GenSpera, Inc. (“GenSpera” or “the Company”) is a biotechnology company developing targeted therapies to treat cancerous tumors. The Company’s novel approach uses a prodrug—an inactive precursor of a drug that converts into its active form at a targeted site—to deliver a potent, cell-killing agent directly to tumors. GenSpera’s prodrugs employ one of two techniques: (1) targeting tumor-associated blood vessels; or (2) targeting tumors directly. In contrast to existing anti-angiogenic drugs, which may only block new blood vessel formation, the Company’s lead prodrug candidate, G-202, attacks existing tumor vasculature, potentially debilitating the tumor’s nutrient supply and causing cancer regression without toxicity to other areas of the body. G-202 has caused tumor regression in animal models of breast, prostate, bladder, and kidney cancer. A Phase I clinical trial with G-202 is ongoing at two major cancer centers. GenSpera’s technology can also be used to attack cancer cells directly by targeting the prodrug to enzymes found solely at tumor sites. Using this approach, GenSpera is developing G-115, which targets prostate cancer.

GenSpera’s technology was developed over 15 years at Johns Hopkins and other global research centers and funded by over $15 million in grants from the U.S. National Institutes of Health, the National Cancer Institute, and the U.S. Department of Defense, among others. The Company holds seven patents and four pending patent applications, which were acquired without any milestone or royalty payments due to third parties. GenSpera’s management team has extensive experience identifying oncology treatments and bringing them to the clinic. The Company’s Scientific Advisory Board is composed of individuals who are both inventors of GenSpera’s technology and major shareholders.

At March 31, 2010, the Company’s cash and cash equivalent position was ~$2.7 million. Subsequently, GenSpera raised roughly $2.7 million in gross proceeds in May 2010.
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Executive Overview

GenSpera, Inc. ("GenSpera" or “the Company”) is a development-stage company focused on discovering and developing targeted cancer therapeutics to treat a wide range of solid tumors, including breast, prostate, bladder, and kidney cancers. The Company’s novel prodrug technology combines a potent cytotoxin with a prodrug delivery system that activates the drug only within the tumor. The Company’s business strategy entails developing a series of therapies based on its target-activated prodrug technology platform, identifying potentially attractive drug candidates with solid intellectual property (IP) protection, and then developing these compounds through Phase I/II clinical trials. Once a candidate reaches this stage, the Company intends to license the rights for further development to more established pharmaceutical companies, which could then finalize drug development and market the resulting therapeutic.

GenSpera initiated a Phase I clinical trial with its lead prodrug, G-202, in early 2010. Upon completion of its Phase I trial, the Company expects to initiate multiple Phase II trials for G-202 in several different cancer types. GenSpera’s second drug, G-115, is designed to target prostate cancer. The Company owns and controls all rights to both G-202 and G-115. GenSpera aims to establish a strategic partnership to maximize the value of its drugs as they advance in the clinic.

Cancer

The human body is composed of trillions of cells that constantly grow, divide, and die. For the most part, the cells in the body are healthy and perform their vital functions. However, when healthy cells do not perform properly, they typically self-destruct and are replaced. Cancer cells reproduce uncontrollably, regardless of their abnormalities. While the exact mechanism of transformation that causes normal cells to become cancerous remains unknown, cancer cells are believed to develop due to mutations caused by changed or damaged DNA. Instead of dying when they should, the abnormal cells constantly grow and divide, producing new cells that are not needed by the body.

Once cancerous cells are established, they may rapidly invade surrounding tissues. As cancer cells grow, they require sufficient nutrition, which is initially acquired by feeding off of the body’s systems in a parasitic manner. In order for a tumor to continue to grow beyond the size of roughly a pinhead, its cancer cells recruit blood vessels through a process called tumor angiogenesis. This development enables cancer cells to achieve self-sustainable growth by acquiring their own blood supply, which further fuels the spread of tumors.

Cancer is the second most common cause of death in the U.S. In 2009, the American Cancer Society (ACS) estimated that roughly 1,500 individuals succumb to cancer daily, totaling more than 560,000 cancer-related deaths annually (Source: the ACS’s Cancer Facts & Figures 2009). The disease also impacts countries on an economic level. In 2009, the U.S. National Institutes of Health approximated the overall cost of cancer in 2008 to be $228.1 billion, encompassing $93.2 billion for direct medical costs and $134.9 billion for lost productivity due to illness or premature death.

Traditional chemotherapy involves treating patients with cytotoxins, which are compounds or agents that are toxic to cells. In the early stages of cancer, chemotherapy may be combined with surgery or radiation to improve the efficacy of a treatment regimen. In the later stages of the disease—after the cancer has spread to another region of the body—chemotherapy is often the only treatment option for many forms of cancer. However, traditional chemotherapies have several prominent disadvantages:

(1) they affect both healthy and cancer cells indiscriminately, causing negative side effects;

(2) they exert their toxic effect when cells divide, which can be ineffective in tumors with cells that divide at a slower rate than cells in normal tissues; and

(3) cancer cells may develop a drug resistance after repeated exposure to current chemotherapies, thus limiting the number of times a therapy can be used effectively.
GenSpera’s Prodrug Chemotherapy

Researchers are investigating prodrug chemotherapy as a technique to deliver higher concentrations of cytotoxic agents to tumors while avoiding the toxicity to healthy tissues of the body. Prodrug technology entails administering an inactive form of a cytotoxin, called a prodrug, to a patient. The prodrug is designed to be activated only through a conversion that occurs exclusively at a tumor site. If successfully developed, GenSpera believes that prodrug therapies could provide an effective therapeutic approach for a broad range of solid tumors caused by breast, prostate, lung, colon, and other cancers. The Company’s prodrug technology involves attaching a targeting/masking agent to an active drug, temporarily making the drug both soluble for intravenous administration and inactive in the bloodstream. Once the compound reaches its target, the masking agent is removed by an enzyme found at the tumor location. With the agent detached, the drug is reactivated and becomes insoluble, precipitating directly into nearby cancer cells. The cancer is killed due to the toxic effects of the activated drug.

GenSpera uses a two-tiered strategy to target its medicine specifically to tumor sites. The Company identifies select enzymes (proteases) that are found at higher levels in tumors relative to other tissues in the body. Upon identifying these enzymes, GenSpera creates peptides that are recognized predominantly by those enzymes in the tumor and not by enzymes in normal tissues. Because enzyme recognition is required to remove the selected peptide (the targeting/masking agent) and activate the cytotoxin, this aspect seeks to ensure that the compound does not cause toxicity in areas of the body other than the targeted site.

GenSpera uses 12ADT—a chemically modified form of the cytotoxin thapsigargin that kills fast-, slow-, and non-dividing cells—as the therapeutic element of its prodrugs, including in its lead candidate, G-202. Thapsigargin is a potent and novel cytotoxin extracted from the plant Thapsia garganica (T. garganica), which is 10- to 100-fold more potent than the National Cancer Institute’s reference chemotherapeutic agents. 12ADT functions by dramatically raising the level of calcium inside cells, which leads to cell death. The Company believes that 12ADT addresses several issues prevalent with current chemotherapies as it does not appear to trigger the development of resistance to its effects, and it is able to target cells regardless of their rate of division. GenSpera believes that targeting cell death independently of cell division is important for three reasons: (1) it allows the drug to be effective against tumor cells that divide more slowly than normal cells in the body (e.g., prostate cancer); (2) the drug can be effective against the very slowly dividing blood vessel cells within solid tumors; and (3) the drug may also kill cancer stem cells, which in general are also very slow dividing.

Presently, the Company is engaged in the development of two prodrug candidates, G-202 and G-115, as overviewed in Figure 1. GenSpera uses two approaches in its prodrugs: (1) targeting the blood supply that supports tumor growth; and (2) targeting the tumor directly. Each of these approaches is briefly summarized following Figure 1 and more fully detailed on page 19.

<table>
<thead>
<tr>
<th>Product Candidate</th>
<th>Activation Enzyme</th>
<th>Indication</th>
<th>Stage of Development</th>
</tr>
</thead>
<tbody>
<tr>
<td>G-202</td>
<td>Prostate-specific membrane antigen (PSMA)</td>
<td>Solid tumors</td>
<td>Preclinical, Phase I, Phase II, Phase III</td>
</tr>
<tr>
<td>G-115</td>
<td>Prostate-specific antigen (PSA)</td>
<td>Prostate cancer</td>
<td></td>
</tr>
</tbody>
</table>

Source: GenSpera, Inc.
Targeting the Blood Supply that Supports Tumor Growth

GenSpera is currently developing its lead prodrug candidate, G-202, which targets the blood vessels of solid tumors. G-202 is selectively activated within solid tumors by an enzyme present on the tumor blood vessels, thus destroying the existing tumor by ceasing its blood supply. The Company believes that this technique is a dramatic improvement to existing anti-angiogenic drugs, which primarily stop the growth of new blood vessels.

G-202 combines the cytotoxic activity of 12ADT with a specific peptide that masks its activity until it is delivered to the target site. The peptide can only be removed by prostate-specific membrane antigen (PSMA)—a process referred to as the targeted delivery of the active drug. PSMA is expressed in non-cancerous and cancerous prostate tissues, at lower levels in some non-prostate tissues (e.g., kidney), and in the endothelial cells of vasculature associated with non-prostate cancers (Source: *Journal of Carcinogenesis* 2006, 15[5]:21). Because PSMA is expressed in tumor-associated blood vessels, G-202 and any other PSMA-targeted prodrugs that GenSpera may develop could be able to attack the blood supplies of many different tumor types.

In preclinical testing, G-202 was shown to cause tumor regression in animal models with solid tumors of breast, prostate, bladder, and kidney cancer. G-202’s demonstrated anti-tumor effects in these cancer types further the belief that G-202 may have broad application as a therapy for a variety of human solid tumors due to its ability to selectively target PSMA-producing endothelial cells within tumors. In these studies, the administration of G-202 caused noticeable regression of the tumor—in some cases with no visible re-growth for approximately one month following the last treatment. G-202 was well tolerated at dose levels that caused regression of tumor growth, with no signs of toxicity. At the highest doses, transient weight loss was documented, which quickly recovered after each course of the therapy. The data also indicated that even after repeated dosing cycles, G-202 did not activate drug resistance in tumor cells.

GenSpera believes that its prodrug technology may be able to eliminate cancerous tumors with dosing that is effective for a significant length of time. Further, the Company believes that this is more likely to be achieved in humans than in mice due to the ability to infuse the drug into human patients and an anticipated longer half-life of G-202 in the human bloodstream versus that of laboratory animals. Given its efficacy profile, G-202 could also be effective as a monotherapy, thus reducing the costs and time required to conduct clinical trials.

Current Status of G-202

In early 2010, GenSpera initiated a Phase I study with G-202 to evaluate its safety and its pharmacokinetics in cancer patients. The open-label, dose-escalation Phase I study could include up to 30 refractory cancer patients—individuals who have relapsed following treatment with other chemotherapies—with any type of solid tumor. GenSpera expects this approach to enhance patient accrual rates and provide the Company with safety data across a range of cancer types. While the primary endpoints of the study are to evaluate the safety, tolerability, and pharmacokinetics of the drug in humans, the design of the trial also allows the collection of efficacy data.

The trial is ongoing at two major cancer centers: (1) the Sidney Kimmel Comprehensive Cancer Center at Johns Hopkins in Baltimore, Maryland; and (2) the University of Wisconsin Carbone Cancer Center in Madison, Wisconsin. As of June 2010, four patients had been enrolled in the study. Dependent upon the successful completion of the Phase I study, GenSpera plans to conduct up to four Phase II clinical trials over 18 months to determine the therapeutic efficacy of G-202 in different tumor types. Moreover, GenSpera may seek to establish a strategic partnership to maximize the value of G-202 as the compound progresses through future clinical trials.
Targeting the Tumor Directly

Compared to other cancers, prostate cancer has a large proportion of slowly proliferating cells that are resistant to treatment with conventional cytotoxic agents. Current therapies generally attack and destroy rapidly dividing cells, and thus spare cancer cells that divide slowly—a feature that is characteristic of prostate cancers. GenSpera’s technology has a broad range of potential applications, including techniques that pair cytotoxic derivatives of thapsigargin to peptides in order to target prostate tumors directly. As such, GenSpera sought to develop a prodrug candidate using this approach that avoided the negative aspects of current prostate cancer therapies.

GenSpera’s first approach to this strategy couples thapsigargin to peptides that are selectively cleaved by a prostate cancer-specific protease called prostate-specific antigen (PSA), which led to GenSpera’s lead candidate for this technique, G-115. PSA is active within tumor sites and in normal prostate tissue but is inactive within the bloodstream—characteristics that form the basis for tumor-specific delivery of cytotoxic agents. G-115 was selected as the lead development candidate in the PSA-targeted prodrug program due to its ability to dramatically inhibit the growth of tumors in animal models of human prostate cancer. GenSpera aims to obtain Investigational New Drug (IND) approval for G-115 in the third quarter 2011. Additionally, the Company seeks to establish a strategic partnership to maximize the value of G-115 as it progresses in the clinic.

It is important to note that G-115 and G-202 are non-competing product candidates; the Company intends to market G-115 to urologists and market G-202 to medical oncologists.

Corporate Information

Incorporated in Delaware in 2003, GenSpera was founded as part of a business development initiative based on technology and IP owned by Johns Hopkins. In early 2004, the IP underlying the Company’s technologies was assigned from Johns Hopkins to its co-inventors—Drs. John T. Isaacs, Samuel R. Denmeade, Soren Brogger Christensen, and Hans Lilja—who in turn awarded an option to license the IP to the Company in return for continued protection of the patent portfolio. GenSpera exercised this option in early 2008 by reimbursing Johns Hopkins for previous patent prosecution costs. The co-inventors, who now comprise the Company’s Scientific Advisory Board, assigned the IP to the Company in April 2008. GenSpera’s activities between 2004 and 2007 were limited to continued prosecution of the relevant patent portfolio.

After filing its Form 10-K in March 2010 without a “going concern” statement and raising $2.7 million in gross proceeds in May 2010, GenSpera believes that its ability to continue supporting its development initiatives financially differentiates the Company from many other biotechnology entities.

Presently, GenSpera’s Common Stock trades on the Over-the-Counter Bulletin Board (OTC.BB) under the ticker symbol “GNSZ.”

Headquarters and Employees

GenSpera’s headquarters are located in San Antonio, Texas, where the Company leases a roughly 850-square foot facility. GenSpera employs two full-time individuals who serve as the Company’s executive officers.
Growth Strategy

Leveraging the management team’s expertise for identifying promising treatments and bringing them into the clinic, GenSpera’s business strategy entails developing therapeutics based on the Company’s target-activated prodrug technology platform. The Company plans to advance these candidates through Phase I/II clinical trials and subsequently license the rights for further development to major pharmaceutical companies. While the Company has identified four prodrug candidates—G-202, G-114, G-115, and Ac-GKAFRR-L12ADT (as overviewed on pages 23-32)—GenSpera is presently focused on advancing G-202 and G-115. The Company will likely commence the development of the remaining candidates when sufficient resources are available. GenSpera is focused on minimizing its infrastructure in order to keep its burn rate as low as possible as it develops its lead molecules.

Manufacturing and Development

Under the direction of key personnel, GenSpera plans to outsource its preclinical development (e.g., toxicology), manufacturing, and clinical development initiatives to contract research organizations (CROs) and contract manufacturing organizations (CMOs). Commonly engaged in the pharmaceutical and biotechnology industries, CROs and CMOs are third parties that act on behalf of their clients and specialize in the execution of project-oriented research activities and processes. The Company intends to maintain Good Laboratory Practice (GLP) and Good Manufacturing Practice (GMP) standards by outsourcing its activities to organizations with approved facilities and manufacturing practices.

Commercialization

After developing candidates through Phase I/II clinical trials, the Company intends to license its therapies to third parties for further clinical development and commercialization. Table 1 highlights several recent examples of agreements for the license or sale of various clinical-stage cancer therapies. GenSpera believes that G-202 offers an even greater value for potential partners because it may address a broad range of solid tumors.

<table>
<thead>
<tr>
<th>Year</th>
<th>Acquirer/Licenser from drug developer</th>
<th>Pharmaceutical Application Clinical Status</th>
<th>Benefits to Drug Developer</th>
</tr>
</thead>
<tbody>
<tr>
<td>2007</td>
<td>GlaxoSmithKline plc from Synta Pharmaceuticals Corp.</td>
<td>• Metastatic melanoma drug • Phase II</td>
<td>• Upfront cash payment of $80M • Up to $300M at commercial milestones and up to $585M at other identified milestones</td>
</tr>
<tr>
<td>2009</td>
<td>Astellas Pharma Inc. from Medivation, Inc.</td>
<td>• Prostate cancer drug • Phase III</td>
<td>• Upfront payment of $110M • Up to $655M in milestone payments</td>
</tr>
<tr>
<td></td>
<td>sanofi-aventis SA from Exelixis Inc.</td>
<td>• Two inhibitors of cancer proteins • Phase I</td>
<td>• Upfront cash payment and potential development and regulatory milestone payments that could exceed $1B</td>
</tr>
<tr>
<td></td>
<td>sanofi-aventis SA from BiPar Sciences, Inc.</td>
<td>• An inhibitor of cancer protein • Phase II</td>
<td>• Acquisition of company for up to $500M, divided into varying milestone payments</td>
</tr>
<tr>
<td></td>
<td>Johnson &amp; Johnson from Cougar Biotechnology, Inc.</td>
<td>• Prostate cancer drug • Phase III</td>
<td>• Acquisition of company for nearly $1B</td>
</tr>
<tr>
<td>2010</td>
<td>Novartis AG from Array BioPharma Inc.</td>
<td>• Several cancer protein inhibitors • Phase I</td>
<td>• Upfront payment of $45M and up to $422M for development and regulatory milestones</td>
</tr>
</tbody>
</table>

Sources: GenSpera, Inc. and Crystal Research Associates, LLC.
Research Programs and Grants

In addition to developing second-generation approaches to its current programs, GenSpera intends to continue the characterization of its lead molecules in the laboratories of Drs. Isaacs and Denmeade at the Sidney Kimmel Comprehensive Cancer Center at Johns Hopkins. To date, the development of GenSpera’s technology platform has been supported by approximately $10 million in scientific grants for research performed in Dr. Isaacs’ and Dr. Denmeade’s laboratories at the Kimmel Cancer Center. The funding for research at the Kimmel Cancer Center has been provided by the U.S. National Institutes of Health, the National Cancer Institute’s Rapid Access to Intervention Development (RAID) program, the NCI’s Specialized Programs of Research Excellence (SPORE), and the Prostate Cancer Foundation (formerly CaPCURE), among others. Additional grants totaling roughly $5 million have supported research in Europe and elsewhere by Drs. Christensen and Lilja. GenSpera believes that the more than 15 years of support of its technologies by peer-reviewed funding agencies offers evidence of the technology platforms’ acceptance and recognized potential by academic and medical communities.
Intellectual Property

The intellectual property (IP) supporting GenSpera’s technology was developed over 15 years at Johns Hopkins University and the University of Copenhagen with over $15 million in scientific grants from the National Cancer Institute, the U.S. Department of Defense, and the U.S. National Institutes of Health, among others. GenSpera’s IP portfolio contains seven issued patents and four pending patent applications (as listed in Table 2), which protect the Company’s core prodrug platform and ancillary technologies. While GenSpera plans to continue to prosecute claims included in all four of its U.S. patent applications, the Company is placing a particular emphasis on protecting the outstanding claims covered in its core PSMA prodrug patent application. PSMA is a protein that is over-expressed in prostate cancer and in the tumor-associated vasculature of several solid tumor types, which is targeted by GenSpera’s lead prodrug candidate, G-202. The Company also protects its proprietary information through confidentiality agreements with employees, consultants, sponsored researchers, and significant scientific collaborators.

Table 2
GenSpera, Inc.
INTELLECTUAL PROPERTY

<table>
<thead>
<tr>
<th>Number</th>
<th>Country</th>
<th>Filing Date</th>
<th>Issue Date</th>
<th>Exp. Date</th>
<th>Title</th>
</tr>
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<tbody>
<tr>
<td>6,265,540</td>
<td>U.S.</td>
<td>05/19/1998</td>
<td>07/24/2001</td>
<td>05/18/2018</td>
<td>Tissue specific prodrug (PSA)</td>
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<td>6,410,514</td>
<td>U.S.</td>
<td>06/07/2000</td>
<td>06/25/2002</td>
<td>06/06/2020</td>
<td>Tissue specific prodrug (PSA)</td>
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<tr>
<td>6,504,014</td>
<td>U.S.</td>
<td>06/07/2000</td>
<td>01/07/2003</td>
<td>06/06/2020</td>
<td>Tissue specific prodrug (thapsigargin)</td>
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<tr>
<td>7,053,042</td>
<td>U.S.</td>
<td>07/28/2000</td>
<td>05/30/2006</td>
<td>07/27/2020</td>
<td>Activation of peptide prodrugs by hK2</td>
</tr>
<tr>
<td>7,635,682</td>
<td>U.S.</td>
<td>01/06/2006</td>
<td>12/22/2009</td>
<td>01/05/2026</td>
<td>Tumor activated prodrugs (G-115)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Number</th>
<th>Country</th>
<th>Filing Date</th>
<th>Issue Date</th>
<th>Exp. Date</th>
<th>Title</th>
</tr>
</thead>
<tbody>
<tr>
<td>US 2007/0160536</td>
<td>U.S.</td>
<td>01/06/2006</td>
<td>N/A</td>
<td>N/A</td>
<td>Tumor activated prodrugs (PSA, G-115)</td>
</tr>
<tr>
<td>US 2008/0247950</td>
<td>U.S.</td>
<td>03/15/2007</td>
<td>N/A</td>
<td>N/A</td>
<td>Activation of peptide prodrugs by hK2</td>
</tr>
<tr>
<td>US 2009/0163426</td>
<td>U.S.</td>
<td>11/25/2008</td>
<td>N/A</td>
<td>N/A</td>
<td>Tissue specific prodrugs (PSMA)</td>
</tr>
<tr>
<td>US 2010/0120697</td>
<td>U.S.</td>
<td>11/05/2009</td>
<td>N/A</td>
<td>N/A</td>
<td>Tumor activated prodrugs (G-115)</td>
</tr>
</tbody>
</table>

Sources: the U.S. Patent and Trademark Office (USPTO), the World Intellectual Property Organization (WIPO), and GenSpera, Inc.

GenSpera owns and controls all rights to both G-202 and G-115, the Company’s second anti-cancer drug in development. In December 2009, the Company was issued its most recent U.S. patent, entitled “Tumor activated prodrugs” (No. 7,635,682), which strengthens GenSpera’s IP position for G-115 and its use in prostate cancer and other prostate conditions (e.g., enlarged prostate).

The patents and patent applications supporting GenSpera’s technology were initially owned by Johns Hopkins University and assigned to the Company in April 2008. The co-inventors of the IP—Drs. Isaacs, Denmeade, Christensen, and Lilja—remain affiliated with GenSpera as members of its Scientific Advisory Board. While GenSpera has no further financial obligations to the inventors of the IP or to Johns Hopkins, the institution retains a fully paid, royalty-free, non-exclusive license to use the IP for nonprofit purposes.

To maintain a competitive advantage, GenSpera plans to continue to pursue protection for its core and ancillary technologies, either independently or with scientific collaborators or strategic partners. Primarily, GenSpera is focused on filing patent applications in the U.S. The Company also intends to obtain licenses or options to acquire licenses to IP that may be useful in furthering GenSpera’s research, development, and commercialization initiatives.
Company Leadership

GenSpera's leadership possesses biotechnology and pharmaceutical experience, including identifying and bringing oncology treatments to clinical development. Dr. Craig A. Dionne, the Company's chief executive officer, chief financial officer, president, and chairman, is also a co-founder of the Company, and brings roughly 21 years of experience identifying and investigating clinical oncology treatments. Supporting Dr. Dionne is Dr. Russell Richerson, who has over 25 years of experience in the biotechnology and diagnostics industries. GenSpera’s Board of Directors oversees the conduct of and supervises the Company’s key management. The Company’s Scientific Advisory Board includes Drs. Isaacs and Denmeade, who are also co-founders of GenSpera. GenSpera’s executive management and Board of Directors are overviewed in Table 3 followed by detailed biographies; the Scientific Advisory Board is summarized on pages 11-12.

<table>
<thead>
<tr>
<th>Name</th>
<th>Title</th>
</tr>
</thead>
<tbody>
<tr>
<td>Craig A. Dionne, Ph.D.</td>
<td>Chief Executive Officer, Chief Financial Officer, President, and Chairman of the Board</td>
</tr>
<tr>
<td>Russell Richerson, Ph.D.</td>
<td>Chief Operating Officer and Secretary</td>
</tr>
<tr>
<td>John M. Farah, Jr., Ph.D.</td>
<td>Director</td>
</tr>
<tr>
<td>Scott Ogilvie, J.D.</td>
<td>Director</td>
</tr>
</tbody>
</table>

Source: GenSpera, Inc.

Craig A. Dionne, Ph.D., Chief Executive Officer, Chief Financial Officer, President, and Chairman of the Board

Dr. Dionne has roughly 21 years of experience in the pharmaceutical industry, with increasing levels of responsibility throughout his career. He recently served as executive vice president, research and therapeutics with the Prostate Cancer Foundation, a nonprofit foundation that seeks improved treatments for advanced prostate cancer. This position gave Dr. Dionne a global perspective of potential advances in this therapeutic arena. Prior to the Prostate Cancer Foundation, he served five years as vice president in discovery research at Cephalon, Inc. (CEPH-NASDAQ), where he was responsible for drug discovery programs in the areas of oncology and neurobiology. His efforts at Cephalon were key in the identification of four drugs that were brought into clinical evaluation over a span of six years, including two targeted primarily toward prostate cancer and one anti-angiogenic agent for solid tumors. Dr. Dionne has extensive experience in corporate collaborations, having served on joint management teams in various capacities with eight different corporate partners while with Cephalon. Prior to joining Cephalon in 1992, he received a doctorate from the University of Texas in 1984, was trained as a postdoctoral fellow at Dana-Farber Cancer Institute, and worked as a research fellow at Rhone-Poulenc Rorer Pharmaceuticals, Inc. Dr. Dionne has had an extensive scientific career as demonstrated by co-inventorship on six issued U.S. patents as well as co-authorship of over 50 scientific publications.

Russell Richerson, Ph.D., Chief Operations Officer and Secretary

Dr. Richerson has over 25 years of experience in the biotechnology and diagnostic industries. Dr. Richerson most recently served as vice president of operations with the Molecular Profiling Institute (a for-profit spinoff of the Translational Genomics Research Institute) and Ameripath, Inc., and as the chief operating officer at the International Genomics Consortium (a nonprofit medical research organization). During Dr. Richerson’s career, he has served as the vice president of diagnostic research and development with Ventana Medical Systems (acquired in 2008 by Roche Diagnostics Division, a subsidiary of F. Hoffmann-La Roche Ltd [ROG-SWX]), Prometheus Laboratories Inc., and CellzDirect, Inc. In addition, Dr. Richerson spent 11 years at Abbott Laboratories (ABT-NYSE) in numerous management roles, most recently as director of molecular probes and director of the AxSym® program. His career also includes positions at Pandex Laboratories (acquired by Baxter International Inc. [BAX-NYSE]), E. I. du Pont de Nemours and Company (DD-NYSE), Coulter Diagnostics, and Boehringer Mannheim Diagnostics. Dr. Richerson holds a B.S. in medical technology from Louisiana State University and a Ph.D. in biochemistry from the University of Texas at Austin.
John M. Farah, Jr., Ph.D., Director on the Board of Directors

Dr. Farah is vice president, intercontinental operations at Cephalon, which he joined in 1992. He is responsible for ensuring corporate support and managing sales performance of international partners in the Americas and Asia Pacific with specific growth initiatives for Cephalon in China and Japan. Dr. Farah’s prior roles included the responsibility to promote and negotiate research and development and commercial alliances with multinational and regional pharmaceutical firms, as well as responsibilities in scientific affairs, product licensing, and academic collaborations. He currently serves on the Board of Directors of Aeolus Pharmaceuticals Inc. (AOLS-OTC).

Scott Ogilvie, J.D., Director on the Board of Directors

Mr. Ogilvie is president of AFIN International, Inc., a private equity/business advisory firm that he founded in 2006. Prior to December 31, 2009, he was chief executive officer of Gulf Enterprises International, Ltd, a company that brings strategic partners, expertise, and investment capital to the Middle East and North Africa. Mr. Ogilvie previously served as chief operating officer of CIC Group, Inc., an investment manager. He began his career as a corporate and securities lawyer with Hill, Farrer & Burrill LLP and has extensive public and private corporate board experience in finance, real estate, and technology companies. Mr. Ogilvie currently serves on the Board of Directors of Neuralstem, Inc. (CUR-NYSE Amex), Innovative Card Technologies, Inc. (INVC-OTC), and Preferred Voice Inc, (PRFV-OTC).

Scientific Advisory Board

GenSpera’s Scientific Advisory Board, listed in Table 4, encompasses leading researchers who are inventors of the Company’s technology as well as shareholders. The members of the Company's Scientific Advisory Board are active in terms of creating new intellectual property that GenSpera can use as well as in helping the Company develop its drug candidates through the preclinical process.

<table>
<thead>
<tr>
<th>Name</th>
<th>Title</th>
</tr>
</thead>
<tbody>
<tr>
<td>John T. Isaacs, Ph.D.</td>
<td>Chief Scientific Advisor, Chairman of the Scientific Advisory Board, and Co-founder</td>
</tr>
<tr>
<td>Samuel R. Denmeade, M.D.</td>
<td>Chief Clinical Advisor and Co-founder</td>
</tr>
<tr>
<td>Soren Brogger Christensen, Ph.D.</td>
<td>Member</td>
</tr>
<tr>
<td>Hans Lilja, M.D., Ph.D.</td>
<td>Member</td>
</tr>
</tbody>
</table>

Source: GenSpera, Inc.

John T. Isaacs, Ph.D., Chief Scientific Advisor, Chairman of the Scientific Advisory Board, and Co-founder

Dr. Isaacs is a professor of oncology at the Kimmel Cancer Center as well as a professor in the urology department of the James Buchanan Brady Urological Institute, part of Johns Hopkins School of Medicine in Baltimore, Maryland. He has had a longstanding interest in the regulation of the growth of both the normal and abnormal prostate, with a particular interest in developing new approaches for the treatment of prostate cancer. Dr. Isaacs is the editor-in-chief of the journal *The Prostate* and is on the Editorial Board of *Clinical Cancer Research, Cancer Research, Endocrine Related Cancer, and Cancer and Metastases Reviews*. From 1990 to 1991, he was president of the Society for Basic Urological Research. Dr. Isaacs has also been the director of the Cellular and Molecular Medicine Graduate Program in the Johns Hopkins School of Medicine and has served on the Experimental Therapeutics Study Section with the U.S. National Institutes of Health. He has been a director of the Experimental Therapeutics Division and a co-director of both the Prostate Cancer Program and the Chemical Therapeutics Program in the Kimmel Cancer Center. He also served on the Scientific Advisory Board of Cephalon, and has published over 300 scientific publications.
Samuel R. Denmeade, M.D., Chief Clinical Advisor and Co-founder

Dr. Denmeade is an associate professor of oncology and pharmacology at the Kimmel Cancer Center and an associate professor in the Chemical and Biomolecular Engineering Department at Johns Hopkins. He is also a member of the Anti-Cancer Drug Development Training Program at Johns Hopkins. Dr. Denmeade’s laboratory focus is on the development of targeted therapies for the treatment of prostate cancer. In particular, his laboratory has developed prodrug therapies that are activated by prostate cancer-specific proteases. Dr. Denmeade has published more than 40 papers in this area. He receives funding through the National Cancer Institute’s Prostate Cancer SPORE award and through awards from both the prostate and breast cancer research programs of the U.S. Department of Defense. Dr. Denmeade is a Board-certified medical oncologist and a member of the clinical Genitourinary Oncology Group at Johns Hopkins. He has been the principal investigator on a number of prostate cancer clinical trials. Dr. Denmeade is on the Editorial Board of The Prostate and is serving on the Physical Imaging Study Section for the Department of Defense’s Prostate Cancer Research Program. He is a consultant for Cephalon and Protox Therapeutics Inc. (PRX-TSX), and also serves on Protox’s Scientific Advisory Board.

Soren Brogger Christensen, Ph.D.

Dr. Christensen is professor, Department of Medicinal Chemistry, the Faculty of Pharmaceutical Sciences, University of Copenhagen, Denmark, and is internationally recognized for his work in the isolation and identification of natural products with significant biological activities. He has also served as chairman of the Department of Medicinal Chemistry. He discovered the antiparasitic effects of licochalcone A and is co-inventor on several patents in the IP portfolio of LICA Pharmaceuticals A/S, a closely held Danish biopharmaceutical research and development company. Dr. Christensen co-founded LICA Pharmaceuticals and formerly served as a member of its Board of Directors. He was the first to elucidate the chemical structure of thapsigargin, and his laboratory is actively engaged in exploring further derivatives of the bioactive compound. Dr. Christensen has co-authored over 130 scientific publications and is co-inventor on several key patents in GenSpera’s IP portfolio.

Hans Lilja, M.D., Ph.D.

Dr. Lilja is attending research clinical chemist at the Department of Clinical Laboratories with joint appointments at the Departments of Urology and Medicine (Genitourinary Oncology Services) at Memorial Sloan-Kettering Cancer Center (MSKCC) in New York. He is also professor (visiting) at the Department of Laboratory Medicine, Lund University, University Hospital (UMAS), Malmö, Sweden, where he previously was trained and most recently held a tenured full-time position as professor and chief physician until he was recruited to MSKCC. Dr. Lilja is an internationally recognized authority on the biology of PSA and hK2. He holds several diagnostic patents and is co-inventor for one of the most widely used commercial PSA assays. Dr. Lilja has won numerous international awards for his work on PSA assay methods and has co-authored more than 130 peer-reviewed publications. His continuing research on prostate cancer biomarkers has expanded to also include additional human glandular kallikreins, which hold an important place in GenSpera’s pipeline of prostate cancer therapeutics.
Core Story

GenSpera, Inc. ("GenSpera" or "the Company") is a development-stage biotechnology company focused on the discovery and development of prodrug cancer therapeutics. A prodrug is an inactive precursor of a drug that is converted into its active form at a targeted site. The Company believes that, if successfully developed, prodrug therapies have the potential to provide an effective therapeutic approach for a broad range of solid tumors.

GenSpera has proprietary technologies that appear to meet the requirements for an effective prodrug in animal models. In addition, the cytotoxic agent used in the Company’s prodrug candidates may address several limitations of current cancer drugs due to the following key characteristics of GenSpera’s cytotoxic agent: (1) it kills slow- and non-dividing cancer cells as well as rapidly proliferating (dividing) cancer cells; (2) it does not appear to trigger tumor resistance to its effects; and (3) it limits cytotoxicity to healthy cells by remaining inactive until it reaches the tumor site. GenSpera plans to develop its prodrug candidates through Phase I/II clinical trials and then license the experimental drugs to third parties under the assumption that such entities would continue to develop, market, sell, and distribute the resulting products.

CANCER

The human body is composed of trillions of cells that constantly grow, divide, and die. For the most part, the cells in the body are healthy and perform their vital functions. When healthy cells do not behave properly, they self-destruct and are replaced. However, cancer cells differ in this aspect because they reproduce regardless of their abnormalities. There are over 200 types of cancers, all of which entail the uncontrolled division and growth of abnormal cells in the body. Cancers are most often named for the organ or type of cell in which they originate, despite the fact that cancer cells can eventually spread into other parts of the body through the bloodstream or lymphatic system.

While the exact mechanism of transformation that causes normal cells to become cancerous remains unknown, cancer cells are believed to originally develop as a result of mutations due to changed or damaged DNA. The abnormal cells continuously grow and divide, producing new cells that are not needed by the body. Once cancer cells develop, they are able to rapidly invade surrounding tissues to the extent that normal processes in the surrounding tissue and organs may be inhibited or completely stopped. Another difference between cancer cells and healthy cells is that when touching another cell healthy cells undergo contact inhibition—a natural process that prevents the cell from multiplying. In contrast, cancer cells continue to multiply even when in contact with other cells, enabling the disease to form a mass of tissue called a tumor or growth.

The Growth and Spread of Cancer

As cancer cells grow, they require sufficient nutrition, which is initially acquired by feeding off of the body's systems in a parasitic manner. However, in order to grow beyond the size of roughly a pinhead, cancer cells also recruit blood vessels through a process called tumor angiogenesis. This activity enables cancer cells to achieve self-sustainable growth by acquiring their own blood supply, which further promotes the growth and spread of tumors. Figure 2 (page 14) illustrates the lifecycle of cancerous cells, from their origin, through the recruitment of blood vessels for growth, and to their spread to other parts of the body.
Cancer spreads, or **metastasizes**, when cancer cells break away from the primary tumor site and move to other regions of the body via the blood or the lymphatic system. When the cells reach a new destination, they may once again begin to divide and invade the surrounding area. Ultimately, these cells form a new tumor, called a metastatic tumor, in a different part of the body. The cancer cells located in the metastatic tumor are similar to the cells from the original tumor. For example, if the cancer originated in the breast and subsequently spread to the lungs, the cells located in the lungs would be considered metastatic breast cancer cells.

While cancer can be fatal even if it does not metastasize, a significant portion of cancer-related deaths are caused by tumor metastases. Most common forms of cancer (e.g., prostate, breast, colon, and lung cancer) develop in organs that can be completely or partially removed by surgery. Although removing such organs has negative effects, these procedures could cure patients if the cancer does not spread. The metastasis of cancer causes many serious consequences, particularly if it spreads to an essential part of the body (e.g., the brain) or if excessive cell division in an organ disrupts the body’s natural metabolism (Source: American Cancer Society [ACS]).

**Current Cancer Treatments**

Once cancer has metastasized, it can either be treated with a single therapy or a combination of therapies. The type of treatment generally depends on the type of primary cancer, the patient’s age and health, the size and location of the metastasis, and the treatments the patient has had in the past. The following therapies can be used for various purposes, including to control or stop the growth of cancer or to relieve the symptoms or side effects caused by other treatment methods.

- **Chemotherapy.** This technique uses a drug or a combination of drugs to slow or reverse the spread of cancer. Chemotherapeutics work by targeting rapidly dividing cells, such as cancer cells. However, fast-growing healthy cells—including blood cells forming in the bone marrow as well as cells in the digestive tract, reproductive system (sexual organs), and hair follicles—may also be affected. Side effects include fatigue, nausea, vomiting, pain, hair loss, and anemia, among others.

- **Surgery.** The Mayo Foundation for Medical Education and Research considers cancer surgery to be the foundation of cancer treatment. An operation to repair or remove part of the body is used for a variety of purposes, including for cancer prevention, diagnosis, determining the cancer’s progression (stage), to enhance the efficacy of an alternate therapy, or to relieve symptoms or side effects. As such, cancer surgery may be employed alone or may be supplemented with other treatments, such as chemotherapy, radiation, biological therapy, or hormone therapy.

- **Radiation Therapy.** This treatment uses high-energy radiation that kills cancer cells and reduces the size of tumors. Like surgery, radiation therapy is a localized treatment that only affects cancer cells in the treated area. Radiation most often comes from a machine (external radiation), but the therapy can
also be administered from a small container of radioactive material implanted directly into or near the tumor (internal radiation). The National Cancer Institute estimates that 50% of cancer patients are treated with radiation therapy, either alone or in combination with other cancer treatments. Side effects include skin changes, fatigue, diarrhea, hair loss at the treated area, nausea, vomiting, and swelling, among others.

- **Biological Therapy.** This type of treatment works with a patient’s immune system to help fight cancer or control side effects caused by other cancer treatments. While the exact mechanism that causes biological therapy to be effective is unknown, researchers speculate that the technique may prevent cancer from spreading, stop or slow the growth of cancer cells, or enhance the immune system’s ability to destroy cancer cells. Side effects include fatigue, fever, chills, gastrointestinal upset, and body aches, among others. While this type of therapy can come in the form of pills or at-home injections, other biological treatments must be given intravenously at the hospital or a clinic.

- **Hormone Therapy.** Depending on the type of cancer, hormones can either help cancer cells to grow (e.g., prostate cancer and breast cancer), or hormones can kill cancer cells or slow or stop their growth. As such, hormone therapy may entail taking medications that interfere with natural hormone activity or stop the production of hormones. In some cases, surgical removal of the gland that produces the hormones may be necessary. Side effects of various hormone treatments include hot flashes, impotence, a loss of desire for sexual relations, male breast enlargement, nausea, vomiting, vaginal spotting, irregular menstrual periods, fatigue, skin rash, loss of appetite or weight gain, and headaches, among others.

- **Cryosurgery.** This is a non-surgical technique that entails freezing and killing abnormal cells on the skin or inside the body to treat several conditions, including non-metastatic liver cancer, cancer that has spread to the liver from another site, non-metastatic prostate cancer, cancerous and non-cancerous bone tumors, early-stage skin cancers, and some precancerous conditions. While still under study to determine its long-term efficacy, cryosurgery is believed to be less costly than other treatments with fewer side effects.

Traditional, localized approaches—such as surgery or radiation therapy—can be largely ineffective for metastatic cancers because the effects of the treatment are primarily exhibited at the treatment site. In addition, while chemotherapy is widely used for metastatic disease, this type of therapy causes significant repercussions. Many traditional chemotherapies target cancer cells by killing cells during the act of cell division, as cancer cells divide more rapidly than normal cells of the body. However, this strategy also harms healthy cells of the body that also rapidly divide, causing hair loss, gastrointestinal distress, and bone marrow suppression. Additionally, these therapies have little activity against slow-growing tumors (e.g., prostate cancer) and have virtually no activity on cancer stem cells—which drive the growth of tumors yet have a slow proliferation rate. Cancer stem cells that are not eradicated during treatment can lead to cancer recurrence. As well, many cancers can also become resistant to conventional chemotherapies. Numerous approaches have attempted to resolve these drawbacks by targeting the delivery of cancer drugs directly to the tumor site. Nevertheless, GenSpera believes that these approaches have generally had limited success due to the factors summarized below.

- Conventional targeted delivery systems may still allow the cancer therapeutic to enter into the bloodstream, making it difficult to deliver sufficient concentrations to the tumor and increasing the chances of side effects in normal tissues.

- Angiogenesis inhibitors, which aim to cut off the blood supply to tumors, primarily prevent new blood vessels from growing. Therefore tumor growth can be slowed, but not reversed.

- The development of resistance to the drug as well as a medicine’s ineffectiveness against slowly dividing tumor cells continue to be major obstacles facing drug therapies for cancer.

Roche Holding AG’s Avastin®, a widely used angiogenesis inhibitor, primarily stops the growth of new tumor-related blood vessels. Avastin® is often used in combination with traditional chemotherapeutic agents, which shrink the tumors while Avastin® slows the growth. In lung cancer patients, using Avastin® in combination with paclitaxel and carboplatin (PC) increased median survival by roughly two months versus PC therapy alone. Similar results were reported for Avastin® in combination with IFL chemotherapy in colorectal cancer. Despite the limited survival advantage and its high costs at roughly
$4,400 per month—global sales of Avastin® in 2009 were 6.2 billion Swiss francs (approximately $5.8 billion) (Source: BIOWORLD Today and Roche Holdings AG [www.roche.com]).

While this may validate the approach of targeting cancer blood vessels, GenSpera believes that significant improvements can be made to Avastin® and other existing therapies. For example, when Avastin® is used in combination with other chemotherapeutic agents, the patient retains all of the side effects of the original chemotherapy as well as the new side effects of Avastin®. The Company’s product candidate, G-202, is being developed as a monotherapy. Additionally, while Avastin® blocks a protein to prevent the formation of new tumor vasculature, GenSpera’s prodrug technology differs in that it kills new and existing tumor vasculature. Furthermore, G-202 attacks cancer cells independently of cell division and may therefore be useful in slow- as well as fast-growing tumors. G-202 may also kill cancer stem cells—cells that have a very low proliferation rate but are largely responsible for tumor growth. Because of their slow-dividing nature, cancer stem cells may evade treatment by standard chemotherapeutic agents and are often the cause of cancer relapse.

**Market Size for Cancer Therapies**

Although significant improvements in cancer diagnosis have been made in recent years and various therapies have been developed to treat the disease, cancer continues to be a leading healthcare challenge worldwide. In the U.S., nearly one out of two men and roughly one out of three women develop cancer in their lifetime (Source: the ACS’s *Cancer Facts & Figures 2009*). In 2009 alone, the ACS estimated that nearly 1.5 million new cases of cancer would be diagnosed in the U.S. and over 560,000 patients would die as a result of cancer—over 1,500 people per day—making cancer the second most common cause of death in the U.S. behind cardiovascular disease.

With global sales of $47.7 billion, cancer is considered to be one of the largest, fastest growing markets in the pharmaceutical sector (Source: Business Insights’ *The Cancer Market Outlook to 2014*, December 2009). The cancer therapy market encompasses four key segments: (1) chemotherapy; (2) hormone therapy; (3) target therapy; and (4) immunotherapy. BBC Research indicated that target therapy presently captures the largest share of the market with estimated 2008 sales of $22.9 billion. Further, this segment is expected to triple by 2013, reaching roughly $69.1 billion in sales (Source: *Cancer Therapies: Technologies and Global Markets*). In contrast, the second largest category, chemotherapy, was expected to reach approximately $14.3 billion in 2008. Figure 3 portrays the estimated values of each of the four key segments in the global cancer therapy market from 2006 to 2013.

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**Table 5 (page 17) from the ACS estimates the new cases and related deaths in 2010 for a variety of cancers common in the U.S. GenSpera believes that its prodrug therapy, if successfully developed, has the potential to treat a broad range of solid tumors. The highlighted sections in Table 5 summarize the potential U.S. patient populations that may be amenable to the Company’s prodrug therapies, representing potential target markets.**

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*Source: BBC Research & Consulting’s Cancer Therapies: Technologies and Global Markets (May 2008).*
<table>
<thead>
<tr>
<th>Estimate New Cases and Deaths by SEX</th>
<th>U.S., 2010*</th>
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</thead>
<tbody>
<tr>
<td><strong>Estimated New Cases</strong></td>
<td>Both Sexes</td>
</tr>
<tr>
<td>All Sites</td>
<td>1,529,580</td>
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<tr>
<td>Oral cavity &amp; pharynx</td>
<td>36,540</td>
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<tr>
<td>Tongue</td>
<td>10,990</td>
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<tr>
<td>Mouth</td>
<td>10,840</td>
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<tr>
<td>Pharynx</td>
<td>12,660</td>
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<tr>
<td>Other oral cavity</td>
<td>2,050</td>
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<tr>
<td>Digestive system</td>
<td>274,330</td>
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<tr>
<td>Esophagus</td>
<td>16,640</td>
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<tr>
<td>Stomach</td>
<td>21,000</td>
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<tr>
<td>Small intestine</td>
<td>6,960</td>
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<tr>
<td>Colon†</td>
<td>102,900</td>
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<tr>
<td>Rectum</td>
<td>39,670</td>
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<tr>
<td>Anus, anal canal, &amp; anorectum</td>
<td>5,260</td>
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<tr>
<td>Liver &amp; intrahepatic bile duct</td>
<td>24,120</td>
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<tr>
<td>Gallbladder &amp; other biliary</td>
<td>9,760</td>
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<tr>
<td>Pancreas</td>
<td>43,140</td>
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<tr>
<td>Other digestive organs</td>
<td>4,880</td>
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<tr>
<td>Respiratory system</td>
<td>240,610</td>
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<tr>
<td>Larynx</td>
<td>12,720</td>
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<tr>
<td>Lung &amp; bronchus</td>
<td>222,520</td>
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<tr>
<td>Other respiratory organs</td>
<td>5,370</td>
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<tr>
<td>Bones &amp; joints</td>
<td>2,650</td>
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<tr>
<td>Soft tissue (including heart)</td>
<td>10,520</td>
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<tr>
<td>Skin (excluding basal &amp; squamous)</td>
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<tr>
<td>Melanoma-skin</td>
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<tr>
<td>Other nonepithelial skin</td>
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<tr>
<td>Breast</td>
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<tr>
<td>Genital system</td>
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<tr>
<td>Uterine cervix</td>
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<tr>
<td>Uterine corpus</td>
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<tr>
<td>Ovary</td>
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<td>Vulva</td>
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<td>Prostate</td>
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<tr>
<td>Testis</td>
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<td>Penis &amp; other genital, male</td>
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<td>Urinary system</td>
<td>131,260</td>
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<td>Urinary bladder</td>
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<tr>
<td>Kidney &amp; renal pelvis</td>
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<tr>
<td>Urter &amp; other urinary organs</td>
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<tr>
<td>Eye &amp; orbit</td>
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<tr>
<td>Brain &amp; other nervous system</td>
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<tr>
<td>Endocrine system</td>
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<td>Thyroid</td>
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<tr>
<td>Other endocrine</td>
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<tr>
<td>Lymphoma</td>
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<td>Hodgkin lymphoma</td>
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<td>Non-Hodgkin lymphoma</td>
<td>65,540</td>
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<td>Myeloma</td>
<td>20,180</td>
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<tr>
<td>Leukemia</td>
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<tr>
<td>Acute lymphocytic leukemia</td>
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<td>Chronic lymphocytic leukemia</td>
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<td>12,330</td>
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<td>Chronic myeloid leukemia</td>
<td>4,870</td>
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<tr>
<td>Other leukaemia</td>
<td>5,530</td>
</tr>
<tr>
<td>Other &amp; unspecified primary sites†</td>
<td>30,680</td>
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</tbody>
</table>

* Rounded to the nearest 10; estimated new cases exclude basal and squamous cell skin cancers and in situ carcinomas except urinary bladder. About 54,010 female carcinoma in situ of the breast and 46,770 melanoma in situ will be newly diagnosed in 2010.

† Estimated deaths for colon and rectum cancers are combined. † More deaths than cases may reflect lack of specificity in recording underlying cause of death on death certificates or an undercount in the case estimate.

Note: Estimated new cases are based on 1995-2006 incidence rates from 44 states and the District of Columbia as reported by the North American Association of Central Cancer Registries, representing about 89% of the U.S. population. Estimated deaths are based on data from U.S. Mortality Data, 1969 to 2007, National Center for Health Statistics, CDC, 2010.

GENSPERA’S PRODRUG TECHNOLOGY

Prodrug chemotherapy is a relatively new approach to treat cancer that is being investigated as a means to deliver higher concentrations of cytotoxic agents to tumors while avoiding the toxicity of these high doses in other areas of the body. In prodrug chemotherapy, the patient is administered the inactive form of a cytotoxin—called a “prodrug”—which converts into its active form only at the targeted tumor site. GenSpera aims to develop a prodrug chemotherapy that improves upon the weaknesses of current therapies and evades the obstacles listed on page 15, particularly for metastatic cancers. As overviewed on page 19, GenSpera’s prodrugs target cancer cells in one of two ways: (1) by targeting tumor-associated blood vessels; or (2) by targeting tumors directly.

The Company’s lead prodrug candidate, G-202, is designed to attack existing tumor vasculature, potentially debilitating the tumor’s nutrient supply and causing cancer regression without affecting healthy tissues within the body. GenSpera believes that it has validated G-202 as a drug candidate to treat various forms of solid tumors—including breast, bladder, kidney, and prostate cancers—based on its ability to cause tumor regression in animal models of these diseases. GenSpera is conducting Phase I clinical testing of G-202 at two major cancer centers: (1) the Sidney Kimmel Comprehensive Cancer Center at Johns Hopkins; and (2) the University of Wisconsin Carbone Cancer Center.

GenSpera’s technology can also be used to attack cancer cells directly by targeting the prodrug to enzymes believed to be found solely at a tumor location. The Company’s primary candidate using this approach, G-115, targets prostate-specific antigen (PSA), a protein produced by the cells of the prostate gland that is used by physicians as a “tumor marker” to detect the presence of prostate cancer.

GenSpera believes that its prodrugs may minimize toxicity to other areas of the body because it is converted into its active cytotoxic form by specific enzymes that are over-expressed at cancer sites. In addition to minimizing side effects in healthy tissues, the prodrugs are designed to have greater anti-tumor efficacy based upon the novel mode of action of the Company’s proprietary cytotoxic agent. GenSpera’s prodrug technology is fully detailed below and on pages 19-23 of the Core Story. The Company believes that its prodrug therapy, if successfully developed, has the potential to treat a broad range of solid tumors.

Prodrugs are often developed by adding an appendage to the original parent drug molecule in order to alter its physicochemical properties and enhance its ability to be delivered. Thus, prodrugs are created to overcome the barrier(s) to utility found in the parent drug molecule. There are several common barriers to drug delivery, including lack of site specificity, which often causes negative side effects in patients. The process of transforming GenSpera’s prodrugs—from the original cytotoxin thapsigargin to its derivative 12ADT to its final prodrug format—is depicted in Figure 4.

GenSpera is working to design prodrugs that deliver the parent drug through the systemic circulation to the desired site of action in a safe, effective, and efficient manner. The Company seeks to identify specific enzymes that are found at high levels in tumors relative to other tissues in the body. Upon identifying these enzymes, GenSpera creates peptides that are recognized predominantly by those enzymes in the
tumor and not by enzymes in normal tissues. This double layer of recognition adds to the tumor-targeting benefits found in the Company’s prodrugs. GenSpera leverages two separate approaches as platforms to develop its prodrug candidates, each of which overviewed below.

(1) Targeting the Tumor’s Blood Supply. For this technique, the Company has designed a prodrug candidate that includes a peptide to temporarily mask the activity of the cytotoxin while also helping to target the cytotoxin to the site of the tumor. The peptide, which also facilitates intravenous delivery of the prodrug, can only be removed by an enzyme that is commonly found in blood vessels supporting solid tumors. Once the peptide is removed, the cytotoxin is released into the cells of the tumor’s blood vessels, causing the network to collapse. Once the tumor’s nutrient source has been destroyed, the tumor cells begin to starve, ultimately leading to their death. This platform is applied to G-202, GenSpera’s lead prodrug candidate.

(2) Targeting the Tumor Cells Directly. This method involves selecting peptides for prodrugs that can be removed by enzymes found in tumors. Once the peptide is removed, the active drug precipitates directly into nearby cancer cells. The drug’s cytotoxicity causes the death of cancer cells within the tumor. Using this approach, GenSpera has developed two prodrug candidates for prostate cancer—including G-115, its lead development candidate for this platform—with encouraging results in animal studies thus far. The Company also intends to target other forms of cancer via this platform. GenSpera chose G-115 as its lead development candidate in the PSA-targeted prodrug program due to its enhanced PSA-substrate and in vivo anti-tumor activity, as well as its broader intellectual property (IP) coverage.

GenSpera’s target-activated prodrug technology platforms may enable the Company to address a wide range of solid tumors, as shown in Figure 5. GenSpera has also proposed several alternate applications for its technology, including serving as an aid in diagnostic imaging or alleviating symptoms associated with benign prostatic hypertrophy (BPH).
The Potential of GenSpera’s Prodrug Therapies

Numerous cancer treatments (e.g., chemotherapy) use cytotoxins—compounds or agents that are toxic to cells—to kill cancer cells. Cytotoxins can have direct destructive effects on specific cells in the body. However, current chemotherapies for cancer patients entail the use of compounds that are toxic to both cancer and healthy cells. In the early stages of cancer, chemotherapy can be combined with surgery or radiation, but in the later stages of the disease, it is the preferred or only treatment option for many patients. However, the Company believes that there are several major drawbacks of chemotherapy, as listed below.

- **Side Effects.** Currently available cytotoxic agents cause toxicity to both cancer cells and non-cancer cells in the body, often leading to serious side effects. In particular, rapidly dividing cells—such as those from hair follicles or bone marrow—often suffer significant damage from chemotherapy, potentially leading to hair loss and impaired immune systems, among other side effects.

- **Incomplete Tumor Kill.** Many leading chemotherapies act by preventing further division of cancer cells. Once a cytotoxin is administered to a cancer patient, the fast-dividing cancer cells are often destroyed at a more rapid rate than slower-dividing, non-cancerous cells. As such, efficacy is generally lower for tumors containing cells that divide slowly.

- **Resistance.** After repeated exposure to chemotherapy drugs, cancer cells often develop a resistance to the agent, thus limiting the number of times that a treatment can be administered effectively.

Conversely, prodrug chemotherapy is being investigated as a means to deliver higher concentrations of the cytotoxic agents to the tumor location, while avoiding the toxicity of these higher doses in the rest of the body. Prodrug therapy entails administering the cytotoxin to the patient in an inactive form, which is converted into the active cytotoxin at the tumor site. GenSpera believes that, if successfully developed, prodrug therapies have the potential to be effective treatments for a broad range of solid tumors. In animal models, the Company’s proprietary technologies appear to meet the requirements for an effective prodrug, as further detailed on pages 25-29 under “Antitumor Efficacy Studies of G-202.” GenSpera anticipates that its cytotoxin addresses two additional issues prevalent with current cancer therapeutics: (1) it kills both slowly and non-dividing cancer cells as well as rapidly dividing cancer cells; and (2) it does not appear to prompt drug resistance in cancer cells.

**How GenSpera’s Prodrugs Work**

GenSpera’s prodrugs employ a novel process to prevent its cytotoxin from harming healthy, non-cancerous cells in the body, whether rapidly or slowly dividing. The Company’s technology supports the creation of prodrugs by attaching targeting/masking agents to a cytotoxin in a manner that allows conversion of the prodrug to its active form only at a tumor site. GenSpera has chemically modified a potent cytotoxin called thapsigargin to develop 12ADT, a derivative of thapsigargin that is also capable of killing fast-, slow-, and non-dividing cells indiscriminately.

The Company believes that targeting cell death independently of cell division is important for three reasons:

1. It allows the drug to be effective against tumor cells that divide more slowly than normal cells in the body (e.g., prostate cancer);

2. The drug can be effective against the slow-dividing blood vessel cells within solid tumors; and

3. The drug may also kill cancer stem cells, which in general also divide slowly.

A simplified depiction of GenSpera’s therapeutic process is provided in Figure 6 (page 21), followed by details of thapsigargin, 12ADT, and the targeting/masking agents.
Thapsigargin

Thapsigargin is a highly potent compound extracted from the Mediterranean plant *Thapsia garganica* (*T. garganica*). Thapsigargin is a well-characterized and studied compound that, unlike other cytotoxic agents, has the ability to kill slow and non-dividing cells in addition to the rapidly dividing cells that are typically associated with cancer. It is important to note that thapsigargin induces apoptosis, or programmed cell death, which is a type of non-inflammatory cell death that also occurs during normal tissue development. Thapsigargin elicits cell death by blocking the SERCA pump, which works to maintain a low level of calcium within cells. Once the SERCA pump is blocked, the concentration of cytosolic calcium increases rapidly, leading to the death of the treated cells via apoptosis. Because the SERCA pump is expressed in the majority of tissues in the body, it is critical for the delivery mechanism to only release the active drug at the cancer sites or tumor vasculature within the patient.

While *T. garganica* is relatively common in the wild, to the Company's knowledge, Thapsibiza S.L. is the only commercial supplier of *T. garganica* seeds, which are harvested from the coastal regions of the Mediterranean Sea. In April 2007, GenSpera obtained the proper permits from the U.S. Department of Agriculture (USDA) for the import of *T. garganica* seeds into the U.S. and, in January 2008, entered into a five-year sole-source agreement with Thapsibiza. Per the material terms of the agreement, the Company is permitted to seek additional suppliers to supplement its supply from Thapsibiza. However, Thapsibiza is expected to exclusively provide *T. garganica* seeds to GenSpera at the current price of €300 per kilogram (kg). Thapsibiza can increase the price without notice to compensate for increased governmental taxes. As long as GenSpera continues to develop drugs derived from thapsigargin, the Company is required to purchase a minimum of 50 kg of *T. garganica* seeds per harvest period year.
Thapsigargin Production Technology Research

GenSpera’s research for G-202 is covered by patents and supported by laboratories at Johns Hopkins and the University of Copenhagen via academic grants awarded to the Company’s collaborators. The University of Copenhagen has received a $4.4 million grant from the Danish Research Council for Strategic Research to develop a metabolically engineered moss strain as a sustainable production platform for plant products. GenSpera is contributing a total of $100,000 to the grant. Production of thapsigargin has been selected as a pilot project called Sustainable Production of Thapsigargin using Light (SPOTLight). Dr. Craig A. Dionne, GenSpera’s chief executive officer, chief financial officer, president, and chairman, serves on the Advisory Board for SPOTLight. A positive outcome of GenSpera’s trials with a prodrug of thapsigargin (e.g., G-202) could create significant demand for thapsigargin. SPOTLight is focused on meeting this demand by developing a sustainable platform thapsigargin supply using only carbon dioxide, water, and sunlight with easily cultivated moss cells.

The National Cancer Institute’s Evaluation of Thapsigargin’s Effects

Thapsigargin has been included on the National Cancer Institute’s Developmental Therapeutics Program, an anticancer compound screening program designed to identify novel chemical leads and biological mechanisms. The National Cancer Institute’s experiments demonstrated that thapsigargin possesses broad growth inhibitory activity and is 10 to 100 times more potent than Bristol-Myers Squibb Co.’s (BMY-NYSE) Taxol® (paclitaxel) or doxorubicin, which served as the reference cytotoxic agents. A similar experiment published in the *Journal of the National Cancer Institute* in 2003 evaluated a broader panel of prostate cancer cell lines and other non-cancer cell types than the previous screening. Equivalent results were achieved, indicating that thapsigargin killed cells non-selectively, regardless of whether the test cells were normal, malignant, or of prostatic origin. Further, the data also showed that thapsigargin effectively killed both rapidly and very slowly dividing cells in contrast to Taxol® or doxorubicin, which diminished in efficacy when tested against these slower dividing cells. Figure 7 illustrates the ability of each of the different drugs to achieve apoptosis in high- or low-proliferation cell cultures that were exposed to the product for 48 hours.

Creating Prodrugs from Thapsigargin

In the past, thapsigargin has not been used as a cancer drug because its natural form is toxic to healthy body tissues in addition to cancer cells and tumors. Over the past 15 years, the members of GenSpera’s Scientific Advisory Board have focused on targeting the active drug specifically to the tumor in order to reduce or eliminate the damage to healthy cells and tissues. More specifically, the Company has
investigated ways to attach masking agents that render thapsigargin and its analogs temporarily nontoxic, while maintaining the ability to selectively remove the masking agent at the tumor site, thus activating the compound exactly where its toxic effects are more effective.

Important factors in the development of GenSpera's prodrug were determining how to attach the peptide to thapsigargin and identifying the best location for attachment. The laboratory of Dr. Samuel R. Denmeade (biography on page 12), one of GenSpera’s co-founders and currently the chief clinical advisor of the Company’s Scientific Advisory Board, discovered that GenSpera’s target enzymes—enzymes found primarily at tumor sites—were unable to remove the final amino acid of peptides attached directly to thapsigargin. Therefore, the development of a prodrug required the identification of a chemical derivative of thapsigargin that met several key requirements:

- was equally as effective as thapsigargin in killing cancer cells;
- could be chemically coupled to the desired peptide; and
- was able to regain its activity once it was exposed to the target enzyme at the tumor site.

12ADT

As GenSpera's targeting/masking agents cannot be directly attached to thapsigargin, the Company created 12ADT, a therapeutically active analog of thapsigargin that fulfills the key requirements listed above. 12ADT enables GenSpera to attach its targeting/masking agents to its proprietary compounds and is currently the chief therapeutic component of GenSpera’s products, including its lead prodrug candidate G-202. 12ADT functions by dramatically raising the level of calcium inside cells, which leads to cell death. It is the subject of the Company’s patent protection, as are the specific peptide sequences that GenSpera attaches to 12ADT (detailed below under “Targeting/Masking Agents”). GenSpera's two core patents—both of which are entitled “Tissue Specific Prodrug”—contain claims that cover the composition of 12ADT. In addition, while the creation of 12ADT is based on numerous years of research by members of GenSpera’s Scientific Advisory Board, the Company does not owe any milestone or royalty payments to third parties.

Targeting/Masking Agents

GenSpera uses peptides to target/mask its agents as they are delivered to the target site (tumors) within the body. Peptides are short strings of amino acids—the building blocks of many components found in cells. The Company selects peptides that specifically target 12ADT to tumors. When attached to 12ADT, GenSpera’s targeting/masking agent is able to temporarily mask the activity of 12ADT, essentially making it inactive. The agent also makes the prodrug highly soluble in blood, making intravenous administration possible. The Company’s technology capitalizes on the ability of the masking peptides to be removed by chemical reactors in the body called enzymes as well as the highly specific recognition of particular peptides by particular enzymes. As such, the peptide can only be removed by a specific enzyme found at the target (tumor) site. Once the peptide is removed, 12ADT returns to its naturally active, insoluble state and precipitates directly into the nearby cells. A set of GenSpera’s patents and patent applications cover the process by which the Company’s refined and specific targeting/masking peptides combine with 12ADT.

G-202: GENSPERA’S LEAD PRODRUG CANDIDATE

G-202 is a prodrug chemotherapy designed to treat cancer by selectively destroying the blood supply that supports solid tumors. GenSpera creates G-202 by chemically modifying thapsigargin to its equally active derivative, 12ADT, which is then coupled with a specific peptide that masks the activity of the drug until it is delivered to its target site. G-202’s protective peptide is preferentially removed by the prostate-specific membrane antigen (PSMA).
PSMA Expression

PSMA is a prostate differentiation antigen that is also expressed at minimal levels in a few specific cell types beyond the prostate. The antigen was first identified as a surface protein in normal (non-cancerous) prostate epithelium and in a large percentage of primary and metastatic prostate cancers. For this reason, PSMA is currently used as a marker for prostate cancer. ProstaScint®, a product marketed by EUSA Pharma (USA), Inc., is an approved agent that uses labeled antibodies to seek and bind to PSMA in order to identify both primary prostate cancer and its metastases.

While PSMA was originally thought to be specific to the prostate, it is now known to be expressed in the vasculature of many solid tumors (e.g., breast, colon, lung, prostate, etc.). As such, PSMA-targeted prodrugs may be able to attack many different tumor types. As such, GenSpera has chosen PSMA as the activating enzyme in G-202. To the Company’s knowledge, data to date has suggested that the expression of PSMA by endothelial cells is confined to tumor vasculature. Based on this information, the Company does not expect PSMA-targeted therapeutics to harm blood vessels in any non-cancerous tissues. Therefore, prodrugs that are PSMA-targeted may have a broader application because they are designed to specifically attack the blood supply of a large number of different tumor types, and thus have a clinical profile of a potent angiotoxic agent.

The Clinical Opportunity: Disrupting Tumor Blood Supply

Angiogenesis—the physiological process involving the formation of new blood vessels from pre-existing vessels—is a standard process in growth and development as well as in wound healing. However, angiogenesis is also the process by which a tumor may grow beyond a clinically insignificant size. Without the nutrition and oxygenation from a local blood supply, tumors cannot grow to be more than several millimeters in size.

Interrupting angiogenesis to tumors has been employed as a method to slow or potentially reverse tumor growth. For example, Roche Holding AG's Avastin®, an FDA-approved monoclonal antibody, inhibits the activity of vascular endothelial growth factor (VEGF), a substance that is important for the growth and survival of endothelial cells. Other approved drugs may also work in part via anti-angiogenesis, including Bayer HealthCare’s and Onyx Pharmaceuticals, Inc.’s (ONXX-NASDAQ) Nexavar®, Pfizer Inc.’s (PFE-NYSE) Sutent®, and Celgene Corporation’s Thalomid®—all of which are further detailed in the Competition section on pages 33-37. While these medicines may have limited therapeutic effects—with median patient survival times increasing only by several months—GenSpera anticipates that the aforementioned products may help clinically validate the therapeutic approach of disrupting blood supply to tumors in addition to confirming the market potential.

The sales of several currently marketed anti-angiogenic agents are shown in Table 6. Although these medicines are approved and have hundreds of millions to billion dollar sales—achievements that validate both the cellular target of the products as well as the market potential—the Company believes that there remains a vast window for improvement in therapeutic response. GenSpera’s approach, called tumor angiotoxicity, is designed to destroy both the existing and newly growing tumor vasculature versus solely blocking new blood vessel formation (as found in commercially available products). The Company anticipates that its technique may lead to a more immediate collapse of nutrient supply to the tumors, improving the rate of destruction at the tumor site.

<table>
<thead>
<tr>
<th>Company Name</th>
<th>Drug</th>
<th>Sales (2009)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Roche Holdings AG</td>
<td>Avastin®</td>
<td>$5.9 billion</td>
</tr>
<tr>
<td>Pfizer Inc.</td>
<td>Sutent®</td>
<td>$964 million</td>
</tr>
<tr>
<td>Celgene Corporation</td>
<td>Thalomid®</td>
<td>$437 million</td>
</tr>
<tr>
<td>Bayer HealthCare and Onyx Pharmaceuticals, Inc.</td>
<td>Nexavar®</td>
<td>$235 million</td>
</tr>
</tbody>
</table>

Sources: Crystal Research Associates, LLC and company websites.
Based on preclinical studies, the Company believes that G-202 has the characteristics of a favorable prodrug candidate. To be clinically successful, GenSpera believes that the preclinical candidate should possess certain attributes, as listed on the left side of Table 7. The right side of Table 7 lists the pharmacokinetics of G-202 in relation to the Company’s desired prodrug characteristics.

<table>
<thead>
<tr>
<th>Favorable Prodrug Characteristic</th>
<th>G-202 Pharmacokinetic Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>The prodrug is relatively stable in its inactive form in the bloodstream.</td>
<td>▪ The half-life (time for the initial concentration to be reduced by 50%) of the inactive prodrug in the bloodstream is 5.5 hours in mice and 10 hours in monkeys.</td>
</tr>
<tr>
<td>There is a high accumulation of the active drug in the tumor tissue, with very low concentrations in other tissues.</td>
<td>▪ Tumor tissue accumulates significant levels of the active form (12ADT) at 24 hours after the last dose, with less than a 50% decrease over the subsequent two days, and with relatively high levels still found after five days. ▪ Only very low concentrations of 12ADT were found in other tissues, such as the kidney, skeletal muscle, or brain.*</td>
</tr>
<tr>
<td>There are low concentrations of the active form of the drug in the bloodstream relative to the inactive prodrug.</td>
<td>▪ The concentration of 12ADT in the bloodstream never exceeded 1% of the concentration of G-202 (the inactive prodrug) over a 16-hour period.</td>
</tr>
<tr>
<td>The difference in toxicity between the inactive prodrug and its corresponding active drug should be as high as possible.</td>
<td>▪ 12ADT is toxic to mice at over 1,000-fold lower doses than the therapeutic doses of G-202.</td>
</tr>
<tr>
<td>The activated drug is actively taken up by adjacent cancer cells for a “bystander” killing effect.</td>
<td>▪ Target enzymes were selected for their activity on the outer surface of cells. Hence, the activated drug is free to kill any cells within its immediate vicinity.</td>
</tr>
</tbody>
</table>

*Higher concentrations were found in the liver without apparent toxicity, which the Company believes is most likely a part of the liver’s normal function in clearing cytotoxic drugs from the bloodstream.

Source: GenSpera, Inc.

In summary, the Company believes that G-202 exhibits favorable characteristics as a prodrug candidate. G-202 is stable in its inactive form in the bloodstream and, therefore, dosing levels and the frequency of infusions are easily controlled. In addition, G-202 has demonstrated the ability to target tumors with minimal leakage of the active form into the bloodstream or other tissues. As such, the dosing levels of G-202 could be increased, if necessary. G-202 may also not require combination with other therapies, which could permit faster and less costly clinical trials.

**Anti-tumor Efficacy Studies of G-202**

GenSpera has conducted several studies using G-202 in a variety of animal models with solid tumors, including prostate, bladder, renal (kidney), and breast cancers. In these studies, the administration of G-202 stopped tumor growth—and in most cases caused noticeable regression of the tumor—with no visible re-growth for roughly one month following the last treatment. G-202 was well tolerated at dose levels that caused cessation of tumor growth with no signs of toxicity. At the highest doses, transient weight loss was documented, which quickly recovered after each course of therapy. In addition, the data indicated that even after several dosing cycles, G-202 did not activate drug resistance in tumor cells. The Company has also completed definitive toxicology studies of G-202 in rats and monkeys.
G-202’s Anti-tumor Activity Against Prostate Cancer

To initially determine whether G-202 could serve as an anti-tumor agent in live animals, GenSpera evaluated the agent for activity in the LNCaP mouse xenograft model. LNCaP cells express PSMA at roughly the same level as found in a human prostate. When administered via intravenous injection at a dose of 17 mg/kg daily for four days, G-202 nearly ceased tumor growth. G-202 was also well tolerated with no signs of morbidity or toxicity during the study.

The Company subsequently evaluated G-202 for anti-tumor efficacy against the CWR22R-H human prostate tumor xenograft, which expresses higher levels of PSMA than LNCaP tumors. Mice with CWR22R-H tumors were treated with G-202 at either 17 mg/kg for 10 consecutive days or 56 mg/kg for three consecutive days. Both treatment regimens inhibited tumor growth for approximately 32 to 40 days before re-growth was detected. In addition, between the two different regimens, no significant difference in anti-tumor response was observed. The anti-tumor effect of the more intense dosing regimen, which is shown in Figure 8, was accompanied by weight loss (maximum of roughly 17%), which resolved and reached control values by day 27. The lower-intensity dosing regimen of G-202, which was equivalent to the total dose of the 56 mg/kg regimen, was also accompanied by a marginal (<10%) but rapidly reversible weight loss. The results of these two studies demonstrated that G-202 could produce significant anti-tumor effects at doses that were well tolerated by mice.

GenSpera’s next study was designed to evaluate G-202 for its ability to exert extended anti-tumor activity after several administration cycles at high dose levels. Mice with CWR22R-H were treated with four courses of G-202 intravenously at a dose of 56 mg/kg for three consecutive days. Each treatment course occurred at 10-day intervals. The data from the study demonstrated that the dosing regimen significantly inhibited tumor growth over the course of therapy from day 7 until the experiment was terminated on day 69. Similar to the previous experiment with 56 mg/kg dosing levels, tumor growth became apparent roughly 20 days after the last treatment. In addition, the mice also temporarily lost weight (maximum of approximately 12%), which recovered quickly after each course of therapy.

Based on the preliminary findings, which evaluated established but small tumors, GenSpera could not determine whether G-202 caused tumor regression or merely extended a stagnant growth period. As such, the Company also evaluated the ability of G-202 to cause tumor regression in animals with relatively large CWR22R-H tumors that had re-grown after the discontinuation of treatment with GenSpera’s related PSMA-targeted produg, 12ADT-Asp-Glu (also called G-201). In this study, G-202 was administered intravenously at a dose of 56 mg/kg for three separate cycles of dosing: days 1-3, days 16-18, and days 26-28. Data indicated that each dosing cycle caused noticeable regression of the large tumors, suggesting that G-202 exerts cytotoxic and tumor-reducing effects in vivo. In addition, the tumors did not manifest a resistance to the drug, even after multiple dosing cycles.
G-202’s Anti-tumor Effect Against Human Bladder, Kidney, and Breast Cancers

Some researchers have demonstrated PSMA expression in the new vasculature of a variety of tumor types, including renal, bladder, colon, neuroendocrine, pancreatic, lung cancers, and the majority of breast cancers and sarcomas. Prostate cancer is not included because research has shown that endothelial cells within prostate cancers do not appear to make PSMA (Source: Cancer Research 1999; 59: 3192-3198). A report published in 2006 demonstrated that PSMA may serve as an interface on the molecular level, coordinating both extracellular and intracellular signals during angiogenesis (Source: Molecular and Cellular Biology 26(14):5310-5324). In this study, PSMA knockout mice had marked impairment of new blood vessel formation, indicating that G-202 may also be effective against non-prostate cancer tumors due to its ability to selectively destroy PSMA-producing endothelial cells present within the tumor.

Based on these findings, the Company evaluated the anti-tumor effect of G-202 in a mouse model of human bladder cancer by administering three consecutive daily 56 mg/kg doses of G-202 to nude mice bearing TSU-Pr1 xenografts for four cycles. The treated mice exhibited significant inhibition of tumor growth versus vehicle-treated controls, which received vehicle alone—15% solutol/15% propylene glycol in phosphate-buffered saline. The treated versus control (T/C) ratio of the tumor volume on day 25 was 0.33. By day 33, the tumor volume in six of the eight treated control animals was greater than 0.5 cc, while all but one of the animals treated with G-202 was less than 0.5 cc. Calculated from the ratios of measured tumor volume at day 33 to starting tumor volume for each individual animal, the relative volume of control tumors was 13.2 versus 3.2 for G-202-treated tumors, as shown in Figure 9.

In addition, GenSpera evaluated G-202 for anti-tumor efficacy in a mouse model of human renal (kidney) cancer. Five consecutive daily doses of 56 mg/kg were given to nude mice for nearly three courses of treatment. In total, 14 doses were administered, as indicated by the downward facing arrows shown in Figure 10 (page 28). As displayed in Figure 10, the data from this study demonstrated tumor regression when contrasted with the size of the tumor at the beginning of the experiment. After the third course of G-202 treatment, the treated versus control (T/C) ratio on day 30 was 0.12 versus vehicle controls. By day 30, the group treated with G-202 experienced a 75% reduction in overall tumor size. The experiment was also repeated at a dosing level of 5.6 mg/kg (one-tenth of the original dose) on the same dosing schedule. These results further validated the anti-tumor effects of G-202—even at a significantly lower dose—relative to vehicle controls, with a T/C ratio of 0.57 on day 30.
G-202 was also tested using a human breast tumor model in nude mice to evaluate its anti-tumor effects. Only two consecutive daily doses of 56 mg/kg G-202 were administered to the mice. This therapy resulted in complete regression in six out of eight treated animals, indicating that 75% of animals may have been cured with only one cycle of dosing. Tumors that re-grow can be treated with repeated dosings resulting in permanent tumor regressions. The data from this study is shown in Figure 11.

It is important to note the broad therapeutic safety window for G-202. In the human renal cancer model, 14 daily doses of drug were administered to mice, as illustrated by downward facing arrows in Figure 10. Nevertheless, the Company anticipates that only two doses may be necessary as demonstrated in the human breast cancer model, potentially allotting a safety window that the Company believes is significant for cancer therapeutics.
Summary of Data

The anti-tumor effects of G-202 on the growth of breast, renal, bladder, and prostate cancer xenografts indicate that G-202 may have broad application as a therapy for a variety of human solid tumors due to its ability to selectively target PSMA-producing endothelial cells within these tumors. Further, the effects are achievable at doses of G-202 that are well tolerated in mice. GenSpera believes that it may be able to eliminate cancerous tumors with dosing that is effective for an extended length of time. Further, the Company also anticipates that this is more likely to be achieved in humans than in mice due to the ability to infuse the drug and a potentially longer half-life of G-202 in the human bloodstream versus that of laboratory animals. Given its efficacy profile, GenSpera also expects that G-202 will likely be effective as a monotherapy, thus reducing the costs and time required to conduct clinical trials.

Development Status

In September 2009, GenSpera announced that the FDA approved its IND application to initiate a Phase I study with G-202 in cancer patients. The ongoing study (overviewed below) is designed to evaluate its safety and pharmacokinetics in humans, and determine an appropriate dosing regimen for subsequent clinical studies. Assuming successful completion of the Phase I clinical trial, GenSpera expects to conduct up to four Phase II clinical trials to determine the therapeutic efficacy of G-202 in various cancer types.

Phase I Clinical Study

The Phase I trial is ongoing at two locations: (1) the University of Wisconsin Carbone Cancer Center in Madison, Wisconsin, with Dr. George Wilding as principal investigator; and (2) the Sidney Kimmel Comprehensive Cancer Center at Johns Hopkins, Baltimore, Maryland, for which Dr. Michael Carducci is serving as principal investigator. GenSpera is conducting this study in cancer patients with any type of solid tumor who have relapsed after treatment with other anticancer agents—a strategy that is expected to facilitate enrollment and provide safety data across a variety of cancer patients. While the primary endpoints of the open-label, dose-escalation study are to evaluate the safety, tolerability, and pharmacokinetics of the drug in humans, the design of the trial also allows the collection of efficacy data.

In January 2010, GenSpera treated the first patient at the University of Wisconsin Carbone Cancer Center. The study is progressing according to a predetermined schedule (in terms of dose escalation) agreed to by the FDA. In the first cohort, participants enroll one after another, with each patient requiring roughly six weeks. GenSpera seeks to enroll up to 30 patients in the Phase I study. As of June 2010, four patients had been enrolled in the study. The Company expects to complete the trial in the second quarter 2011.

Planned Phase II Clinical Program

Dependent upon the successful completion of the Phase I study, GenSpera plans to conduct up to four Phase II clinical trials over 18 months to determine the therapeutic efficacy of G-202 in different tumor types. Each trial is expected to focus on a single tumor type—a strategy that the Company believes can maximize efficiency. The Phase II program requires additional G-202 product, which could take approximately one year and $1.5 million to produce.

GenSpera plans to license G-202 during the Phase II program. In the event that the Company is unable to do so on acceptable terms, it will likely proceed with Phase III clinical trials.

Clinical Strategy

Unlike the first generation of angiogenic drugs, which only blocked new vessel formulation, GenSpera believes that its approach destroys the existing tumor vasculature, collapsing the tumor’s nutrient supply and destroying the tumor at a more rapid rate. Because G-202 is capable of attacking the blood supply to a variety of solid tumors, the Company believes that there is a wide range of clinical applications for G-202, including those overviewed in Table 8 (page 30). The Company expects to continue to evaluate G-202 in other preclinical animal models of various cancers in addition to evaluating other potential clinical opportunities as human patient data is collected in early clinical trials.
GenSpera believes that G-202 could have activity across a wide variety of solid tumors. The Company plans to evaluate G-202 in multiple cancer types, creating value for potential corporate partners by demonstrating breadth of activity.

Manufacturing and Development

To leverage GenSpera’s experience and available financial resources, the Company does not intend to develop its own manufacturing facilities. Rather, GenSpera plans to outsource all drug manufacturing to a Good Manufacturing Practice (GMP)-compliant contract manufacturer. GenSpera entered into an Alliance Agreement with Cedarburg Hauser Pharmaceutical Services to perform most of the Company’s contract manufacturing efforts. Under the terms of this agreement, independent work orders have been and will likely continue to be constructed for various tasks, including manufacture of chemical intermediates and reference standards, manufacture of G-202 in compliance with GMP standards, and development of analytical methods in support of GenSpera’s development programs. The Company plans to continually improve its efficiency by refining its current manufacturing process as well as the final drug formulation to simplify storage and related procedures. GenSpera has identified a viable formulation of G-202 for the clinic and for commercial purposes. The Company has sufficient G-202 to supply the Phase I clinical trial.

GenSpera believes that its current stockpile of *T. garganica* seeds can generate 2.5 kg of drug product, and as such, the Company is in possession of sufficient material to take G-202 through late-stage clinical testing.

Other Potential Business Applications for G-202

In addition to chemotherapy, GenSpera believes that G-202 has several characteristics that may be beneficial in other business applications, including prostate cancer and benign prostatic hypertrophy (BPH). BPH is a condition in older men that is characterized by an enlarged prostate in addition to urinary problems. GenSpera anticipates that the size of the prostate gland may be reduced when G-202 is administered to treat tumors because PSMA is found within both the healthy and enlarged prostate. This potential size reduction may have a positive effect by relieving symptoms associated with prostate cancer and BPH. Approximately one in four men over the age of 40 experience BPH symptoms (Source: Boehringer Ingelheim Limited). The American Urological Association (AUA) estimates that BPH is prevalent in over 50% of men above age 60, a figure that increases to 90% by age 85 (Source: the AUA’s Clinical Guidelines on the Management of BPH 2003). The potential utility of G-202 in the treatment of BPH is closely related to its toxicity profile in humans. While not currently a primary business focus, this potential effect is expected to be closely monitored in early clinical trials.

DEVELOPING PRODRUGS TO TARGET TUMORS DIRECTLY

GenSpera’s technology has a broad range of potential applications, including the pairing of cytotoxic derivatives of thapsigargin to peptides that are only removable by enzymes expressed in a particular tumor type. The laboratories of Dr. John T. Isaacs and Dr. Denmeade are working to develop other lead compounds that specifically release thapsigargin within prostate tumors. Biographies for Dr. Isaacs and...
Dr. Denmeade, who are co-founders of GenSpera presently serving as Scientific Advisory Board members, are provided on page 11 and page 12, respectively. The Company’s first approach coupled thapsigargin to peptides that were selectively cleaved by a prostate cancer-specific protease called prostate-specific antigen (PSA), which led to GenSpera’s development candidate, G-115. The Company’s second program entails the delivery of thapsigargin selectively to prostate tumors via human glandular kallikrein 2 (hK2), a distinct prostate cancer-specific protease. The hK2 program has resulted in several molecules with anticancer properties in animal models, and the Company is currently using peptide optimization to identify a legitimate development candidate.

PSA-targeted Prodrugs for Prostate Cancer

Compared to other cancers, prostate cancer has a large proportion of slowly proliferating cells that are resistant to treatment with conventional cytotoxic agents. Currently available therapies generally attack and destroy rapidly dividing cells, as opposed to cells that divide slowly. The side effects associated with the present therapies are severe because cell division is also a frequent and normal process of healthy non-malignant tissues. Therefore, to develop an efficacious drug, it is important to use an agent that is capable of killing slowly proliferating cancer cells and can be delivered in a way that minimizes the toxicity to other parts of the body. GenSpera believes that its core technologies accomplish these two primary objectives.

PSA is active within tumor sites and in the normal prostate tissue but is inactive in the bloodstream—characteristics that form the basis for tumor-specific delivery of cytotoxic agents. While PSA is not active in the bloodstream, its level is measurable and is used as a clinical test to detect prostate cancer and follow response to therapy.

To develop a PSA-activated prodrug, GenSpera focused on the identification of a derivative of thapsigargin that could be chemically coupled to a PSA-substrate peptide, yet retain all the activity of the parent agent after it was released from the prodrug by PSA. The Company focused on the peptide’s synthetic feasibility, optimization of its activity as a PSA substrate, and its relative stability to cleavage by other proteases that are common in the body. Adhering to these conditions, GenSpera identified an initial PSA-activated prodrug, G-114. However, continued screening and optimization of PSA-cleavable peptides led to the discovery of G-115, an improved prodrug that the Company believes exhibits 10-fold greater activity as a PSA substrate versus G-114. G-114 has been designated as the back-up drug for G-115 due to its significant activity as an anti-tumor agent in animal models of prostate cancer.

G-115

G-115 is a prodrug that is selectively activated within prostate tumors by PSA. GenSpera chose G-115 as its lead development candidate in the PSA-targeted prodrug program due to its enhanced PSA-substrate and in vivo anti-tumor activity, as well as its broader intellectual property (IP) coverage. To evaluate G-115’s anti-tumor response, mice bearing a high PSA-producing human prostate cancer xenograft were treated with 7 mg/kg for 10 consecutive days. The data from the study, shown in Figure 12, demonstrated that G-115 inhibited tumor growth and exhibited well-behaved pharmacokinetics in mice.

GenSpera plans to obtain an IND for G-115 in the third quarter 2011. The Company seeks to establish a strategic partnership to maximize the value of G-115 as it progresses in the clinic. It is important to note that G-115 and G-202 are non-competing product candidates: the Company intends to market G-115 to urologists, while marketing G-202 to medical oncologists. In the U.S. and many counties in Europe, urologists are the first medical professionals to treat prostate cancer patients. When their methods and tools fail, the patient is often recommended to a medical oncologist.

Source: GenSpera, Inc.
Prostate cancer cells also secrete the protease hK2, which may be used to activate thapsigargin prodrugs. Through the laboratories of Dr. Isaacs and Dr. Denmeade, GenSpera has identified peptides that are selectively cleaved by hK2. The Company has coupled them to 12ADT to generate a family of hK2-activated thapsigargin prodrugs, including Ac-GKAFRR-L12ADT, which GenSpera considers to be the most fully characterized prodrug of this species. In 2006, the data obtained from Ac-GKAFRR-L12ADT were published in *The Prostate*. Ac-GKAFRR-L12ADT demonstrated an anti-tumor effect in animal models of prostate cancer while it was being administered. However, the prodrug was rapidly cleared from the body and there was a moderately low range of therapeutic doses relative to toxicity. Presently, the Company is focused on developing second-generation hK2-activated thapsigargin prodrugs with improved formulations and increased half-lives.
Competition

The pharmaceutical, biopharmaceutical, and biotechnology industries are highly competitive. While GenSpera is not aware of any medicine in development that is designed to destroy both existing and newly grown tumor vasculature in a manner similar to G-202, the Company may face competition from other companies that are marketing or developing agents that function, at least in part, by attacking tumor-associated blood vessels (e.g., OXiGENE, Inc.’s ZYBRESTAT™ or Roche’s Avastin®, which are overviewed on page 36 and page 37, respectively). GenSpera believes that the already-approved anti-angiogenic products validate the concept of targeting tumor-associated endothelial cells as a commercially viable cancer therapy.

Nevertheless, it is important to note that the molecular mechanism of G-202’s cytotoxicity is novel among anticancer agents as it inhibits the activity of the SERCA pump to cause cell death (as detailed on page 21). Further, G-202 as a monotherapy has led to tumor regression in animal models of cancer versus existing anti-angiogenic agents that may only be able to prevent further tumor growth. Currently marketed drugs have been approved based upon anti-tumor effects demonstrated in the clinic. However, a significant unmet need persists for more effective anticancer agents that significantly extend patient survival in addition to having anti-tumor effects. As well, to the Company’s knowledge, it is the only entity with a targeting mechanism based upon the enzymatic activity of PSMA. GenSpera believes that this is, in part, due to the substrate specificity of the enzyme as well as the Company’s solid patent position in this area.

GenSpera may also compete with large, well-funded companies that are developing or have developed drug candidates of a different type that address the same patient populations. While not an exhaustive collection of GenSpera’s competitors, Table 9 lists the companies that are believed to be representative of the type of competition that the Company may face.

<table>
<thead>
<tr>
<th>Company</th>
<th>Symbol (Exchange)</th>
<th>Last Trade (07/06/10)</th>
<th>52-week Range</th>
<th>Avg Vol (3 month)</th>
<th>Market Cap</th>
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<td>Access Pharmaceuticals, Inc.</td>
<td>ACCP (OTC.BB)</td>
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<td>—</td>
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<td>Bayer HealthCare (part of Bayer AG)*</td>
<td>BAYN (XETRA)</td>
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<td>€35.36 - €56.71</td>
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<td>$12.92B</td>
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<tr>
<td>Biotest AG*</td>
<td>BIO (XETRA)</td>
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<td>€31.09 - €47.49</td>
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<td>234.13M</td>
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<td>F. Hoffmann-La Roche Ltd**</td>
<td>ROG (SWX)</td>
<td>CHF 146.80</td>
<td>CHF 143.70 - CHF 187.40</td>
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* Amounts in euros; €1 = US$1.26 at 07/06/10. ** Amounts in Swiss francs; CHF 1 = US$0.94 at 07/06/10.

Sources: Yahoo! Finance, Google Finance, and Crystal Research Associates, LLC.
Access Pharmaceuticals, Inc. (www.accesspharma.com)

Access Pharmaceuticals is a Texas-based biopharmaceutical company that develops and commercializes products for the treatment and supportive care of cancer patients. Access Pharmaceuticals’ pipeline includes ProLindac™, an oncology drug that is currently in Phase II clinical testing in Europe with ovarian cancer patients. ProLindac™ is a novel DACH platinum prodrug that has been shown in both preclinical models and in human trials to be active in a wide variety of solid tumors, and may eliminate some of the toxic side effects seen in currently marketed DACH platinum medicines. ProLindac™ has completed a Phase II monotherapy study in ovarian cancer patients. Also in development is thiarabine (4-thio Ara-C), a small molecule therapy licensed from the Southern Research Institute in Birmingham, Alabama, that has been evaluated in two Phase I studies in patients with solid tumors. Based on preclinical and clinical data to date, Access Pharmaceuticals plans to further investigate thiarabine in leukemia and lymphoma patients.

ACT Biotech, Inc. (www.actbiotech.com)

ACT Biotech is a closely held, California-based pharmaceutical company focused on the development and commercialization of targeted cancer drugs. Its lead program is the development of Telatinib, a small molecule angiogenesis inhibitor. Data from over 250 patients involved in two Phase I monotherapy trials and four combination therapy trials showed signs of efficacy in various cancer types and improved safety versus other VEGF or VEGF receptor-directed anti-angiogenic drugs. In June 2010, Telatinib received Orphan Drug designation from the FDA for the treatment of gastric cancer. Telatinib is currently in Phase II clinical testing in the U.S. and Europe for the first-line treatment of advanced gastric cancer patients in combination with standard-of-care chemotherapy for which results are expected in late 2010. A second ACT Biotech program entails the development of ACTB1003, an oral kinase inhibitor with multiple modes of action, which targets cancer by inhibiting angiogenesis, inducing tumor cell apoptosis, and targeting specific cancer mutations. The company’s IND application for ACTB1003 was accepted by the FDA in early 2010, and ACT Biotech is preparing for Phase I clinical trials in multiple cancer indications.

Bayer HealthCare (www.bayer.com)

Bayer HealthCare, the U.S.-based pharmaceuticals unit of Bayer HealthCare LLC (a division of Bayer AG), has collaborated with Onyx (detailed on page 36) to develop and market Nexavar® (sorafenib) in tablet form. Nexavar® is a multi-kinase inhibitor indicated for the treatment of advanced renal cell carcinoma, the most common type of kidney cancer, and for unresectable hepatocellular carcinoma, the most common form of liver cancer. In advanced renal cell carcinoma patients, Nexavar® demonstrated improved progression-free survival from 12 weeks to 24 weeks. The active ingredient in Nexavar®, sorafenib, targets both the tumor cell and tumor vasculature. Nexavar® is approved in over 80 countries for liver cancer and in more than 90 countries for advanced kidney cancer. Nexavar® is also being evaluated as an individual or combination therapy in lung, thyroid, breast, and colorectal cancer, among others.

Biogen Idec Inc. (www.biogenidec.com)

Headquartered in Massachusetts, Biogen Idec is a biotechnology company engaged in the development, manufacturing, and commercialization of novel therapies. Under a collaboration agreement, Biogen Idec and Genentech, Inc. (a wholly owned member of the Roche Group) are developing and marketing Rituxan® (rituximab), a targeted B-cell therapy that is indicated for the treatment of non-Hodgkin’s lymphoma. Rituxan® is approved as a single agent or a combination therapy for treatment of select stages and forms of B-cell non-Hodgkin’s lymphoma and as part of a combination therapy to treat chronic lymphocytic leukemia. The company’s lead development candidate is a humanized anti-CD20 monoclonal antibody called GA101, which is being developed under a collaboration agreement with Genentech. GA101 is in Phase III clinical trials for chronic lymphocytic leukemia and Phase II testing for non-Hodgkin’s B-cell lymphoma. Biogen Idec is also developing a candidate with technology licensed from ImmunoGen, called BIIB015, for the treatment of solid tumors. BIIB015 has ImmunoGen’s cell-killing agent, DM4, attached. Phase I clinical testing with BIIB015 is ongoing.
Biotest AG (www.biotest.com)

Biotest is a German pharmaceutical company that specializes in hematology and immunology products. Through the licensing of technology from ImmunoGen, Biotest is currently developing BT-062, a Tumor-Activated Prodrug (TAP) compound for the treatment of multiple myeloma. BT-062 comprises a monoclonal antibody and ImmunoGen’s cell-killing agent, DM4. In early 2008, BT-062 was granted Orphan Drug status by the FDA, and in December 2008, the European Commission also granted BT-062 Orphan Drug designation for multiple myeloma after the compound was assessed by the European Medicines Agency (EMEA). BT-062 is currently undergoing Phase I clinical trials at four cancer clinics in the U.S. to test the safety and tolerability of the compound for use in humans, as well as to provide initial information on its efficacy. Biotest is also making preparations to extend the clinical development program to Europe.

Celgene Corporation (www.celgene.com)

New Jersey-based Celgene is a multinational biopharmaceutical company that aims to improve the lives of patients worldwide. Celgene currently markets Thalomid® (thalidomide), an orally administered immunomodulatory agent. Thalomid® has been shown to have anti-angiogenic activity. Thalomid® is indicated for the treatment of patients with newly diagnosed multiple myeloma (cancer of the plasma cell) when used in combination with dexamethasone, a steroid that reduces inflammation and swelling. As well, Celgene is developing Revlimid® (lenalidomide), which is approved in combination with dexamethasone to treat multiple myeloma patients who have received at least one prior therapy. Revlimid® is also in clinical testing for chronic lymphocytic leukemia, non-Hodgkin’s lymphoma, and solid tumors, among other indications. Celgene plans to complete a Phase III trial with its lead compound for small cell lung cancer, Amrubicin, in 2010.

Genentech, Inc. (www.gene.com)

Headquartered in San Francisco, California, Genentech is a biotechnology company that focuses on the discovery, development, manufacture, and commercialization of medicines for patients with significant unmet medical needs. Genentech is a wholly owned member of the Roche Group and serves as the headquarters for all Roche pharmaceutical operations in the U.S., including marketing products such as Avastin® (bevacizumab), a bioengineered antibody for treating various types of colon, breast, lung, and other cancers (described in greater detail on page 37). Genentech’s pipeline includes two Phase II oncology candidates: (1) ABT-263, a small molecule in Phase I clinical trials in combination with other medicines to treat solid tumors and hematologic malignancies and in Phase I/II clinical trials as a single agent for various forms of cancer; and (2) MetMAb, a humanized monoclonal antibody in Phase II clinical testing in combination with Tarceva® (erlotinib) for second- and third-line metastatic non-small cell lung cancer.

ImmunoGen, Inc. (www.immunogen.com)

Headquartered in Massachusetts, ImmunoGen is focused on the development of targeted anticancer therapeutics. ImmunoGen’s pipeline is primarily created from the company’s proprietary TAP technology, which was designed to facilitate the development of anticancer drugs that are more effective and better tolerated than currently available therapies. A TAP compound consists of a monoclonal antibody that binds specifically to an antigen found on cancer cells that is attached to a potent cell-killing agent. ImmunoGen has several TAP compounds in human clinical testing: (1) BT-062, which is in Phase I for the treatment of multiple myeloma; and (2) IMGN388, a TAP compound that uses an integrin-targeting antibody developed by Centocor, Inc. (part of Johnson & Johnson [JNJ-NYSE]), which is in Phase I clinical testing for the treatment of solid tumors. ImmunoGen has also licensed rights to develop products using its TAP technology to several major biotechnology and pharmaceutical companies, including Genentech, sanofi-aventis, Biogen Idec, Biotest, Bayer HealthCare, and Amgen, Inc. (AMGN-NASDAQ).
Ipsen S.A. (www.ipsen.com)

Headquartered in Paris, France, Ipsen is a global biopharmaceutical group focused on specialty and primary care drugs in oncology, endocrinology, neurology, and hematology, among others. The company is supporting over 20 research and development programs, including several in the oncology arena: (1) Decapeptyl®, a Phase III hormone therapy for breast cancer; (2) Toremifene citrate, a Phase III candidate being developed to treat and prevent prostate cancer; and (3) BN 83495 (STX 64), a Phase I therapy for post-menopausal breast cancer expressing estrogenic receptors, among others. Ipsen's pipeline also includes several preclinical anticancer candidates, such as Angiomates (STX 140), a family of small molecules that exhibit both anti-angiogenic and anti-proliferative (killing cancer cells) properties.

Merck & Co., Inc. (www.merck.com)

With headquarters in Whitehouse Station, New Jersey, Merck is a global pharmaceutical company developing medicines, vaccines, biologic therapies, and consumer and animal products. Its oncology program entails products for the prevention, treatment, and supportive care of cancer. The company aims to be the leader in the discovery, development, and delivery of targeted anticancer therapies and is also pursuing cancer vaccines. Merck’s targeted oncology candidates focus on key pathways and processes involved in cancer growth and progression. The company has two Phase III cancer candidates: (1) Ridaforolimus (MK-8669, AP23573), an oral inhibitor of mTOR—a protein involved in regulating normal cell growth, division, and survival as well as new blood vessel formation—to treat soft tissue sarcoma; and (2) V503, a vaccine to prevent nine types of human papillomavirus (HPV).

Onyx Pharmaceuticals, Inc. (www.onyx-pharm.com)

Onyx is a California-based biopharmaceutical company that seeks to improve the lives of cancer patients. In addition to the collaboration with Bayer HealthCare for the development and marketing of Nexavar®, Onyx is also developing carfilzomib, a proteasome inhibitor that is being evaluated in multiple clinical settings, including an ongoing Phase IIb trial in patients with relapsed and refractory multiple myeloma, a Phase II trial in patients with relapsed and/or refractory multiple myeloma who have relapsed after one to three prior therapies, and a Phase Ib/II study in patients with solid tumors. Onyx's pipeline also includes PD 0332991, a cell cycle kinase inhibitor in Phase II clinical development with Pfizer, Inc., and ONX 0801, an inhibitor of thymidylate synthase (TS), a crucial enzyme for cell growth and division. ONX 0801 functions through a combination of two approaches: (1) receptor-mediated targeting of tumor cells; and (2) inhibiting TS. ONX 0801 is currently in Phase I clinical testing. Onyx has an exclusive worldwide license to ONX 0801, which was originally discovered at the Institute of Cancer Research in London, called BGC 945. Onyx has also acquired an option to license rights to ONX 0803, an orally available, potent, and selective inhibitor of Janus kinase 2 (JAK2), and ONX 0805, also a JAK2 inhibitor, from Singapore-based S*BIO Pte Ltd. JAK2 has been implicated across a broad range of conditions, including cancer and autoimmune diseases, and may be used as a potential target in next-generation cancer therapies. The agreement grants Onyx option rights to exclusively develop and commercialize the compounds for any potential indication in the U.S., Canada, and Europe.

OXiGENE, Inc. (www.oxigene.com)

OXiGENE is a biopharmaceutical company based in Massachusetts that aims to develop therapeutics for patients with cancer and sight-threatening eye diseases and conditions. OXiGENE is developing several pipeline candidates based on vascular disruption technology, which aims to deprive solid tumors of their blood supply via vascular-disrupting agents (VDAs). In contrast to anti-angiogenic therapies, which prevent the formation of new blood vessels, VDAs disrupt newly formed blood vessels by rapidly reducing blood flow to the tumor, therefore depriving it of oxygen and nutrients that are necessary for survival. OXiGENE’s VDAs include ZYBRESTAT™, which is in clinical trials for anaplastic thyroid cancer (Phase II/III), non-small cell lung cancer (Phase II), and ovarian cancer (Phase II), as well as OXi4503, which is currently being evaluated as a monotherapy in patients with advanced solid tumors in a Phase I dose-escalation study. Nevertheless, data from preclinical studies executed by OXiGENE and its scientific collaborators have shown that OXiGENE’s VDAs may work synergistically with select anti-angiogenic drugs to produce anti-tumor effects.
Pfizer Inc. (www.pfizer.com)

Headquartered in New York, Pfizer is a research-based biomedical and pharmaceutical company. While Pfizer markets treatments for a variety of cancers, one of its more recent developments is its angiogenesis inhibitor, Sutent®, which is administered orally via tablet. By blocking the actions of VEGF, Sutent® is able to cut off the blood supply that feeds tumors and destroys cellular reproduction. Sutent® is indicated for the treatment of advanced gastrointestinal tumors and advanced renal cell carcinomas. In addition, Sutent® is currently involved in Phase II testing for gastric and colorectal cancer and Phase III clinical trials for various other cancers with unmet medical needs, including breast, lung, liver, and prostate cancers.

Roche Holdings AG (www.roche.com)

Headquartered in Basel, Switzerland, Roche is a global healthcare company with products addressing oncology, virology, inflammation, metabolism, the central nervous system, in vitro diagnostics, tissue-based cancer diagnostics, and diabetes, among others. Roche acquired Genentech as well as its pipeline in 2009, including Avastin®. Approved by the FDA in February 2004, Avastin® is considered to be the first FDA-approved therapy for an angiogenesis inhibitor. Avastin® is designed to specifically restrain vascular endothelial growth factor (VEGF), a potent protein that is believed to be a critical element of a tumor's ability to grow and spread in the body. While Avastin® has not demonstrated activity as a monotherapy, it has been indicated for colon and lung cancer when administered intravenously following chemotherapy. Avastin® has been shown to increase progression-free survival by roughly four months for carcinoma of the colon or rectum, and by approximately two months for unresectable, locally advanced, recurrent or metastatic non-squamous, non-small cell lung cancer. Moreover, Avastin® is also FDA-approved as a breast cancer treatment, although there is currently no data supporting a reduction in disease-related symptoms or an increased survival rate for this indication. Several serious side effects are associated with Avastin®, including gastrointestinal perforation, slow wound healing, and severe bleeding, among others.

sanofi-aventis SA (www.sanofi-aventis.com)

Headquartered in Paris, France, sanofi-aventis is a pharmaceutical group engaged in the research, development, manufacture, and marketing of medicines, vaccines, and integrated healthcare solutions. The company is developing product candidates in various therapeutic areas, including oncology, cardiovascular disease, metabolic disorders, thrombosis, central nervous system, internal medicine, ophthalmology, and vaccines, among others. In the oncology arena, sanofi-aventis has six candidates in late phases of development. Of these, two have Fast Track status from the FDA: (1) cabazitaxel, a tubulin inhibitor, for the second-line treatment of prostate cancer; and (2) BSI-201, a PARP-1 inhibitor, to treat triple negative metastatic breast cancer. Cabazitaxel is a new molecular entity in the registration phase. Additionally, using ImmunoGen’s TAP technology, sanofi-aventis is developing SAR3419, which is in Phase I clinical testing for the treatment of non-Hodgkin’s lymphoma. Currently in Phase I studies, SAR3419 is a compound that combines a CD19-targeting monoclonal antibody and a toxic substance called maytansinoid DM4.
Milestones

Recent Milestones

GenSpera has achieved several significant milestones in the past 12 months, as listed below.

- Received FDA approval for its IND application for G-202 in September 2009
- Began trading on the Over-the-Counter Bulletin Board (OTC.BB) under the symbol “GNSZ” in November 2009
- Commenced the Phase I clinical study with G-202 at both the Sidney Kimmel Comprehensive Cancer Center at Johns Hopkins and the University of Wisconsin Carbone Cancer Center
- Treated the first patient in the Phase I study with G-202 in January 2010
- Raised roughly $2.7 million in gross proceeds in May 2010

Potential Milestones

The Company has identified the following key milestones that it aims to achieve in the next several years relating to its product candidates, as summarized below.

- Complete the Phase I clinical trial with G-202 in the second quarter 2011
- Complete Phase II clinical studies with G-202 in the fourth quarter 2012
- License G-202 to a third party during Phase I/II
  
  Note: If GenSpera is unable to license G-202 to a third party during Phase I/II on acceptable terms, the Company intends to proceed with Phase III Clinical trials.

- Finalize formulation and toxicology work for G-115, potentially filing an IND in the second half of 2011

Figure 13 depicts the Company’s intended development timelines for G-202 and G-115.

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**Figure 13**
GenSpera, Inc.

**POTENTIAL MILESTONES**

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<th>2010</th>
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<th>2012</th>
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<td>3Q</td>
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<td>G-202 GMP</td>
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<td>G-115 Phase I</td>
<td>1Q</td>
<td>2Q</td>
<td>3Q</td>
<td>4Q</td>
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</table>

*Source: GenSpera, Inc.*
Key Points to Consider

- GenSpera’s prodrug technology entails the attachment of a targeting/masking agent to an active drug, making the drug inactive and soluble in the bloodstream. The Company uses specific peptides as its targeting/masking agents, and an analog of the cytotoxin thapsigargin, called 12ADT, as the active drug. The Company’s prodrugs are designed to either target the blood supply that is supporting tumor growth or target the tumor directly.
  - The prodrug can only be activated once the targeting/masking agent is removed by an enzyme that is expressed at the site of a tumor. With the agent detached, the drug becomes insoluble and precipitates directly into nearby cells.
  - The Company’s patent-protected peptides, which are based on years of research by GenSpera’s Scientific Advisory Board members, were developed to attack multiple targets.

- Traditional cancer treatments (e.g., surgery or radiation therapy) are localized therapies that lose efficacy once the cancer has spread. Chemotherapeutics are also widely used, but as many are not targeted, their toxic effects harm both healthy tissues and cancer cells.
  - In contrast, GenSpera’s prodrug technology may be more effective for cancer that has spread due to its ability to deliver higher concentrations of cytotoxic agents to tumors while avoiding the toxicity of these higher doses in the rest of the body.

- The Company’s lead prodrug candidate, G-202, is composed of 12ADT and a targeting/masking agent. Once G-202 is administered intravenously into the bloodstream, it can only be activated at a target site by prostate-specific membrane antigen (PSMA), which is expressed in cancer-supporting vasculature but not in normal blood vessels.
  - G-202 may be able to inhibit further tumor angiogenesis and attack existing tumor vasculature—stopping growth by depleting the cancer’s nutrient supply and potentially causing tumor regression—versus the current generation of anti-angiogenesis drugs that GenSpera believes only slow or prevent further growth of tumors, but do not cause tumor regression.
  - Based on G-202’s preclinical data in breast, prostate, bladder, and kidney cancer, as well as its specific, robust mechanism of action, the Company believes that G-202 can demonstrate increased efficacy and less toxicity versus currently available chemotherapies, which cause toxicity to all rapidly dividing cells in the body, including healthy, non-cancerous cells.
  - A Phase I clinical trial with G-202 is ongoing at two major cancer centers. The trial is advancing according to a schedule determined in dialog with the U.S. Food and Drug Administration, with four patients enrolled to date. The study’s primary endpoints include determining the safety, tolerability, and pharmacokinetics of G-202. Efficacy data may be collected in parallel. If successful, the Company plans to initiate up to four Phase II clinical trials in various cancer types.

- GenSpera’s second prodrug technology approach attacks cancer cells directly by targeting enzymes found primarily in tumors. G-115, the primary development candidate in this area, targets a protein used by physicians to detect the presence of prostate cancer, called prostate-specific antigen (PSA). The Company plans to obtain an Investigational New Drug (IND) for G-115 in the third quarter 2011.

- GenSpera is supported by a management team with extensive experience in identifying oncology treatments and bringing them to the clinic as well as its Scientific Advisory Board, which is composed of members who are all inventors of the technology as well as shareholders.

- As of March 31, 2010, the Company’s cash and cash equivalent position was ~$2.7 million. In May 2010, GenSpera raised an additional $2.7 million in gross proceeds.
Historical Financial Results

Table 10, Table 11 (page 41), and Table 12 (page 42) provide a summary of GenSpera's key historical financial statements: its Condensed Statements of Losses, Condensed Balance Sheets, and Condensed Statements of Cash Flows.

Table 10
GenSpera, Inc.
(A Development Stage Company)
CONDENSED STATEMENTS OF LOSSES
FOR THE THREE MONTHS ENDED MARCH 31, 2010 AND 2009
AND FOR THE PERIOD FROM INCEPTION (NOVEMBER 21, 2003) TO MARCH 31, 2010
(Unaudited)

<table>
<thead>
<tr>
<th></th>
<th>Three Months ended March 31, 2010</th>
<th>Three Months ended March 31, 2009</th>
<th>Cumulative Period from November 21, 2003 (date of inception) to March 31, 2010</th>
</tr>
</thead>
<tbody>
<tr>
<td>Operating expenses:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>General and administrative expenses</td>
<td>$395,880</td>
<td>$199,717</td>
<td>$3,110,269</td>
</tr>
<tr>
<td>Research and development</td>
<td>354,065</td>
<td>309,502</td>
<td>5,865,606</td>
</tr>
<tr>
<td>Total operating expenses</td>
<td>749,945</td>
<td>509,219</td>
<td>8,975,875</td>
</tr>
<tr>
<td>Loss from operations</td>
<td>(749,945)</td>
<td>(509,219)</td>
<td>(8,975,875)</td>
</tr>
<tr>
<td>Finance cost</td>
<td>—</td>
<td>(472,938)</td>
<td>(518,675)</td>
</tr>
<tr>
<td>Change in fair value of derivative liability</td>
<td>(1,423,492)</td>
<td>(572,785)</td>
<td>(2,854,042)</td>
</tr>
<tr>
<td>Interest income (expense), net</td>
<td>3,373</td>
<td>(2,608)</td>
<td>(13,001)</td>
</tr>
<tr>
<td>Loss before provision for income taxes</td>
<td>(2,170,064)</td>
<td>(1,557,550)</td>
<td>(12,361,593)</td>
</tr>
<tr>
<td>Provision for income taxes</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Net loss</td>
<td>$ (2,170,064)</td>
<td>$ (1,557,550)</td>
<td>$ (12,361,593)</td>
</tr>
<tr>
<td>Net loss per Common Share, basic and diluted</td>
<td>$ (0.14)</td>
<td>$ (0.12)</td>
<td></td>
</tr>
<tr>
<td>Weighted average shares outstanding</td>
<td>15,649,956</td>
<td>12,699,314</td>
<td></td>
</tr>
</tbody>
</table>

Source: GenSpera, Inc.
<table>
<thead>
<tr>
<th>Assets</th>
<th>March 31, 2010</th>
<th>December 31, 2009</th>
</tr>
</thead>
<tbody>
<tr>
<td>Current assets:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cash</td>
<td>$2,711,281</td>
<td>$2,255,311</td>
</tr>
<tr>
<td>Total current assets</td>
<td>2,711,281</td>
<td>2,255,311</td>
</tr>
<tr>
<td>Fixed assets, net of accumulated depreciation of $1,500 and $708</td>
<td>14,333</td>
<td>15,125</td>
</tr>
<tr>
<td>Intangible assets, net of accumulated amortization of $30,695 and $26,858</td>
<td>153,473</td>
<td>157,310</td>
</tr>
<tr>
<td>Total assets</td>
<td>$2,879,087</td>
<td>$2,427,746</td>
</tr>
</tbody>
</table>

| Liabilities and stockholders' deficit |       |                  |
| Current liabilities: |       |                  |
| Accounts payable and accrued expenses | $232,070 | $78,198          |
| Accrued interest - stockholder | 9,195   | 8,107            |
| Convertible Note payable - stockholder, current portion | 35,000   | 35,000           |
| Total current liabilities | 276,265 | 121,305          |
| Convertible Note payable, net of discount of $0 and $11,046 | — | — |
| Convertible Notes payable - stockholder, long-term portion | 70,000  | 70,000           |
| Derivative liabilities | 3,655,387 | 2,290,686       |
| Total liabilities | 4,001,652 | 2,481,991       |

| Commitments and contingencies |       |                  |
| Stockholders' equity deficit: |       |                  |
| Preferred Stock, par value $.0001 per share; 10,000,000 shares authorized, none issued and outstanding | — | — |
| Common Stock, par value $.0001 per share; 80,000,000 shares authorized, 16,033,187 and 15,466,446 shares issued and outstanding | 1,603 | 1,547 |
| Additional paid-in capital | 11,237,425 | 10,135,737 |
| Deficit accumulated during the development stage | (12,361,593) | (10,191,529) |
| Total stockholders' equity deficit | (1,122,565) | (54,245) |
| Total liabilities and stockholders' equity deficit | $2,879,087 | $2,427,746 |

Source: GenSpera, Inc.
### Table 12
GenSpera, Inc.
(A Development Stage Company)
CONDENSED STATEMENTS OF CASH FLOWS
FOR THE THREE MONTHS ENDED MARCH 31, 2010 AND 2009
AND FOR THE PERIOD FROM INCEPTION (NOVEMBER 21, 2003) TO MARCH 31, 2010
(Unaudited)

<table>
<thead>
<tr>
<th></th>
<th>Three months ended March 31, 2010</th>
<th>Three months ended March 31, 2009</th>
<th>Cumulative Period from November 21, 2003 (date of inception) to March 31, 2010</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cash flows from operating activities:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Net loss</td>
<td>$(2,170,064)</td>
<td>$(1,557,550)</td>
<td>$(12,361,593)</td>
</tr>
<tr>
<td>Adjustments to reconcile net loss to net cash used in operating activities:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Depreciation and amortization</td>
<td>4,629</td>
<td>3,837</td>
<td>32,195</td>
</tr>
<tr>
<td>Stock-based compensation</td>
<td>186,742</td>
<td>29,554</td>
<td>2,667,086</td>
</tr>
<tr>
<td>Warrants issued for financing costs</td>
<td>467,840</td>
<td>467,840</td>
<td></td>
</tr>
<tr>
<td>Change in fair value of derivative liability</td>
<td>1,423,492</td>
<td>572,785</td>
<td>2,854,042</td>
</tr>
<tr>
<td>Contributed services</td>
<td></td>
<td></td>
<td>774,000</td>
</tr>
<tr>
<td>Amortization of debt discount</td>
<td></td>
<td>5,098</td>
<td>20,675</td>
</tr>
<tr>
<td>Changes in assets and liabilities:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Increase (decrease) in accounts payable and accrued expenses</td>
<td>154,960</td>
<td>(94,479)</td>
<td>267,689</td>
</tr>
<tr>
<td>Cash used in operating activities</td>
<td>(400,241)</td>
<td>(572,915)</td>
<td>(5,278,066)</td>
</tr>
<tr>
<td>Cash flows from investing activities:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Acquisition of property and equipment</td>
<td>15,833</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Acquisition of intangibles</td>
<td>184,168</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cash used in investing activities</td>
<td></td>
<td></td>
<td>(200,001)</td>
</tr>
<tr>
<td>Cash flows from financing activities:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Proceeds from sale of Common Stock and Warrants</td>
<td>806,210</td>
<td>699,985</td>
<td>8,034,347</td>
</tr>
<tr>
<td>Proceeds from exercise of Warrants</td>
<td>50,001</td>
<td></td>
<td>50,001</td>
</tr>
<tr>
<td>Proceeds from Convertible Notes - stockholder</td>
<td></td>
<td></td>
<td>155,000</td>
</tr>
<tr>
<td>Repayments of Convertible Notes - stockholder</td>
<td></td>
<td></td>
<td>(50,000)</td>
</tr>
<tr>
<td>Cash provided by financing activities</td>
<td>856,211</td>
<td>699,985</td>
<td>8,189,348</td>
</tr>
<tr>
<td>Net increase in cash</td>
<td>455,970</td>
<td>127,070</td>
<td>2,711,281</td>
</tr>
<tr>
<td>Cash, beginning of period</td>
<td>2,255,311</td>
<td>534,290</td>
<td></td>
</tr>
<tr>
<td>Cash, end of period</td>
<td>$2,711,281</td>
<td>$661,360</td>
<td>$2,711,281</td>
</tr>
<tr>
<td>Supplemental cash flow information:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cash paid for interest</td>
<td>$</td>
<td>$79</td>
<td></td>
</tr>
<tr>
<td>Cash paid for income taxes</td>
<td>$</td>
<td>$</td>
<td></td>
</tr>
<tr>
<td>Non-cash financial activities:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Derivative liability reclassified to equity upon exercise of Warrants</td>
<td>$58,791</td>
<td>$</td>
<td></td>
</tr>
</tbody>
</table>

Source: GenSpera, Inc.
Risks

Some of the information in this Executive Informational Overview® (EIO®) relates to future events or future business and financial performance. Such statements can only be predictions and the actual events or results may differ from those described due to the risks presented in GenSpera's statements on Forms 10-K, 10-Q, as well as other forms filed from time to time. The content of this report with respect to GenSpera has been compiled primarily from information available to the public released by the Company through U.S. Securities and Exchange Commission (SEC) filings. GenSpera is solely responsible for the accuracy of this information. Information as to other companies has been prepared from publicly available information and has not been independently verified by the Company. Certain summaries of activities have been condensed to aid the reader in gaining a general understanding. For more information about GenSpera, please refer to the Company's website at www.genspera.com.

Investors should carefully consider the risks and information about GenSpera's business described below. Investors should not interpret the order in which these considerations are presented as an indication of their relative importance. The risks and uncertainties described below are not the only risks that the Company faces. Additional risks and uncertainties not presently known to GenSpera or that the Company currently believes to be immaterial may also adversely affect its business. If any of the following risks and uncertainties develops into actual events, GenSpera’s business, financial condition, and results of operations could be materially and adversely affected.

RISKS RELATING TO THE COMPANY’S STAGE OF DEVELOPMENT

As a result of GenSpera’s limited operating history, investors cannot rely upon its historical performance to make an investment decision.

Since inception in 2003 and through March 31, 2010, the Company has raised approximately $8,084,000 in capital. During this same period, GenSpera has recorded accumulated losses totaling $12,361,593. As of March 31, 2010, the Company had working capital of $2,435,016 and a stockholders’ deficit of $1,122,565. GenSpera’s net losses for the two most recent fiscal years ended December 31, 2008 and 2009 were $3,326,261 and $5,132,827, respectively. Since inception, the Company has generated no revenue.

GenSpera’s limited operating history means that there is a high degree of uncertainty in its ability to achieve the following: (1) develop and commercialize its technologies and proposed products; (2) obtain approval from the FDA; (3) achieve market acceptance of its proposed product, if developed; (4) respond to competition; or (5) operate the business, as management has not previously undertaken such actions as a company. No assurances can be given as to exactly when, if at all, GenSpera will be able to fully develop, license, commercialize, market, sell, and derive material revenues from its proposed products in development.

The Company will need to raise additional capital to continue operations.

GenSpera currently generates no cash. The Company has relied entirely on external financing to fund operations. Such financing has come primarily from the sale of Common Stock to third parties and the exercise of Warrants/Options. GenSpera has expended and will continue to expend substantial amounts of cash in the development, preclinical, and clinical testing of its proposed products. The Company will require additional cash to conduct drug development, to establish and conduct preclinical and clinical trials, and for general working capital needs. GenSpera anticipates that it will require an additional $13 million to take its lead drug through Phase II clinical evaluations, which is currently anticipated to occur in the fourth quarter 2012.

As of March 31, 2010, GenSpera had cash on hand of approximately $2,711,000, and an additional $2,695,000 was raised in a financing completed in May 2010. Collectively, the Company anticipates that these funds can support its operations through June 2011. Presently, the Company has an average monthly cash burn rate of roughly $185,000, which GenSpera expects to increase to approximately
$550,000 per month in the second half of 2010 as the Company funds development of G-115 and manufactures more G-202 in preparation for the Phase II clinical program. It is expected that the monthly burn rate will decrease considerably in the first half of 2011 and then increase again with the advent of Phase II clinical studies. These projections are based upon the assumption that the Company does not engage in an extraordinary transaction or otherwise face unexpected events or contingencies. Accordingly, GenSpera will need to raise additional capital to fund anticipated operating expenses after June 2011. In the event the Company is not able to secure financing, GenSpera may have to delay, reduce the scope of, or eliminate one or more of its research, development, or commercialization programs. Any such change may materially harm its business, financial condition, and operations.

GenSpera’s long-term capital requirements are expected to depend on many factors:

- its development programs;
- the progress and costs of preclinical studies and clinical trials;
- the time and costs involved in obtaining regulatory clearance;
- the costs involved in preparing, filing, prosecuting, maintaining, and enforcing patent claims;
- the costs and the ability of the Company to license its products;
- competing technological and market developments;
- market acceptance of its proposed products, if developed; and
- the costs for recruiting and retaining employees, consultants, and professionals.

GenSpera cannot assure investors that financing, whether from external sources or related parties, will be available if needed or on favorable terms. If additional financing is not available when required or is not available on acceptable terms, the Company may be unable to fund operations and planned growth, develop or enhance its technologies, take advantage of business opportunities, or respond to competitive market pressures.

Raising needed capital may be difficult as a result of GenSpera's limited operating history.

When making investment decisions, investors typically look at a company’s historical performance in evaluating the risks and operations of the business and the business’s future prospects. GenSpera’s limited operating history makes such evaluation and an estimation of its future performance substantially more difficult. As a result, investors may be unwilling to invest in the Company or such investment may be on terms or conditions that are not acceptable. If GenSpera is unable to secure such additional finance, it may need to cease operations.

The Company may not be able to commercially develop its technologies.

GenSpera has concentrated its research and development on its prodrug technologies. The Company’s ability to generate revenue and operate profitably will depend on its ability to develop these technologies for human applications. GenSpera’s technologies are primarily directed toward developing therapeutic cancer agents. The Company cannot guarantee that the results obtained in the preclinical and clinical evaluation of its therapeutic agents will be sufficient to warrant FDA approval. Even if GenSpera’s therapeutic agents are approved by the FDA, there is no guarantee that they will exhibit an enhanced efficacy relative to competing therapeutic modalities such that they will be adopted by the medical community. Without significant adoption by the medical community, the Company’s agents will have limited commercial potential, which could harm GenSpera’s ability to generate revenues, operate profitably, or remain a viable business.
Inability to complete preclinical and clinical testing and trials will impair GenSpera’s viability.

In the first quarter 2010, the Company commenced its first clinical trials of G-202 at the University of Wisconsin Carbone Cancer Center in Madison, Wisconsin, and at the Sydney Kimmel Comprehensive Cancer Center at Johns Hopkins University. Although GenSpera has commenced its clinical trials, the outcome of the trials is uncertain and, if the Company is unable to satisfactorily complete such trials or if such trials yield unsatisfactory results, GenSpera will be unable to commercialize its proposed products. No assurances can be given that the Company’s clinical trials will be successful. The failure of such trials could delay or prevent regulatory approval and could harm GenSpera’s ability to generate revenues, operate profitably, or remain a viable business.

Future financing will result in dilution to existing stockholders.

GenSpera will require additional financing in the future. The Company is authorized to issue 80 million shares of Common Stock and 10 million shares of Preferred Stock. Such securities may be issued without the approval or consent of GenSpera’s stockholders. The issuance of equity securities in connection with a future financing will result in a decrease of the Company’s current stockholders’ percentage ownership.

RISKS RELATING TO INTELLECTUAL PROPERTY AND GOVERNMENT REGULATION

GenSpera may not be able to withstand challenges to its intellectual property (IP) rights.

The Company relies on IP, including issued and pending patents, as the foundation of its business. GenSpera’s IP rights may come under challenge. No assurances can be given that, even if issued, the Company’s patents will survive claims alleging invalidity or infringement on other patents. The viability of GenSpera’s business will suffer if such patent protection becomes limited or is eliminated.

The Company may not be able to adequately protect its IP.

Considerable research with regard to GenSpera’s technologies has been performed in countries outside the U.S. The laws protecting IP in some of those countries may not provide protection for the Company’s trade secrets and IP. If GenSpera’s trade secrets or IP are misappropriated in those countries, the Company may be without adequate remedies to address the issue. At present, GenSpera is not aware of any infringement of its IP. In addition to patents, the Company relies on confidentiality and assignment of invention agreements to protect its IP. These agreements provide for contractual remedies in the event of misappropriation. GenSpera does not know to what extent, if any, these agreements and any remedies for their breach will be enforced by a court. In the event the Company’s IP is misappropriated or infringed upon and an adequate remedy is not available, its future prospects will greatly diminish.

GenSpera’s proposed products may not receive FDA approval.

The FDA and comparable government agencies in foreign countries impose substantial regulations on the manufacture and marketing of pharmaceutical products through lengthy and detailed laboratory, preclinical, and clinical testing procedures, sampling activities, and other costly and time-consuming procedures. Satisfaction of these regulations typically takes several years or more and varies largely based upon the type, complexity, and novelty of the proposed product. On September 4, 2009, GenSpera received approval from the FDA for its first IND to commence clinical trials with G-202. Although the Company began the G-202 Phase I clinical trial on January 19, 2010, GenSpera cannot assure investors that it will successfully complete the trial. Further, the Company cannot yet accurately predict when it might first submit any product license application for FDA approval or whether any such product license application would be granted on a timely basis, if at all. Any delay in obtaining, or failure to obtain, such approvals could have a materially adverse effect on the commercialization of GenSpera’s products and the viability of the Company.
GENERAL RISKS RELATING TO GENSPERA’S BUSINESS AND BUSINESS MODEL

The Company depends on Dr. Craig A. Dionne, its chief executive officer, chief financial officer, president, and chairman, and Dr. Russell Richerson, the Company’s chief operating officer and secretary, for its continued operations.

GenSpera only has two full-time employees. The loss of Dr. Dionne or Dr. Richerson would be detrimental to the business. Although the Company has entered into employment agreements with Dr. Dionne and Dr. Richerson, there can be no assurance that these individuals will continue to provide services to GenSpera. A voluntary or involuntary termination of employment by Dr. Dionne or Dr. Richerson could have a materially adverse effect on the business. Further, as part of their employment agreements, Dr. Dionne and Dr. Richerson agreed to not compete with the Company for a certain amount of time following the termination of their employment. Once the applicable time of these provisions expires, Dr. Dionne and Dr. Richerson may be employed by a competitor of GenSpera in the future.

GenSpera may be required to make significant payments to members of its management team if the Company terminates their employment or institutes a change of control.

GenSpera is a party to employment agreements with Dr. Dionne and Dr. Richerson. In the event that the Company terminates the employment of either of these executives or experiences a change in control or, in certain cases, if such executives terminate their employment with GenSpera, such executives will be entitled to receive certain severance and related payments. Additionally, in such instance, certain securities held by Dr. Dionne and Dr. Richerson will become immediately vested and exercisable. Upon the occurrence of any such event, the Company’s obligation to make such payments could significantly impact its working capital and, accordingly, GenSpera’s ability to execute its business plan, which could have a materially adverse effect on the business. Also, these provisions may discourage potential takeover attempts.

The Company will require additional personnel to execute its business plan.

GenSpera’s anticipated growth and expansion into areas and activities requiring additional expertise—such as clinical testing, regulatory compliance, manufacturing, and marketing—may require the addition of new management personnel and the development of additional expertise by existing management. There is intense competition for qualified personnel in such areas. There can be no assurance that the Company will be able to continue to attract and retain the qualified personnel necessary for the development of its business.

GenSpera’s competitors have significantly greater experience and financial resources.

The Company competes against numerous companies, many of which have substantially greater financial and other resources than GenSpera. Several such enterprises have research programs and efforts to treat the same diseases that GenSpera targets. Companies such as Merck and Ipsen, among others, have substantially greater resources and experience than the Company does and are situated to compete with GenSpera effectively. As a result, the Company’s competitors may bring rival products to market that would result in a decrease in demand for GenSpera’s product, if developed, which could have a materially adverse effect on the viability of the Company.

GenSpera intends to rely exclusively upon the third-party FDA-approved manufacturers and suppliers for its products.

The Company currently has no internal manufacturing capability and will rely exclusively on FDA-approved licensees, strategic partners, or third-party contract manufacturers or suppliers. Should GenSpera be forced to manufacture its products, the Company cannot give investors any assurance that it will be able to develop internal manufacturing capabilities or procure third-party suppliers. In the event that GenSpera seeks third-party suppliers, they may require the Company to purchase a minimum amount of materials or could require other unfavorable terms. Any such event would materially impact GenSpera’s prospects and could delay the development and sale of its products. Moreover, the Company cannot give investors any assurance that any contract manufacturers or suppliers that it selects will be able to supply products in a timely or cost-effective manner or in accordance with applicable regulatory requirements or GenSpera’s specifications.
The Company’s business is dependent upon securing sufficient quantities of a natural product that currently grows in very specific locations outside of the U.S.

The therapeutic component of GenSpera’s products, including G-202, is referred to as 12ADT. 12ADT functions by dramatically raising the levels of calcium inside cells, leading to cell death. 12ADT is produced from a material called thapsigargin. Thapsigargin is derived from the seeds of a plant referred to as *Thapsia garganica* (*T. garganica*), which grows along the coastal regions of the Mediterranean Sea. GenSpera currently secures the seeds from Thapsibiza S.L., a third-party supplier. There can be no assurances that the countries from which the Company can secure *T. garganica* will continue to allow Thapsibiza to collect such seeds or to export the seeds to the U.S. In the event that GenSpera is no longer able to import these seeds, the Company will not be able to produce its proposed drug and its business will be adversely affected.

The current manufacturing process of G-202 requires acetonitrile.

The current manufacturing process for G-202 requires the common solvent acetonitrile. Beginning in late 2008, there was a temporary worldwide shortage of acetonitrile for a number of reasons. During that period of time, GenSpera observed that the available supply of acetonitrile was of variable quality, some of which is not suitable for the Company’s purposes. If GenSpera is unable to successfully change its manufacturing methods to avoid the reliance upon acetonitrile, the Company may incur prolonged production timelines and increased production costs if an acetonitrile shortage reoccurs. In an extreme case, this situation could adversely affect GenSpera’s ability to manufacture G-202 altogether, thus significantly impacting the Company’s future operations.

In order to secure market share and generate revenues, GenSpera’s proposed products must be accepted by the healthcare community.

The Company’s proposed products, if approved for marketing, may not achieve market acceptance as hospitals, physicians, patients, or the medical community in general may decide not to accept and use them. GenSpera is attempting to develop products that will likely be first approved for marketing in late-stage cancer where there is no truly effective standard of care. If approved for use in the late stages of disease progression, the drugs will then be evaluated in earlier stages where they would represent substantial departures from established treatment methods and will compete with a number of conventional drugs and therapies manufactured and marketed by major pharmaceutical companies. It is too early in the development cycle for GenSpera to accurately predict its major competitors. Nonetheless, the degree of market acceptance of any of GenSpera’s developed products will depend on the following factors:

- its demonstration to the medical community of the clinical efficacy and safety of its proposed products;
- its ability to create products that are superior to alternatives currently on the market;
- its ability to establish in the medical community the potential advantage of GenSpera’s treatments over alternative treatment methods; and
- the reimbursement policies of government and third-party payors.

If the healthcare community does not accept the Company’s products for any of the foregoing reasons or for any other reason, GenSpera’s business will be materially harmed.

The Company may be required to secure land for cultivation and harvesting of *T. garganica*.

GenSpera believes that it can satisfy its needs for clinical development of G-202 through completion of Phase III clinical studies from *T. garganica* that grows naturally in the wild. In the event that G-202 is approved for commercial marketing, the Company’s current supply of *T. garganica* may not be sufficient for the anticipated demand. GenSpera estimates that in order to secure sufficient quantities of *T. garganica* to commercialize a product comprising G-202, the Company will need to secure roughly 1,000 acres of land to cultivate and grow *T. garganica*. GenSpera anticipates that the cost to lease such land
would be $400,000 per year but has not yet fully assessed what other costs would be associated with a full-scale farming operation. There can be no assurances that the Company can secure such acreage or, even if GenSpera is able to do so, that it could adequately grow sufficient quantities of *T. garganica* to satisfy any commercial objectives that involve G-202. The Company’s inability to secure adequate seeds will result in GenSpera not being able to develop and manufacture its proposed drug and will adversely impact its business.

*T. garganica* and thapsigargin can cause severe skin irritation.

It has been known for centuries that the plant *T. garganica* can cause severe skin irritation when contact is made between the plant and the skin. In 1978, thapsigargin was determined to be the skin-irritating component of the plant *T. garganica*. The therapeutic component of GenSpera’s products, including G-202, is derived from thapsigargin. The Company obtains thapsigargin from the above-ground seeds of *T. garganica*, which are harvested by hand. Those who harvest the seeds must wear protective clothing and gloves to avoid skin contact. Although GenSpera obtains the seeds from a third-party contractor located in Spain who has contractually waived any and all liability associated with collecting the seeds, it is possible that the contractor or those employed by the contractor may suffer medical issues related to the harvesting and subsequently seek compensation from the Company via, for example, litigation. Despite GenSpera’s contractual relationship with the third-party contractor, no assurances can be given that the Company will not be the subject of litigation related to harvesting.

The synthesis of 12ADT must be conducted in special facilities.

There are a limited number of manufacturing facilities qualified to handle and manufacture therapeutic toxic agents and compounds. This limits the potential number of possible manufacturing sites for GenSpera’s therapeutic compounds derived from *T. garganica*. No assurances can be provided that these facilities will be available for the manufacture of the Company’s compounds under its time schedules or within the parameters of its manufacturing budget. In the event facilities are not available for manufacturing GenSpera’s compounds, its business and future prospects will be adversely affected.

G-202 has not been subjected to large-scale manufacturing procedures.

To date, G-202 has only been manufactured at a scale adequate to supply early-stage clinical trials. There can be no assurances that the current procedure for manufacturing G-202 will work at a larger scale adequate for commercial needs. In the event that G-202 cannot be manufactured in sufficient quantities, GenSpera’s future prospects could be significantly impacted.

The Company may not have adequate insurance coverage.

The testing, manufacturing, marketing, and sale of human therapeutic products entail an inherent risk of product liability claims. GenSpera cannot assure investors that substantial claims will not be asserted against the Company. In the event that GenSpera is forced to expend significant funds on defending such claims beyond its current coverage, and in the event those funds come from operating capital, the Company will be required to reduce its business activities, which could lead to significant losses.

Provisions in Delaware law and executive employment agreements may prevent or delay a change of control.

GenSpera is subject to the Delaware anti-takeover laws regulating corporate takeovers. These anti-takeover laws prevent Delaware corporations from engaging in a merger or sale of more than 10% of its assets with any stockholder, including all affiliates and associates of the stockholder, who owns 15% or more of the corporation’s outstanding voting stock for three years following the date that the stockholder acquired 15% or more of the corporation’s assets unless one of the following exemptions applies:

- the Board of Directors approved the transaction in which the stockholder acquired 15% or more of the corporation’s assets;
after the transaction in which the stockholder acquired 15% or more of the corporation's assets, the
stockholder owned at least 85% of the corporation's outstanding voting stock, excluding shares
owned by directors, officers, and employee stock plans in which employee participants do not have
the right to determine confidentially whether shares held under the plan will be tendered in a tender or
exchange offer; or

on or after this date, the merger or sale is approved by the Board of Directors and the holders of at
least two-thirds of the outstanding voting stock that is not owned by the stockholder.

A Delaware corporation may opt out of the Delaware anti-takeover laws if its certificate of incorporation or
bylaws so provide. GenSpera has not opted out of the provisions of the anti-takeover laws. As such,
these laws could prohibit or delay mergers or other takeover or change of control of GenSpera and may
discourage attempts by other companies to acquire the Company.

In addition, employment agreements with certain executive officers provide for the payment of severance
and acceleration of the vesting of Options and Restricted Stock in the event of termination of the
executive officer following a change of control of GenSpera. These provisions could have the effect of
discouraging potential takeover attempts.

RISKS RELATING TO GENSPERA’S COMMON STOCK

There is no established public market for the Company's securities.

On September 18, 2009, GenSpera’s Common Shares began trading on the OTC.BB. Notwithstanding,
there has been sporadic trading in the Company’s Common Shares. Accordingly, there is no established
public market for GenSpera’s securities. An investment in the Company’s Common Stock should be
considered totally illiquid. No assurances can be given that a public market for GenSpera’s securities will
ever materialize. Additionally, even if a public market for the Company’s securities develops and its
securities become traded, the trading volume may be limited, making it difficult for an investor to sell
shares.

GenSpera faces risks related to compliance with corporate governance laws and financial
reporting standards.

The Sarbanes-Oxley Act of 2002 as well as related new rules and regulations implemented by the SEC
and the Public Company Accounting Oversight Board require changes in the corporate governance
practices and financial reporting standards for public companies. These new laws, rules, and regulations,
including compliance with Section 404 of the Sarbanes-Oxley Act of 2002 relating to internal control over
financial reporting, will materially increase the Company's legal and financial compliance costs and make
some activities more time-consuming and burdensome. As a result, management may be required to
devote more time to compliance, which could result in a reduced focus on development, thereby
adversely affecting the Company's development activities. Also, the increased costs will require the
Company to seek financing sooner that may otherwise have been required.

Starting in 2007, Section 404 of the Sarbanes-Oxley Act of 2002 requires a company’s management to
assess the company’s internal control over financial reporting annually and include a report on such
assessment in its Annual Report filed with the SEC. For small reporting companies with fiscal years
ending on or after June 15, 2010, independent registered public accounting firms will be required to audit
both the design and operating effectiveness of internal controls and management's assessment of the
design and the operating effectiveness of such internal controls. If this deadline is not extended,
GenSpera will be required to expand substantial capital in connection with compliance.

Because of GenSpera’s limited resources, the Company’s management has concluded that its internal
control over financial reporting may not be effective in providing reasonable assurance regarding the
reliability of financial reporting and the preparation of financial statements for external purposes in
accordance with U.S. generally accepted accounting principles. To mitigate the current limited resources
and limited employees, GenSpera relies heavily on direct management oversight of transactions, along
with the use of legal and accounting professionals. As it grows, GenSpera plans to increase its employee
count, which will enable the Company to implement adequate segregation of duties within the Committee
of Sponsoring Organizations of the Treadway Commission internal control framework.
GenSpera does not intend to pay cash dividends.

The Company does not anticipate paying cash dividends in the foreseeable future. Accordingly, any gains on an investment in GenSpera will need to come through an increase in the price of the Company’s Common Stock. The lack of a market for GenSpera’s Common Stock makes such gains highly unlikely.

The Company's Board of Directors has broad discretion to issue additional securities.

GenSpera is entitled under its Certificate of Incorporation to issue up to 80,000,000 Common and 10,000,000 “blank check” Preferred Shares. Blank check Preferred Shares provide the Board of Directors broad authority to determine voting, dividend, conversion, and other rights. As of March 31, 2010, the Company had issued and outstanding 16,033,187 Common Shares and 8,003,903 Common Shares reserved for issuance upon the exercise of current outstanding Options, Warrants, and Convertible Securities. Accordingly, GenSpera will be entitled to issue up to 55,962,910 additional Common Shares and 10,000,000 additional Preferred Shares. The Company’s Board may generally issue those Common and Preferred Shares, or Options or Warrants to purchase those shares, without further approval by GenSpera’s shareholders. Any Preferred Shares that GenSpera may issue will have such rights, preferences, privileges, and restrictions as may be designated from time-to-time by its Board, including preferential dividend rights, voting rights, conversion rights, redemption rights, and liquidation provisions. It is likely that the Company will be required to issue a large amount of additional securities to raise capital to further its development and marketing plans. It is also likely that GenSpera will be required to issue a large amount of additional securities to directors, officers, employees, and consultants as compensatory grants in connection with their services, both in the form of stand-alone grants or under the Company’s various stock plans. The issuance of additional securities may cause substantial dilution to GenSpera’s shareholders.

The Company's officers and scientific advisors beneficially own roughly 41% of GenSpera's outstanding Common Shares.

GenSpera’s officers and scientific advisors own approximately 41% of its issued and outstanding Common Shares. As a consequence of their level of stock ownership, the group will substantially retain the ability to elect or remove members of GenSpera’s Board of Directors and thereby control management. This group of shareholders has the ability to significantly control the outcome of corporate actions requiring shareholder approval, including mergers and other changes of corporate control, going private transactions, and other extraordinary transactions any of which may be in opposition to the best interest of the other shareholders and may negatively impact the value of an investment in the Company.
Recent Events

Recently, GenSpera’s technology and its founders have been featured in the San Antonio Business Journal, MSN Money (www.moneycentral.msn.com), and PCRP Perspectives, a newsletter issued as part of the U.S. Department of Defense’s Prostate Cancer Research Program (PCRP). An overview of GenSpera’s recent announcements is provided below, referring the reader to the Company’s website for complete press releases (www.genspera.com).

05/25/2010—GenSpera, Inc. announced that on May 18, 2010, the Company entered into a Securities Purchase Agreement with a number of institutional and accredited investors. GenSpera offered and sold the investors 1,347,500 units at $2.00 per unit resulting in gross proceeds of approximately $2,695,000.

05/18/2010—Announced that Dr. Craig A. Dionne, the Company’s chief executive officer, chief financial officer, president, and chairman, was expected to present a corporate overview, including a company update and future outlook, at the Source Capital Group Small Cap Virtual Conference on Tuesday, May 18, 2010. The virtual conference showcased emerging small- and micro-cap companies in a live forum.

02/02/2010—Announced that the U.S. Patent and Trademark Office (USPTO) issued its patent application, entitled “Tumor Activated Prodrugs,” as U.S. Patent No. 7,635,682. The patent covers the composition and potential uses of G-115, the Company’s second anticancer drug in development, further strengthening GenSpera’s intellectual property (IP) position for G-115 and its use in prostate cancer and other prostate pathologies including enlarged prostate. The term of the patent extends to 2026.

01/27/2010—Announced that the first patient was treated in the Phase I clinical study of GenSpera’s lead prodrug candidate, G-202, at the University of Wisconsin Carbone Cancer Center in Madison, Wisconsin. The trial is also being conducted at the Sidney Kimmel Comprehensive Cancer Center at Johns Hopkins in Baltimore, Maryland.

11/03/2009—Announced that its Common Stock began trading on November 2, 2009, on the Over-the-Counter Bulletin Board (OTC.BB) under the symbol “GNSZ.”


08/14/2009—Received a Notice of Effectiveness from the U.S. Securities and Exchange Commission (SEC) accepting the Company’s Form S-1 (filed in July 2009), the general form for registration of securities under the Securities Act of 1933, and thereby registering GenSpera’s shares for public trading.

08/12/2009—Filed an amendment to the previously submitted Form S-1.

07/31/2009—Filed Form S-1 with the SEC relating to the resale of 4,516,120 shares of the Company’s Common Stock. GenSpera’s Common Stock was not then traded on any market or exchange, and the Company had not yet applied for listing or quotation on any public market. GenSpera anticipated seeking sponsorship to trade its Common Stock on the OTC.BB once the registration became effective.

07/29/2009—Entered into a Securities Purchase Agreement with a number of accredited investors. Per the terms of the agreement, GenSpera sold units aggregating approximately $907,000 to the investors. The price per unit was $1.50. Each unit consisted of the following: (1) one share of the Company’s Common Stock; and (2) one half Common Stock Purchase Warrant. The Warrants had a five-year term and allowed investors to purchase GenSpera’s Common Shares at $3.00 per share. The Warrants contained anti-dilution protection against stock splits, stock dividends, and similar transactions. GenSpera incurred $79,583 in fees and expenses for the transaction. The Company also issued 40,001 additional Common Stock Purchase Warrants under the same terms as the investor Warrants to compensate certain finders.
07/24/2009—Received notification from the FDA that its IND for G-202 was on clinical hold pending the Company’s response to certain questions provided by the FDA. The questions concerned the design of GenSpera’s proposed Phase I clinical trial.

07/10/2009—Issued a Common Stock Purchase Warrant to purchase 150,000 Common Shares as reimbursement for due diligence expenses. The Warrants had a five-year term and entitled the holder to purchase GenSpera’s Common Stock at $3.00 per share.

06/29/2009—Entered into a Securities Purchase Agreement with a number of accredited investors. Per the terms of the agreement, GenSpera sold the investors units aggregating roughly $2,131,000 at a price of $1.50 per unit. Each unit consisted of the following: (1) one share of the Company’s Common Stock; and (2) one half Common Stock Purchase Warrant. The Warrants had a five-year term and allowed investors to purchase GenSpera’s Common Shares at $3.00 per share. The Warrants also contained anti-dilution protection in the event of stock splits, stock dividends, or other similar transactions. GenSpera incurred $142,467 in fees and expenses for the transaction, $50,000 of which had been paid through the issuance of 33,334 units. The Company also issued 43,894 additional Common Stock Purchase Warrants under the same terms as the investor Warrants to compensate certain finders.

Glossary

**Amino Acid**—Organic compounds that are the building blocks of many components found in cells. The sequence of amino acids affects how these components react biologically.

**Angiogenesis**—The physiological process involving the growth of new blood vessels from pre-existing vessels. It is a normal process in growth and development as well as in wound healing.

**Angiotoxic**—Toxic to blood vessels.

**Anti-angiogenic Drugs**—Drugs that attempt to interrupt tumor-associated angiogenesis. Current anti-angiogenic drugs, such as Genentech’s Avastin®, act by blocking the formation of new blood vessels supporting the tumor.

**Apoptosis**—Programmed cell death. This physiological process is necessary for the elimination of superfluous, diseased, or damaged cells.

**Assays**—Biological tests, measurements, or analyses to determine whether compounds have the desired effect either in a living organism, outside an organism, or in an artificial environment.

**Benign Prostatic Hypertrophy (BPH)**—Enlargement of the prostate gland, which surrounds the male urethra, causing frequent urination. This condition is common in older men.

**Cancer Stem Cells**—Self-renewing cells responsible for sustaining a cancer and for producing differentiated progeny that form the bulk of the cancer. Cancer stem cells identified in leukemia and certain solid tumors are believed to be critical therapeutic targets.

**Characterization**—Analysis that may include chemistry data, purity, potency, quality, stability, strength, pharmacokinetics, dose response, and efficacy.

**Chemotherapies**—The treatment of cancers with cytotoxic agents.

**Chronic Lymphocytic Leukemia**—The most common form of childhood leukemia, also known as lymphoblastic leukemia. In this disease, the bone marrow produces large quantities of immature lymphocytes (white blood cells).

**Contact Inhibition**—Cessation of cellular movement, growth, and division upon contact with other cells.

**CWR22R-H**—A cell line derived from human prostatic carcinoma.

**Cytosolic**—Of or pertaining to the cytosol, the aqueous part of the cytoplasm within which various particles and organelles are suspended.

**Cytotoxin**—Any drug that has a toxic effect on cells. Cytotoxic drugs are commonly used in chemotherapy to inhibit the proliferation of cancerous cells.

**DACH Platinum**—A form of platinum that has a similar mechanism of action to other derivatives of platinum. However, it is the only form of platinum shown to be clinically effective in the treatment of colorectal cancer.

**Doxorubicin**—A chemotherapeutic administered via injection to treat a variety of cancers, including bladder, breast, head and neck, some types of leukemia, ovary, and prostate cancers, among others.

**Drug Resistance**—The ability of bacteria and other disease-causing microorganisms to withstand a drug to which they were once sensitive (and were once stalled or killed outright).

**Endothelial Cells**—Cells that line the blood vessels of the body.
Enzymes—Any of several complex proteins that are produced by cells and act as catalysts in specific biochemical reactions. For GenSpera’s purpose, select enzymes found only at tumor sites remove the Company’s targeting/masking agent from 12ADT.

Good Laboratory Practice (GLP)—An international standard that provides a framework to plan, perform, monitor, record, report, and archive laboratory studies. These studies are undertaken to generate data by which the hazards and risks to users, consumers, and third parties can be assessed for pharmaceuticals (only preclinical studies), agrochemicals, cosmetics, and food additives, among others. These practices help to assure regulatory authorities that the data submitted are a true reflection of the results obtained during the study and can therefore be relied upon when making risk/safety assessments.

Good Manufacturing Practice (GMP)—The quality system regulation overseen by the FDA, which includes requirements related to the methods used in, and the facilities and controls used for, designing, manufacturing, packaging, labeling, storing, installing, and servicing medical devices intended for human use.

Half-Life—The time required for half the amount of a drug to be eliminated from the body.

Harvest Period Year—The period from May 1 until April 30 of the following year.

Hepatocellular Carcinoma—A carcinoma derived from parenchymal cells of the liver.

Human Glandular Kallikrein 2 (hK2)—A serine protease in the human kallikrein gene family that is 80% identical to PSA at the protein level. Similar to PSA, hK2 is expressed primarily in the prostate, thus making hK2 an attractive biomarker for prostate cancer development. HK2 is also the activating enzyme for GenSpera’s prodrug candidate Ac-GKAFRR-L12ADT.

IFL Chemotherapy—Concurrent treatment with irinotecan, leucovorin (folinic acid), and fluorouracil.

Infuse—To administer or inject by slowly but continuously introducing a solution into a vein.

Licochalcone A—Extract from Chinese licorice roots.

LNCaP—A widely studied metastatic prostate cancer cell line that is androgen responsive.

Lymphatic System—A network of vessels in the body, separate from blood circulation, that transports immune system cells to fight off germs, infections, and diseases. The lymphatic system includes the lymph nodes, which act as immune system command centers.

Maytansinoid—An agent derived from maytansine. Maytansine is isolated from the East African shrubs Maytenus serrata and M. buchananii. It demonstrates activity related to inhibiting or preventing the proliferation of tumor cells, most likely due to its inhibition of DNA synthesis.

Metastasizes—Cancer spreading from one part of the body to another.

Monoclonal Antibody—Any of a class of highly specific antibodies produced by the clones of a single hybrid cell formed in the laboratory by the fusion of a B-cell with a tumor cell. It is widely used in medical and biological research.

Monotherapy—A therapy that is effective on its own, rather than needing to be combined with other treatments.

Multi-Kinase Inhibitor—A therapy that targets several cancer pathways at once.

Non-Hodgkin’s Lymphoma—Malignant tumors of the lymphatic system consisting of several subtypes of lymphatic cancer.
Nude Mice—Immunologically deficient mice used to permit growth of tumor cells from mice or other species, such as humans.

Orphan Drug—A medication in development that seeks to treat an Orphan Disease, which is a rare illness affecting fewer than 200,000 people, or a common disease that has been ignored because it is less prominent in the U.S. compared with developing nations. According to the U.S. National Institutes of Health, there are approximately 7,000 of these diseases.

Peptides—Any compound consisting of two or more amino acids, the building blocks of proteins. For GenSpera’s purposes, when a specific peptide is attached to thapsigargin or 12ADT, it masks the activity of the cytotoxin. Once removed, the cytotoxin becomes active again.

Pharmacokinetics—The study of how and where drug levels are affected by absorption, distribution, metabolism, and elimination processes in the body.

Physicochemical—Relating to both physical and chemical properties.

Prodrug—A pharmacologic compound that is administered in an inactive form. Once absorbed by the body, it is metabolized and converted to the active form of the drug.

Prostate-Specific Antigen (PSA)—A protein in the blood produced by prostate tissue that serves as a tumor marker. PSA is also the activating enzyme for GenSpera’s prodrug candidates G-114 and G-115.

Prostate-Specific Membrane Antigen (PSMA)—An enzyme that is found in the blood vessels supporting tumors but not in normal blood vessels. It is also found in normal prostate cells and in prostate cancers. PSMA is the activating enzyme for GenSpera’s lead prodrug candidate, G-202.

Proteases—Enzymes that aid in the breakdown of proteins in the body.

PSMA Knockout Mice—Mice that lack the PSMA gene.

SERCA Pump—An intracellular protein that keeps cytosolic calcium low, allowing calcium to regulate important cellular processes such as cell growth, division, differentiation, cell death, and apoptosis (programmed cell death).

Substrate—The substance that is acted upon by an enzyme.

Targeting/Masking Agent—An agent that masks the toxicity of a cytotoxin while simultaneously helping target the cytotoxin to a tumor site.

Taxol® (Paclitaxel)—A chemotherapy drug administered via injection to treat several types of cancer, most commonly ovarian, breast, and non-small cell lung cancer.

Thapsigargin—A potent and novel cytotoxin extracted from the plant, *T. garganica*. It is 10- to 100-fold more potent than the National Cancer Institute’s reference chemotherapeutic agents and is capable of killing fast-, slow-, and non-dividing cancer cells.

TSU-Pr1—A cell line that is derived from the human bladder carcinoma.

Unresectable—Unable to surgically remove part or all of an organ or other structure.

Vasculature—The blood vessels or arrangement of blood vessels in an organ or part.

Xenograft—A surgical graft of tissue from one species to an unlike species.
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