to support continued development of certain of our product candidates. We have additionally contracted with several suppliers of commercial active pharmaceutical ingredients, or APIs, to develop, scale-up the manufacturing process, and ultimately manufacture our products to support commercialization. We do not have, and do not intend to establish in the near term, any of our own internal manufacturing capability to support our product candidates.

For our Ebola and Marburg hemorrhagic fever virus development programs, we have entered into supply agreements with two multinational manufacturing firms for the production of the API for Ebola and Marburg therapeutics. Due to their technical expertise and the sophistication of their manufacturing facilities, we are also considering the same two multinational manufacturing firms for the scale-up of the API in our DMD program. There is a limited number of companies that can produce PMO in the quantities and with the quality and purity

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that we require for our development efforts. If we are required to seek alternative supply arrangements, the resulting delays and potential inability to find a suitable replacement could materially and adversely impact our business.

We also have supply arrangements with several preferred manufacturing firms for the production of the custom raw materials required for PMO production. We believe there are several contract manufacturers capable of manufacturing these materials, and as our products advance, more suppliers might become necessary; however, establishing a relationship with alternative suppliers can be a lengthy process and might cause delays in our development efforts and could materially and adversely impact our business.

Manufacturers and suppliers of product candidates are subject to the FDA s current Good Manufacturing Practices, or cGMP, requirements, and other rules and regulations prescribed by foreign regulatory authorities. We depend on our third party suppliers and manufacturers for continued compliance with cGMP requirements and applicable foreign standards.

Sales and Marketing Strategy

We have not obtained regulatory approval for any of our product candidates and thus have not yet established a commercial organization or distribution capabilities. Due to the rare nature of DMD and the lack of disease-modifying treatments, patients suffering from DMD, together with their physicians, often have a high degree of organization and are well informed, which may simplify the identification of a target population for eteplirsen, our lead product candidate, if it is approved. We believe that, if approved for commercial sale, it will be possible to commercialize eteplirsen with a relatively small specialty sales force that calls on the physicians, foundations and other patient-advocacy groups focused on DMD. Our current expectation is to commercialize eteplirsen ourselves in the United States and plan to recruit a sales force and take other steps to establish the necessary commercial infrastructure at such time as we believe that eteplirsen is approaching marketing approval. However, we may also consider entering into arrangements with other pharmaceutical or biotechnology companies for the marketing and sale of our products either globally or on a country by country basis.

Patents and Proprietary Rights

Our success depends in part upon our ability to protect our core technology and intellectual property. To accomplish this, we rely on a combination of intellectual property rights, including patents, trade secrets, copyrights and trademarks, and contractual protections.

We seek appropriate patent protection for our proprietary technologies by filing patent applications in the United States and other countries. As of February 29, 2012, we owned or controlled approximately 276 U.S. and corresponding foreign patents and 191 U.S. and corresponding foreign patent applications. We intend to protect our proprietary technology with additional filings as appropriate.

Our patents and patent applications are directed to our product candidates as well as to our RNA-based technology platforms. Although we believe our patents and patent applications provide us with a competitive advantage, the patent positions of biotechnology and pharmaceutical companies can be uncertain and involve complex legal and factual questions. We and our collaborators may not be able to develop patentable products or processes or obtain patents from pending patent applications. Even if patent claims are allowed, the claims may not issue, or in the event of issuance, may not be sufficient to protect the technology owned by or licensed to us or our collaborators. For example, our competitor Prosensa has rights to European Patent No. EP 1619249. We opposed this patent in the Opposition Division of the European Patent Office, or the Opposition Division, and in November 2011, we announced that, although we succeeded in invalidating some of the patent s claims, the Opposition Division maintained in amended form certain claims of this patent relating to the treatment of DMD by skipping dystrophin exons 51 and 46. We and Prosensa both have the right to appeal this decision; however, pending final resolution of this matter and any appeal thereof, the patent at issue may provide the basis for

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Prosensa or other parties that have rights to such patent to assert that our drug eteplirsen infringes on such patent. A final resolution of this opposition proceeding may take a number of years and the outcome cannot be predicted or determined as of the date of this report. We are also aware of certain claims that have issued to Prosensa in Japan that may provide the basis for Prosensa or other parties that have rights to these claims to assert that our drug eteplirsen infringes on such claims. We believe we have a basis to invalidate some or all of these claims and are evaluating the potential initiation of invalidation proceedings. Because we have not yet initiated an invalidation proceeding in Japan, the outcome and timing of such proceeding cannot be predicted or determined as of the date of this report. If we are unsuccessful in invalidating other of Prosensa s claims or if previously invalidated claims are restored on appeal, our ability to commercialize both eteplirsen and other therapeutic candidates for our pan-exon strategy could be materially impaired. We are also aware of certain claims that Prosensa has rights to in the United States that may provide the basis for Prosensa or other parties that have rights to these claims to assert that our drug eteplirsen infringes on such claims. We believe we have valid defenses to any such allegations or a basis to invalidate some or all of these claims and do not believe that Prosensa s patent seriously harms our ability to develop and commercialize our products; however, we cannot be certain of this. The DMD patent landscape is continually evolving and multiple parties, both commercial entities and academic institutions, may have rights to claims that could provide these parties a basis to assert that our product candidates infringe on these claims. Similarly, we may be able to assert that certain activities engaged in by these parties infringe on our patent rights. There has been, and we believe that there will continue to be, significant litigation in the biopharmaceutical and pharmaceutical industries regarding patent and other intellectual property rights. We also cannot be certain that other third parties will not assert patent infringement in the future with respect to any of our development programs.

The Company has adopted a Code of Business Conduct and Ethics that is designed to promote the highest standards of ethical conduct by the Company’s and the Bank’s directors, executive officers and employees. The Code of Business Conduct and Ethics has been posted on the Bank’s website, www.thebankofglenburnie.com.

Communications with the Board

The Board of Directors has not established a formal process for stockholders to send communications to the Board. Due to the infrequency of stockholder communications to the Board, the Board does not believe that a formal process is necessary. Furthermore, all of the Company’s Board members are residents of the communities served by the Bank and where most of the Company’s stockholders reside, and therefore are accessible to the great majority of the Company’s stockholders.

SECURITIES OWNERSHIP OF MANAGEMENT

The following table sets forth information with respect to the beneficial ownership of the shares of Common Stock as of the Record Date by (i) each executive officer of the Company named in the Summary Compensation Table included elsewhere in this Proxy Statement, (ii) each current director and each nominee for election as a director and (iii) all directors and executive officers of the Company as a group.

Name	Amount And Nature of Beneficial Ownership [1]	Percent of Class
F. William Kuethe, Jr.	58,630[2]	2.38%
Thomas Clocker	9,785[3]	0.40%
William N. Scherer, Sr.	13,881[4]	0.56%
Karen B. Thorwarth	1,886	0.08%

John E. Demyan	234,419	9.50%
F. W. Kuethe, III	131,203[5]	5.32%
Mary Lou Wilcox	1,844	0.07%
Michael G. Livingston	2,337[6]	0.09%
Norman E. Harrison	720	0.03%
Shirley E. Boyer	18,669[7]	0.76%
Charles Lynch, Jr.	19,623[8]	0.80%
Edward L. Maddox	5,598[9]	0.23%
All directors, nominees and executive officers as a group (13 persons)	500,948	20.31%

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(1) Rounded to nearest whole share. For the definition of "beneficial ownership," see footnote (1) to the table in the section entitled "Voting Securities and Principal Holders Thereof." Unless otherwise noted, ownership is direct and the named individual has sole voting and investment power.

(2) Includes 24,161 shares held jointly with others and 26,300 shares held by The Kuethe Family Trust, of which he and his spouse are trustees.

(3) Includes 7,686 shares as to which he shares voting and investment power.

(4) Includes 12,820 shares as to which he shares voting and investment power.

(5) See footnote (2) to the table in the section entitled "Voting Securities and Principal Holders Thereof".

(6) Includes 2,217 shares to which he shares voting and investment power.

(7) Includes 16,907 shares as to which she shares voting and investment power.

(8) Includes 6,567 shares held for the benefit of two minor children and 1,456 shares held by Mrs. Lynch. Each disclaims beneficial ownership to the shares owned individually by the other.

(9) Includes 1,257 shares as to which he shares voting and investment power.

REPORT OF THE COMPENSATION COMMITTEE

The Bank's Employee Compensation and Benefits Committee acts as the compensation committee for the Company and determines the appropriateness of compensation levels pertaining to the officers of the Bank, other than the Chief Executive Officer and the other executive officers of the Bank (which are determined by the full Board of Directors based upon data provided by, and recommendations of, the Committee, and must be approved by a majority of the independent directors). The overall goal of the Committee is the establishment of compensation policies designed to attract, motivate, reward and retain qualified employees who will execute the Company's strategic goals and thereby increase the value created for shareholders.

The Committee and Board review annually the compensation of the executive officers to insure that the Bank's compensation programs are related to the Bank's financial performance and consistent generally with banks of comparable size in the area. The Committee and the Board of Directors establish the compensation paid to executive officers based upon the individual performance of the executive officer and the overall performance of the Bank. In assessing the performance of an individual executive officer, the Committee considers the executive officer's historical performance, degree of responsibility, level of experience, length of service, contribution to the performance of the Company and commitment to meeting strategic goals. With respect to the salary of the Company's Chief Executive Officer, Mr. Kuethe has voluntarily limited his base salary to its current level.

Bonuses are discretionary and are generally granted to executive officers based on the extent to which the Company achieves annual performance objectives, as established by the Board of Directors. Such performance objectives include dividend growth, asset growth and performance and earnings performance.

In addition, executive officers are entitled to participate in the employee benefits offered to all employees of the Bank.

EMPLOYEE COMPENSATION AND BENEFITS COMMITTEE

Shirley E. Boyer	Thomas Clocker
F. William Kuethe, Jr.	William N. Scherer, Sr.
John E. Demyan	Karen B. Thorwarth
F. W. Kuethe, III	Michael G. Livingston

EXECUTIVE COMPENSATION

Summary Compensation Table

The following table sets forth information regarding the cash and noncash compensation awarded to or earned during the past three fiscal years by the Company’s Chief Executive Officer and by each executive officer whose salary and bonus earned in fiscal year 2005 exceeded $100,000 for services rendered in all capacities to the Company and its subsidiaries.

Name and Principal Position	Year	Annual Compensation Salary	Bonus	Other Annual Compensation	All Other Compensation
F. William Kuethe, Jr.	2005	$ 90,000	$15,000	$ —	$28,857[1]
President and Chief	2004	83,846	20,000	$ —	32,100[1]
Executive Officer	2003	80,000	20,000	$ —	23,389[1]
Michael Livingston	2005	$127,692	$20,000	$ —	$31,646[2]
Executive Vice President, Chief	2004	114,616	$17,500	$ —	17,944[2]
Operating Officer and Deputy Chief Executive Officer	2003	$94,643	$13,500	$ —	15,382[2]

(1) Mr. Kuethe's "Other Compensation" for 2005 consisted of $12,000 in directors' fees, $4,451 as a 5% employer contribution and $6,892 as a Company profit sharing contribution to The Bank of Glen Burnie 401(K) Profit Sharing Plan, and $5,514 representing the dollar value to Mr. Kuethe of the premiums on a term life insurance policy for his benefit; for 2004 consisted of $12,000 in directors' fees, $5,105 as a 5% employer contribution and $8,745 as a Company profit sharing contribution to The Bank of Glen Burnie 401(K) Profit Sharing Plan, and $6,250 representing the dollar value to Mr. Kuethe of the premiums on a term life insurance policy for his benefit; and for 2003 consisted of $9,600 in directors' fees, $4,533 as a 5% employer contribution and $8,193 as a Company profit sharing contribution to The Bank of Glen Burnie 401(K) Profit Sharing Plan, and $1,063 representing the dollar value to Mr. Kuethe of the premiums on a term life insurance policy for his benefit.

(2) Mr. Livingston became Executive Vice President and Deputy Chief Executive Officer in August 2004 and became a Director on January 1, 2005. Mr. Livingston's "Other Compensation" for 2005 consisted of $12,000 in directors' fees, $7,489 as a 5% employer contribution and $11,597 as a Company profit sharing contribution to The Bank of Glen Burnie 401(K) Profit Sharing Plan, and $560 representing the dollar value to Mr. Livingston of the premiums on a term life insurance policy for his benefit; for 2004 consisted of $6,428 as a 5% employer contribution and $11,013 as a Company profit sharing contribution to The Bank of Glen Burnie 401(K) Profit Sharing Plan, and $503 representing the dollar value to Mr. Livingston of the premiums on a term life insurance policy for his benefit; and for 2003 consisted of $5,416 as a 5% employer contribution and $9,789 as a Company profit sharing contribution to The Bank of Glen Burnie 401(K) Profit Sharing Plan, and $177 representing the dollar value to Mr. Livingston of the premiums on a term life insurance policy for his benefit.

Change in Control Severance Plan

In August 2001, the Board of Directors of the Company and the Bank approved amendments to the Company's and the Bank's Change-in-Control Severance Plan, to include the named executive officers in the Plan's coverage. Under the terms of the Plan, in the event the executive voluntarily terminates his employment within two years following a change in control, or in the event the Executive's employment is terminated by the Bank (or its successor) for any reason, other than cause, within two years following a change in control, the executive is entitled to receive an amount equal to the aggregate present value of 2.99 times the executive's average annual taxable compensation from the Bank and the Company for the prior five complete years (or the number of years during which the executive was employed by the Bank, if less). The payment will be made either in a lump sum or in installments, at the option of the executive.

Transactions with Management

All currently outstanding loans to directors and executive officers were made in the ordinary course of business of the Bank and on substantially the same terms, including interest rates and collateral, as those prevailing at the time for comparable transactions with other persons and did not involve more than the normal risk of collectibility or present other unfavorable features.

Compensation Committee Interlocks and Insider Participation

As stated above (See "Corporate Governance - Employee Compensation and Benefits Committee"), the Employee Compensation and Benefits Committee determines the compensation levels for the officers of the Bank, other than the Chief Executive Officer and other executive officers of the Bank which are determined by the full Board of Directors and must be approved by a majority of the independent directors. F. William Kuethe, Jr., President and Chief Executive Officer of the Company and the Bank, and Michael Livingston, Executive Vice President and Chief Operating Officer of the Company and the Bank, serve on the Board and on the Committee. No executive officer of the Company or the Bank serves or has served as a member of the compensation committee of another entity, one of whose executive officers serves on the Employee Compensation and Benefits Committee of the Bank. No executive officer of the Company or the Bank serves or has served as a director of another entity, one of whose executive

officers serves on the Committee.

PERFORMANCE GRAPH

The following graph compares the cumulative total return on the Common Stock during the five years ended December 31, 2005 with that of a broad market index (Nasdaq Composite), and a peer group consisting of publicly traded Maryland, Virginia and District of Columbia commercial banks with total assets between $200 million and $500 million ("Peer Group"). The Peer Group is comprised of Alliance Bankshares Corporation, Benchmark Bankshares, BOE Financial Services of Virginia, Inc., Central Virginia Bankshares, Inc., Fauquier Bankshares, Inc., First National Corporation, James Monroe Bancorp, Inc., Millennium Bankshares Corporation, Monarch Bank, Shore Financial Corporation, Abigail Adams National Bancorp, Inc., and Carrollton Bancorp. The graph assumes $100 was invested on December 31, 2000 in the Common Stock and in each of the indices and assumes reinvestment of dividends.

Total Return Analysis

	12/31/2000	12/31/2001	12/31/2002	12/31/2003	12/31/2004	12/31/2005
Glen Burnie Bancorp	**$ 100.00**	**$ 170.89**	**$ 221.39**	**$ 384.98**	**$ 373.44**	**$ 337.84**
Peer Group	**$ 100.00**	**$ 126.16**	**$ 149.59**	**$ 216.66**	**$ 239.03**	**$ 234.78**
Nasdaq Composite	**$ 100.00**	**$ 79.21**	**$ 54.46**	**$ 82.12**	**$ 89.65**	**$ 91.54**

Source: Zacks Investment Research.

REPORT OF THE AUDIT COMMITTEE

The Audit Committee has reviewed and discussed with management the annual audited financial statements of the Company and its subsidiaries.

The Audit Committee has discussed with Trice Geary & Myers LLC, the independent auditors for the Company for 2005, the matters required to be discussed by Statement on Auditing Standards 61. The Audit Committee has received the written disclosures and the letter from the independent auditors required by Independent Standards Board Standard No. 1 and has discussed with the independent auditors the independent auditors' independence.

Based on the foregoing review and discussions, the Audit Committee recommended to the Company’s Board of Directors that the audited financial statements be included in the Company’s Annual Report on Form 10-K for the year 2005 for filing with the Securities and Exchange Commission.

AUDIT COMMITTEE

William N. Scherer, Sr., Chairman
Shirley E. Boyer
Thomas Clocker
Norman E. Harrison
Karen B. Thorwarth

PROPOSAL II — AUTHORIZATION FOR APPOINTMENT OF AUDITORS

Selection of Auditors

Trice Geary & Myers LLC, which was the Company's independent auditing firm for the 2005 fiscal year, is expected to be retained by the Audit Committee of the Board of Directors to be the Company's independent auditors for the 2006 fiscal year. A representative of Trice Geary & Myers LLC is expected to be present at the Annual Meeting to respond to appropriate questions from stockholders and will have the opportunity to make a statement if he or she so desires. **The Board of Directors recommends a vote FOR the proposal to authorize the Board of Directors to accept the selection of the Audit Committee of an outside auditing firm for the ensuing year.**

Disclosure of Independent Auditor Fees

The following is a description of the fees billed to the Company by Trice Geary & Myers LLC ("TGM") during the years ended December 31, 2004 and 2005:

Audit Fees. Audit fees include fees paid by the Company to TGM in connection with the annual audit of the Company's consolidated financial statements, and review of the Company's interim financial statements. Audit fees also include fees for services performed by TGM that are closely related to the audit and in many cases could only be provided by our independent auditors. Such services include consents related to SEC and other regulatory filings. The aggregate fees billed to the Company by TGM for audit services rendered to the Company for the years ended December 31, 2004 and December 31, 2005 totaled $80,598 and $84,329, respectively.

Audit Related Fees. Audit related services include due diligence services related to mergers and acquisitions, accounting consultations, and employee benefit plan audits. The aggregate fees billed to the Company by TGM for audit related services rendered to the Company for the years ended December 31, 2004 and December 31, 2005 totaled $7,285 and $9,640, respectively.

Tax Fees. Tax fees include corporate tax compliance, counsel and advisory services. The aggregate fees billed to the Company by TGM for the tax related services rendered to the Company for the years ended December 31, 2004 and December 31, 2005 totaled $8,752 and $6,620, respectively.

All Other Fees. The aggregate fees billed to the Company by TGM for all other services rendered to the Company for matters such as general consulting services and services in connection with annual and special meetings of stockholders for the years ended December 31, 2004 and December 31, 2005 totaled $5,044 and $6,703, respectively.

Approval of Independent Auditor Services and Fees

The Company's Audit Committee reviews all fees charged by the Company's independent auditors, and actively monitors the relationship between audit and non-audit services provided. The Audit Committee must pre-approve all audit and non-audit services provided by the Company's independent auditors and fees charged.

OTHER MATTERS

The Board of Directors is not aware of any business to come before the Annual Meeting other than those matters described above in this proxy statement and matters incident to the conduct of the Annual Meeting. However, if any other matters should properly come before the Annual Meeting, it is intended that proxies in the accompanying form will be voted in respect thereof in accordance with the determination of a majority of the named proxies.

SECTION 16(a) BENEFICIAL OWNERSHIP REPORTING COMPLIANCE

Pursuant to regulations promulgated under the Exchange Act, the Company's officers, directors and persons who own more than ten percent of the outstanding Common Stock ("Reporting Person") are required to file reports detailing their ownership and changes of ownership in such Common Stock, and to furnish the Company with copies of all such reports. Based on the Company's review of such reports which the Company received during the last fiscal year, or written representations from Reporting Persons that no annual report of change in beneficial ownership was required, the Company believes that, with respect to the last fiscal year, all persons subject to such reporting requirements have complied with the reporting requirements.

MISCELLANEOUS

The cost of soliciting proxies will be borne by the Company. The Company will reimburse brokerage firms and other custodians, nominees and fiduciaries for reasonable expenses incurred by them in sending proxy materials to the beneficial owners of Common Stock. In addition to solicitations by mail, directors, officers and regular employees of the Company may solicit proxies personally or by telegraph or telephone without additional compensation therefore.

The Company's 2005 Annual Report to Stockholders, including financial statements, has been mailed to all stockholders of record as of the close of business on the Record Date with this Proxy Statement. Any stockholder who has not received a copy of such Annual Report may obtain a copy by writing to the Secretary of the Company. Such Annual Report is not to be treated as a part of the proxy solicitation material or as having been incorporated herein by reference. **A copy of the Company's Form 10-K for the fiscal year ended December 31, 2005 as filed with the Securities and Exchange Commission will be furnished without charge to stockholders as of the Record Date upon written request to Chief Financial Officer, Glen Burnie Bancorp, 101 Crain Highway, S.E., Glen Burnie, Maryland 21061.**

STOCKHOLDER PROPOSALS

Any stockholder desiring to present a proposal at the 2007 Annual Meeting of Stockholders and wishing to have that proposal included in the proxy statement for that meeting must submit the same in writing to the Secretary of the Company at 101 Crain Highway, S.E., Glen Burnie, Maryland 21061, in time to be received by December 14, 2006. The persons designated by the Company to vote proxies given by stockholders in connection with the Company's 2006 Annual Meeting of Stockholders will not exercise any discretionary voting authority granted in such proxies on any matter not disclosed in the Company's 2007 proxy statement with respect to which the Company has received written notice no later than February 26, 2007 that a stockholder (i) intends to present such matter at the 2007 Annual Meeting, and (ii) intends to and does distribute a proxy statement and proxy card to holders of such percentage of the shares of Common Stock required to approve the matter. If a stockholder fails to provide evidence that the necessary steps have been taken to complete a proxy solicitation on such matter, the Company may exercise its discretionary voting authority if it discloses in its 2007 proxy statement the nature of the proposal and how it intends to exercise its discretionary voting authority.

BY ORDER OF THE BOARD OF DIRECTORS

Dorothy A. Abel
SECRETARY

Glen Burnie, Maryland
April 12, 2006

x **PLEASE MARK VOTES AS IN THIS EXAMPLE**

REVOCABLE PROXY

GLEN BURNIE BANCORP

2006 ANNUAL MEETING OF STOCKHOLDERS

The undersigned hereby constitutes and appoints F. William Kuethe, John E. Demyan, and William N. Scherer, Sr., or a majority of them, with full powers of substitution, as attorneys-in-fact and agents for the undersigned, to vote all shares of Common Stock of Glen Burnie Bancorp which the undersigned is entitled to vote at the Annual Meeting of Stockholders, to be held at La Fontaine Bleu, 7514 Ritchie Highway, Glen Burnie, Maryland on Thursday, May 11, 2006 at 2:00 p.m., Eastern Time (the "Annual Meeting"), and at any and all adjournments thereof, as indicated below and as determined by a majority of the named proxies with respect to any other matters presented at the Annual Meeting.

	FOR	VOTE WITHHELD	FOR EXCEPT
1. To elect as directors all nominees listed below:	o	o	o
Shirley E. Boyer			
Michael G. Livingston			
Edward L. Maddox			
Norman E. Harrison			

INSTRUCTION: TO WITHHOLD YOUR VOTE FOR ANY LISTED NOMINEE, MARK THE FOR EXCEPT BOX AND INSERT THAT NOMINEE'S NAME ON THE LINE PROVIDED BELOW.

	FOR	AGAINST	ABSTAIN
2. To authorize the Board of Directors to accept the auditors selected by the Audit Committee for the 2006 fiscal year	o	o	o

The Board of Directors recommends a vote **"FOR"** the above listed propositions.

IF YOU PLAN TO ATTEND THE ANNUAL MEETING, PLEASE CHECK THIS BOX o

THIS PROXY IS SOLICITED BY THE BOARD OF DIRECTORS

THIS PROXY WILL BE VOTED AS DIRECTED, BUT IF NO INSTRUCTIONS ARE SPECIFIED, THIS PROXY WILL BE VOTED FOR EACH OF THE ABOVE NOMINEES AND FOR PROPOSAL II. IF ANY OTHER BUSINESS IS PROPERLY PRESENTED AT THE ANNUAL MEETING, THIS PROXY WILL BE VOTED BY THOSE NAMED IN THIS PROXY IN ACCORDANCE WITH THE DETERMINATION OF A MAJORITY OF THE NAMED PROXIES. THIS PROXY CONFERS DISCRETIONARY AUTHORITY ON THE HOLDERS THEREOF TO VOTE WITH RESPECT TO THE ELECTION OF ANY PERSON AS DIRECTOR WHERE THE NOMINEE IS UNABLE TO SERVE OR FOR GOOD CAUSE WILL NOT SERVE AND MATTERS INCIDENT TO THE CONDUCT OF THE ANNUAL MEETING.

Please be sure to sign and date this Proxy here.

Date _____

_____ _____

Stockholder sign above Co-holder (if any) sign above

Detach above card, sign, date and mail in postage paid envelope provided.

GLEN BURNIE BANCORP

Should the above signed be present and elect to vote at the Annual Meeting or at any adjournment thereof and after notification to the Secretary of the Company at the Annual Meeting of the stockholder's decision to terminate this proxy, then the power of said attorneys and proxies shall be deemed terminated and of no further force and effect. The above signed hereby revokes any and all proxies heretofore given with respect to the shares of Common Stock held of record by the above signed. The above signed acknowledges receipt from the Company prior to the execution of this proxy of notice and a proxy statement and a 2005 Annual Report to stockholders for the annual meeting.

Please sign exactly as your name appears on the envelope in which this proxy was mailed. When signing as attorney, executor, administrator, trustee or guardian, please give your full title. If shares are held jointly, each holder should sign.

PLEASE ACT PROMPTLY
SIGN, DATE & MAIL YOUR PROXY CARD TODAY

IF YOUR ADDRESS HAS CHANGED, PLEASE CORRECT THE ADDRESS IN THE SPACE PROVIDED BELOW AND RETURN THIS PORTION WITH THE PROXY IN THE ENVELOPE PROVIDED
