ACORDA THERAPEUTICS INC
Form 10-K
March 02, 2009

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UNITED STATES
SECURITIES AND EXCHANGE COMMISSION
WASHINGTON, D.C. 20549

FORM 10-K

ý **ANNUAL REPORT PURSUANT TO SECTION 13 OR 15(d) OF THE SECURITIES EXCHANGE ACT OF 1934**

For the fiscal year ended December 31, 2008

OR

o **TRANSITION REPORT PURSUANT TO SECTION 13 OR 15(d) OF THE SECURITIES EXCHANGE ACT OF 1934**

Commission File Number 000-50513

ACORDA THERAPEUTICS, INC.
(Exact name of registrant as specified in its charter)

Delaware	**13-3831168**
(State or other jurisdiction of incorporation or organization)	(I.R.S. Employer identification number)

15 Skyline Drive
Hawthorne, New York 10532
(914) 347-4300
(Address, including zip code, and telephone number, including area code, of registrant's principal executive offices)

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

Title of each class	**Name of each exchange on which registered**
Common Stock $0.001 par value	The NASDAQ Stock Market LLC

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

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

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

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

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

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

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

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

As of June 30, 2008, the aggregate market value of the Registrant's voting stock held by non-affiliates was $538,607,470. For purposes of this calculation, shares of common stock held by directors, officers and stockholders whose ownership exceeds five percent of the common stock outstanding at June 30, 2008 were excluded. Exclusion of shares held by any person should not be construed to indicate that the person possesses the power, direct or indirect, to direct or cause the direction of the management or policies of the Registrant, or that the person is controlled by or under common control with the Registrant.

As of February 13, 2009, the registrant had 37,774,711 shares of common stock, par value $0.001 per share, outstanding. The registrant does not have any non-voting stock outstanding.

DOCUMENTS INCORPORATED BY REFERENCE

The registrant intends to file a proxy statement pursuant to Regulation 14A within 120 days of the end of the fiscal year ended December 31, 2008. Portions of the proxy statement are incorporated herein by reference into the following parts of the Form 10-K:

Part III, Item 10, Directors, Executive Officers and Corporate Governance
Part III, Item 11, Executive Compensation;
Part III, Item 12, Security Ownership of Certain Beneficial Owners and Management and Related Stockholder Matters;
Part III, Item 13, Certain Relationships and Related Transactions, and Director Independence;
Part III, Item 14, Principal Accounting Fees and Services.

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This Annual Report on Form 10-K contains forward-looking statements relating to future events and our future performance within the meaning of Section 27A of the Securities Act of 1933, as amended, and Section 21E of the Securities Exchange Act of 1934, as amended. Stockholders are cautioned that such statements involve risks and uncertainties. These forward-looking statements are based on current expectations, estimates, forecasts and projections about the industry and markets in which we operate and management's beliefs and assumptions. All statements, other than statements of historical facts, included in this report regarding our strategy, future operations, future financial position, future revenues, projected costs, prospects, plans and objectives of management are forward-looking statements. The words "anticipates," "believes," "estimates," "expects," "intends," "may," "plans," "projects," "will," "would," and similar expressions are intended to identify forward-looking statements, although not all forward- looking statements contain these identifying words. We may not actually achieve the plans, intentions or expectations disclosed in our forward-looking statements and you should not place undue reliance on our forward-looking statements. Actual results or events could differ materially from the plans, intentions and expectations disclosed in the forward-looking statements we make. We have included important factors in the cautionary statements included in this report, particularly in the "Risk Factors" section, that we believe could cause actual results or events to differ materially from the forward-looking statements that we make. Our forward-looking statements do not reflect the potential impact of any future acquisitions, mergers, dispositions, joint ventures or investments that we may make. We do not assume any obligation to update any forward-looking statements.

PART I

Item 1. Business.

Company Overview

We are a commercial-stage biopharmaceutical company dedicated to the identification, development and commercialization of novel therapies that improve neurological function in people with multiple sclerosis, or MS, spinal cord injury, or SCI, and other disorders of the central nervous system, or CNS. Our marketed product, Zanaflex Capsules, is approved by the U.S. Food and Drug Administration (FDA) for the management of spasticity. Our lead product candidate, Fampridine-SR, has completed two positive Phase 3 clinical trials for the improvement of walking ability in patients with MS and we submitted a New Drug Application (NDA) to the FDA at the end of January 2009 seeking approval to market it. We have discussed Fampridine-SR with national regulatory authorities in four European Union member states and believe that the current data are sufficient to file a centralized Marketing Authorization Application (MAA) with the European Medicines Agency (EMEA). Our preclinical programs target other aspects of MS, as well as SCI and other CNS disorders, and may also have application for other indications, such as stroke or congestive heart failure. We expect to file an Investigational New Drug (IND) application for Glial Growth Factor 2 (GGF2), the lead product candidate of our neuregulins program, for congestive heart failure in late 2009, pending the successful completion of toxicological and other work.

Approximately 400,000 people in the United States suffer from MS, and it is estimated that 64%-85% of those people experience walking disability. In Europe, approximately 600,000 people suffer from MS, and an additional 55,000-75,000 people in Canada are also diagnosed with this disease.

Our goal is to continue to grow as a fully-integrated biopharmaceutical company by commercializing pharmaceutical products, developing our product candidates and advancing our preclinical programs for these large and underserved markets.

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Company Highlights

Our lead product candidate, Fampridine-SR, completed a second positive Phase 3 clinical trial for improvement of walking ability in people with MS in June 2008 under a Special Protocol Assessment (SPA) from the FDA. An SPA is a process through which the sponsor of a trial seeks written agreement with the FDA regarding the design, size, and conduct of a Phase 3 clinical trial whose data will form the primary basis for an efficacy claim. In this trial, MS-F204, statistical significance was achieved on the primary outcome measure defined in the SPA. A significantly greater proportion of people taking Fampridine-SR had a consistent improvement in walking speed, compared to people taking a placebo. In addition, the effect was maintained throughout the eight-week treatment period, and there was a statistically significant improvement among Timed Walk responders compared to Timed Walk non-responders on the 12-Item MS Walking Scale (MSWS-12), a patient-reported self-rated assessment of walking disability. These clinical trial results were consistent with the results of our first Phase 3 trial, MS-F203, which we reported in September 2006 and which was also conducted under an SPA. The results of these two clinical trials formed the basis of our NDA filing in January 2009. A Thorough QT cardiac study was initiated in September 2007 and favorable results from that study were released in January 2008. This study evaluated the potential for the product candidate to cause an increase in the electrocardiographic QT interval. Fampridine-SR, at both therapeutic and supratherapeutic doses, was found to be no different than placebo. We believe Fampridine-SR is the first potential therapy in late-stage clinical development for MS that seeks to improve the function of damaged nerve fibers and, if approved, could be complementary to existing drugs used to treat MS. To our knowledge, there are no current therapies indicated to improve walking ability in people with MS. We expect to promptly launch and commercialize Fampridine-SR ourselves in the U.S., if it is approved, and are in discussions with potential marketing partners regarding the commercialization of Fampridine-SR in the European Union and other non-U.S. markets. At the same time, we are preparing for the filing of a centralized MAA with the EMEA. We plan to maintain our flexibility in the timing of such a filing in order to optimize our ex-U.S. commercialization pathway and availability to patients.

Sales of Zanaflex Capsules, which we launched in April 2005, and Zanaflex tablets increased from $43.6 million for the year ended December 31, 2007 to $53.4 million for the year ended December 31, 2008. Our Zanaflex Capsules and tablets commercial operations were cash flow positive in 2008. We acquired all marketing, sales and distribution rights in the United States to Zanaflex Capsules and Zanaflex tablets in 2004, based on the strategic fit of these products with our therapeutic focus and expertise. Both products are FDA-approved for the management of spasticity, a symptom of many CNS disorders, including MS and SCI. These products contain tizanidine, one of the two leading drugs used to treat spasticity. Zanaflex Capsules are the only approved capsule formulation of tizanidine and are protected by a patent that expires in 2021. We believe that Zanaflex Capsules offer important pharmacokinetic benefits over Zanaflex tablets and generic equivalents of Zanaflex tablets. As a result, Zanaflex tablets and generic tizanidine tablets are not AB-rated with Zanaflex Capsules by the FDA, meaning that the FDA does not consider the tablet products to be therapeutically equivalent to Zanaflex Capsules. Therefore, under state laws, pharmacists may not properly substitute tablets when filling a prescription for our proprietary Zanaflex Capsules.

As of February 13, 2009, our internal, field-based specialty sales force consisted of 61 sales professionals who call on neurologists, other specialists, and primary care physicians who treat patients with conditions that involve spasticity. We have a separate internal, field-based team responsible for payer strategy, as well as contracting and account management of managed care organizations, pharmacy benefit managers, specialty pharmacies, wholesale

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drug distribution customers, the Veterans Affairs institutions and the Department of Defense. We also engage a small, dedicated sales force of pharmaceutical telesales professionals to contact primary care physicians, specialty physicians and pharmacists. We believe that our sales and marketing infrastructure enables us to efficiently reach virtually all high-volume prescribers of Zanaflex tablets and generic tizanidine. We believe that many of these prescribers would also be potential high-volume prescribers for our lead product candidate, Fampridine-SR, if it is approved. We expect to approximately double the size of our existing sales force in anticipation of the potential launch of Fampridine-SR, if approved.

We have three preclinical programs focused on novel approaches to repair damaged components of the CNS. We believe all of our preclinical programs neuregulins, remyelinating antibodies and chondroitinase have broad applicability and have the potential to be first-in-class therapies. While these programs have initially been focused on MS and SCI, we believe they may be applicable across a number of CNS disorders, including stroke and traumatic brain injury, because many of the mechanisms of tissue damage and repair are similar. In addition, we believe that these programs may have applicability beyond the nervous system, including in such fields as cardiology, oncology, orthopedics and ophthalmology. In 2008, we began to work with a contract manufacturer to develop larger scale manufacturing and purification processes for one of the neuregulins, GGF2, under good manufacturing practices (cGMP) in preparation for a potential future IND application to support human clinical trials. We expect to file such an IND in late 2009, pending the successful completion of additional animal toxicology and other preclinical activities. We have also begun work with a contract manufacturer to scale up manufacturing of one of the remyelinating antibodies under cGMPs in preparation for a future IND application.

Background and Market Opportunity

The Challenge of Nervous System Disorders

The spinal cord and brain together comprise the CNS. The billions of nerve cells that make up the CNS, in conjunction with the nerve bundles that run through all parts of the body, which make up the peripheral nervous system, transmit the electrical impulses necessary to sustain, regulate and monitor every aspect of human life. The spinal cord serves as the master link between the brain and the body and carries information that regulates movement, sensation and involuntary functions, such as breathing, blood pressure, temperature control, and bladder, bowel and sexual functions.

Nerve impulses travel within and between the brain and spinal cord via long, thin fibers, or axons, that transmit information to other nerve cells through microscopic junctions called synapses. When axons are damaged or lost, they do not normally regenerate, and there is only very limited adaptability, or plasticity, of the surviving axons that allow them to take over the role of damaged or lost axons. The myelin sheath that surrounds axons in the brain and spinal cord provides insulation that facilitates the transmission of nerve impulses. We refer to the axon and its surrounding myelin sheath as a nerve fiber. The myelin sheath is composed of multiple layers of tightly packed cell membrane and is vulnerable to damage in conditions like MS and SCI. Once damaged, it is often not effectively repaired by the body. Although nerve fibers can survive in a demyelinated state, their ability to conduct nerve impulses may be completely lost or severely compromised.

Our Approach to the Market for CNS Disorders

We are focused on identifying, developing and commercializing novel pharmaceutical products that address large and underserved CNS markets. We view MS and SCI as the primary markets for

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our products as well as strategic points of access to a broad range of additional neurological conditions for the following reasons:

- Focusing on both MS and SCI provides insight into chronic and acute CNS conditions. MS is a chronic degeneration of the CNS, whereas SCI represents an acute CNS injury followed by a relatively stable chronic condition.

- Many of the mechanisms of secondary tissue damage and potential repair in MS and SCI are shared with other conditions, including stroke and traumatic brain injury.

- The functional deficits and symptoms suffered by MS and SCI patients, such as walking impairments, spasticity and loss of bladder and bowel function, are shared by other CNS disorders.

- A treatment that protects the spinal cord from the consequences of injury, regenerates neural connections, remyelinates or optimizes function of surviving structures in the spinal cord may also be applicable to many conditions affecting the brain and the rest of the nervous system.

For people with MS, SCI and similar chronic neurological conditions, even relatively small and incremental improvements in CNS function can produce meaningful benefits in their quality of life.

Multiple Sclerosis

The National Multiple Sclerosis Society, or NMSS, currently estimates that 400,000 people in the United States have multiple sclerosis. The NMSS estimates that the medical costs associated with treating MS in the United States were approximately $6.2 billion in 2004. Medications accounted for approximately $3.5 billion of these costs. MS is more prevalent in Caucasians and women and is generally diagnosed between the ages of 20 and 50.

MS is a degenerative CNS disorder in which the immune system attacks and damages the insulating myelin sheath. This damage, which can occur at multiple sites in the CNS, blocks or diminishes conduction of electrical impulses. People with MS may suffer impairments in any number of neurological functions. These impairments vary from individual to individual and over the course of time, depending on which parts of the brain and spinal cord are affected, and often include difficulty walking, spasticity, fatigue, lack of stamina and loss or disturbance of vision. They may also include loss of sensation, loss of bowel and bladder control, sexual dysfunction, depression, neuropathic pain, muscle paralysis, dizziness, tremors and cognitive difficulties. Individuals vary in the severity of the impairments they suffer on a day-to-day basis, with impairments becoming better or worse depending on the activity of the disease on a given day.

MS is generally classified by how the disease progresses. The most common classification is relapsing-remitting MS, in which people go through periods during which their disease is relatively stable or in remission, only to experience a recurrence of their disease, known as a relapse, which creates additional damage and loss of function. Approximately 10% of MS cases in the United States are diagnosed as primary progressive MS, which does not involve distinct attacks but rather a steady worsening of symptoms. Secondary progressive MS involves an initial period of relapsing-remitting disease followed by a steady worsening that is punctuated by more severe flare-ups and partial remissions. Most people with relapsing-remitting disease will eventually convert to secondary progressive disease, though this may not occur for many years.

There are no current treatments indicated to address the loss of mobility that is a major aspect of the progressive disability experienced by people with MS.

Spinal Cord Injury

According to the National Spinal Cord Injury Statistical Center (NSCISC), approximately 253,000 people in the United States live with the long-term consequences of SCI and approximately

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11,000 new spinal cord injuries occur each year, typically in young men. The majority of people with SCI are injured under the age of 40; life expectancies for persons with SCI continue to increase, but are still below life expectancies for those with no spinal cord injury. NSCISC estimates that the average lifetime costs directly attributable to SCI for an individual injured at age 25 varies from approximately $650,000 to $2.9 million depending on the severity of the injury.

The spinal cord can be injured by physical trauma that bends the neck or body violently, such as vehicular or diving accidents, or by objects that penetrate or impact the spinal cord, such as a bullet or a knife. The spinal cord can also be injured by tumor compression and loss of blood flow due to damage to major blood vessels or during surgical procedures. When an area of the spinal cord is damaged, motor and sensory function are partially or completely impaired throughout those parts of the body that are below the level of the injury.

Within the last two decades, researchers have shown that the spinal cord is not severed in most people with SCI. Rather, stretching or compression of the cord causes nerve fibers and blood vessels to tear and unleashes a secondary process of bleeding, loss of blood flow and inflammation that causes more tissue damage. The majority of people with spinal cord injury have some axons that survive within or around the site of injury. Because of these surviving axons, approximately 50% of people with SCI have some motor and/or sensory function remaining below the level of the injury and are said to have incomplete SCI. Those with no detectable function below the injury level are said to have complete SCI. Researchers have also shown that many axons that survive trauma are damaged and permanently lose part of their myelin sheath.

In addition to the impact of paralysis on mobility and independence, chronic SCI is often associated with several life-altering conditions that vary depending on the individual, location and the extent of injury. These include spasticity, as well as persistent pain, loss of control of bowel and bladder functions, loss of sexual function, compromised breathing, loss of sensation, and unstable control of blood pressure, heart rate and body temperature. There is no cure for SCI and no approved treatment available that is capable of improving neurological function. Methylprednisolone, a high-dose steroid, is currently the standard of care in the United States. Methylprednisolone is a one-time treatment administered to the patient immediately following an injury to reduce secondary tissue damage. There are several treatments for the symptoms of SCI, many of which are the same treatments used to address the symptoms of MS. We believe that novel therapies that offer even an incremental improvement in these conditions would have a meaningful impact on the quality of life for people with SCI.

Spasticity

Spasticity refers to the often painful involuntary tensing, stiffening or contracting of muscles. Spasticity is not a disease but a symptom of other conditions, such as MS, SCI, stroke, traumatic brain injury and cerebral palsy, where portions of the nervous system that control voluntary movement have been damaged. This damage results in the nerve cells in the spinal cord becoming disconnected from controlling centers in the brain and, as a result, transmitting unregulated impulses to the muscles. People who have spasticity may experience it intermittently it may be triggered by a stimulus, such as pain, pressure sores, cold weather or a urinary tract infection. The majority of people with MS and SCI experience some form of spasticity, as do many people following stroke or brain injuries. We Move, a non-profit organization dedicated to movement disorders, estimates that spasticity affects approximately 500,000 people in the United States and over 12 million worldwide.

Current treatments for spasticity are focused on reducing spasm frequency, pain or irritating stimuli that can provoke spasticity. Treatment of spasticity often involves a combination of physical therapy and oral medications. Baclofen and tizanidine, the active ingredient in the Zanaflex products, are the two most frequently prescribed oral medications for spasticity. For more

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intractable spasticity, treatments sometimes include surgical or chemical destruction of nerve roots in the affected area.

Other Disorders of the Central Nervous System

Neurological injuries and degenerative diseases of the CNS, including stroke, traumatic brain injury, Parkinson's disease and Alzheimer's disease, are among the most devastating and costly of human ailments. These conditions are most often chronic and historically have been extremely difficult to treat. These disorders, like MS and SCI, involve damage to nerve cells and nerve fibers and would likely benefit from similar approaches to tissue protection and repair. For example, the inflammation process that occurs naturally after many types of tissue injury may damage both injured and healthy CNS cells. As with MS and SCI, these conditions could be treated with interventions that replace nerve cells, stimulate new nerve fiber growth, or increase the adaptability of connections within the nervous system.

Our Strategy

Our strategy is to continue to grow as a fully-integrated biopharmaceutical company focused on the identification, development and commercialization of a range of nervous system therapeutics. We are using our scientific, clinical and commercial expertise in MS and SCI as strategic points of access to additional CNS markets, including stroke and traumatic brain injury. Key aspects of our strategy are:

Obtain regulatory approval for Fampridine-SR in MS in the U.S, the European Union and other key non-U.S. markets. One of our key objectives is to obtain regulatory approval of Fampridine-SR for commercial sale in the U.S. and to seek such approval in other key markets as well. In January 2009, we filed an NDA with the FDA for Fampridine-SR for the improvement of walking ability in people with MS. In 2008, we met with national regulatory authorities in four European Union member states to discuss Fampridine-SR. As a result, we believe that the current data are sufficient to file a centralized MAA with the EMEA. We also believe we can file a New Drug Submission (NDS) with Health Canada with our existing clinical trial data.

Leverage the commercial presence of Zanaflex Capsules and our sales and marketing organization for the potential launch of Fampridine-SR in the U.S. We expect that the sales and marketing organization that we have developed and the expertise that we are gaining with Zanaflex Capsules will provide a strong foundation for the commercial launch of Fampridine-SR, if approved by the FDA. Target prescribers for Zanaflex Capsules and Fampridine-SR are likely to overlap substantially. We expect to promptly launch and commercialize Fampridine-SR ourselves in the U.S., if it is approved, and to approximately double the size of our existing sales force in anticipation of that potential launch. Through our acquisition of the Zanaflex products, we have been able to strengthen our long-standing relationships with the physician and patient communities for both MS and SCI. We will also continue to focus on maximizing our revenue from Zanaflex Capsules.

Increase awareness of and help educate health care professionals and consumers about mobility and walking impairment issues for people with MS. Through our increased commitment to educational programs focused on MS, and our relationships with MS physician and consumer communities, we have been working to increase awareness of MS-related mobility and walking impairment and the impact on the individuals living with MS and their families and caregivers, and to increase the dialogue between physicians and patients regarding this impairment. For example, we have provided educational materials to physicians at major neurological conferences. Our consumer-focused activities have included our sponsorship of the National Multiple Sclerosis Society Walk MS programs and a

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published survey of patients and caregivers to assess the impact of walking disability on daily life.

Advance our pipeline of preclinical programs into clinical trials. We have increased our investment in our preclinical programs, with one such program focused on cellular protection, one on remyelination and one on nerve fiber regeneration and enhanced CNS plasticity. In order to advance these programs, we are using our in-house scientific expertise and animal modeling capabilities, supplemented by outside service providers and the development work of our partners. We are also seeking partnering and additional grant funding opportunities for these programs. We have begun development of cGMP manufacturing processes for two of our preclinical programs, GGF2 (neuregulins) and recombinant human IgM22 (rHIgM22, a remyelinating antibody). Based on the promising animal data to date and pending the successful completion of additional animal toxicology and other preclinical activities, we expect to file an IND for GGF2 in congestive heart failure in late 2009.

Pursue marketing strategy for European Union and other markets. We are in discussions with potential marketing partners regarding the commercialization of Fampridine-SR in the European Union and other non-U.S. markets. At the same time, we are preparing for the filing of a centralized MAA with the EMEA. We plan to maintain our flexibility in the timing of such a filing in order to optimize our ex-U.S. commercialization pathway and availability to patients.

Our Product Pipeline

Name	Status	Marketing Rights
Zanaflex Capsules	FDA-approved	U.S.
Zanaflex (tablets)	FDA-approved	U.S.
Fampridine-SR	NDA submitted	Worldwide
Neuregulin Program	Preclinical	Worldwide
Remyelinating Antibodies Program	Preclinical	Worldwide
Chondroitinase Program	Preclinical	Worldwide

Zanaflex Products

Zanaflex Capsules and Zanaflex tablets are short-acting drugs approved by the FDA for the management of spasticity. We acquired all of Elan Pharmaceuticals, Inc.'s (Elan's) U.S. sales, marketing and distribution rights to Zanaflex Capsules and Zanaflex tablets in July 2004. These products contain tizanidine, one of the two leading treatments for the management of spasticity. Zanaflex tablets were approved by the FDA in 1996 and lost compound patent protection in 2002. There are currently 12 generic versions of tizanidine tablets on the market. However, substantial brand loyalty remains in the prescriber community for the Zanaflex brand. Most prescriptions for tizanidine tablets are written as "Zanaflex," although the majority are switched automatically at the pharmacy for a generic tizanidine tablet. Zanaflex Capsules were approved by the FDA in 2002, but were never marketed by Elan. We began marketing Zanaflex Capsules in April 2005.

Clinical trials conducted by Elan demonstrated that Zanaflex Capsules, when taken with food, produce average peak levels of tizanidine in a person's blood that are lower and rise more gradually compared to the peak levels following a similar dose of the tablet form. The FDA recognizes these pharmacokinetic differences and therefore has determined that Zanaflex tablets and generic tizanidine tablets are not therapeutically equivalent, that is, are not AB-rated to Zanaflex Capsules. As a result, under state pharmacy laws, prescriptions written for Zanaflex Capsules may not be filled by the pharmacist with Zanaflex tablets or generic tizanidine tablets, although some

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substitution does take place in practice. Zanaflex Capsules are available in 2 mg, 4 mg and 6 mg doses, while tablet formulations are only available in 2 mg and 4 mg doses. Our goal is to convert sales of Zanaflex tablets and generic tizanidine tablets to sales of Zanaflex Capsules. We discontinued supply of the 2 mg dose of Zanaflex tablets in February 2006 due to a reduction in demand. Demand for the 4 mg Zanaflex tablet is also declining, but currently supports continued supply. The 6 mg capsule gives patients and physicians an additional dosing choice and an opportunity to reduce the number of pills a patient must take daily. In addition, many patients may find capsules easier to swallow than tablets. Also, people who have difficulty swallowing may open the capsule and sprinkle it on food. The pharmacokinetic effect of sprinkling contents of the capsule on food, however, is different from when the intact capsule is taken with food.

As sales have increased, we have seen an increase in the number of third-party payers who have implemented restrictions on the coverage of Zanaflex Capsules. These restrictions have included the implementation of prior authorization reviews or removal from formulary.

In 2008, retail sales of Zanaflex Capsules, Zanaflex tablets and generic equivalents of Zanaflex tablets totaled approximately $315 million. For the same period, retail sales of baclofen totaled approximately $178 million, for an approximate aggregate market of $493 million. The vast majority of these prescriptions were written by a relatively small group of prescribers. Specialists accounted for approximately 40% of tizanidine prescribing. High-volume specialist prescribers were responsible for approximately two to three-and-one-half times more prescriptions per physician than high-volume primary care prescribers. We believe that our internal specialty sales force, including our telesales team, is able to reach virtually all of these high-volume prescribers.

Sales and promotional support for Zanaflex Capsules

To support our commercialization of Zanaflex Capsules, we have established a sales and marketing infrastructure consisting of our internal specialty sales force and a pharmaceutical telesales group. As of February 13, 2009, our internal, field-based specialty sales force consisted of 61 sales professionals who call on neurologists, other specialists and primary care physicians and prescribers treating patients with conditions that involve spasticity, who are high volume prescribers of tizanidine. We have a separate internal, field-based team responsible for payer strategy, as well as contracting and account management of managed care organizations, pharmacy benefit managers, specialty pharmacies, wholesale drug distribution customers, the Veterans Affairs institutions and the Department of Defense. We also have a contract with TMS Professional Markets Group, LLC to provide a small, dedicated sales force of pharmaceutical telesales professionals to contact primary care physicians, specialty physicians and pharmacists. Our current sales and marketing infrastructure enables us to reach virtually all high-volume prescribers of Zanaflex tablets and generic tizanidine. We believe that these prescribers also would be potential high-volume prescribers for our lead product candidate, Fampridine-SR, if approved. Zanaflex Capsules and tablets commercial operations were cash flow positive in 2008 and are expected to be cash flow positive in 2009, with Zanaflex Capsules revenue expected to grow modestly during this period.

Concurrent with our launch of Zanaflex Capsules in April 2005, we initiated a sampling program as well as a number of educational, promotional and drug safety monitoring programs for prescribers and patients. In addition to our programs for prescribers and patients, we also have a number of programs in place to educate pharmacists about Zanaflex Capsules and the pharmacokinetic differences between tizanidine tablets, including generic tizanidine tablets and Zanaflex tablets, and Zanaflex Capsules.

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Pharmacokinetic differences between Zanaflex Capsules and tizanidine tablets

Although tizanidine, the active ingredient in Zanaflex Capsules, Zanaflex tablets and generic tizanidine tablets, is the same, there are some important differences between the capsule and tablet formulations. To establish the differences between Zanaflex Capsules and Zanaflex tablets, Elan conducted a series of studies. The largest study was a single dose clinical trial with 96 healthy volunteers. That trial demonstrated that Zanaflex Capsules, when taken with food, resulted, on average, in a more gradual rise in tizanidine plasma levels and a lower peak concentration. (Plasma is the fluid portion of blood without red and white blood cells and other proteins normally found in blood.) By contrast, the trial demonstrated that Zanaflex Capsules taken without food resulted in essentially the same pharmacokinetic profile as the tablet formulation of tizanidine. The results of the trial are illustrated in Figure 1 below.

Figure 1. Average Plasma Concentration Over Time

Average plasma concentrations of tizanidine in subjects following a single dose of two 4 mg Zanaflex tablets or two 4 mg Zanaflex Capsules, taken either with or without food.

As a result of the difference in absorption rate and plasma level when taken with food, the FDA determined that neither Zanaflex tablets nor generic tizanidine tablets are therapeutically equivalent, or AB-rated, to Zanaflex Capsules. Therefore, under most state pharmacy laws, pharmacists cannot fill prescriptions written for Zanaflex Capsules with Zanaflex tablets or generic tizanidine tablets. The FDA-approved package insert for Zanaflex Capsules contains the following language regarding the differences between the products: "Food has complex effects on tizanidine pharmacokinetics, which differ with different formulations. These pharmacokinetic differences may result in clinically significant differences when (1) switching administration of the tablet between the fed or fasted state, (2) switching administration of the capsule between the fed or fasted state, (3) switching between the tablet and capsule in the fed state, or (4) switching between the intact capsule and sprinkling the contents of the capsule on applesauce. These changes may result in increased adverse events or delayed/more rapid onset of activity, depending on the nature of the switch. For this reason, the prescriber should be thoroughly familiar with the changes in kinetics associated with these different conditions."

In July 2006, we received regulatory approval of a new package insert for Zanaflex which provides for updated safety information and enhanced differentiation between capsules and tablets.

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The new language adds that "ZANAFLEX CAPSULES ARE NOT BIOEQUIVALENT TO ZANAFLEX® TABLETS IN THE FED STATE. THE PRESCRIBER SHOULD BE THOROUGHLY FAMILIAR WITH THE COMPLEX EFFECTS OF FOOD ON TIZANIDINE PHARMACOKINETICS."

The most frequent adverse events associated with the use of tizanidine include dry mouth, drowsiness, fatigue and dizziness. These events are generally mild to moderate and are believed to be dose-related. In one single-dose study where patients were not titrated (that is, gradually increased in dose), two-thirds of patients experienced hypotension. Zanaflex Capsules have a short-acting effect, and patients are advised to take it at the times during the day when they most need relief from spasticity.

Fampridine-SR

Fampridine-SR is a small molecule drug contained in a sustained-release tablet form. Laboratory studies have shown that fampridine, the active ingredient in Fampridine-SR, improves impulse conduction in nerve fibers in which the myelin sheath has been damaged. Fampridine is not currently FDA-approved for use in MS or any other indications. Fampridine-SR is a sustained release formulation of fampridine that produces lower peak and more sustained blood levels than immediate-release formulation. We believe that Fampridine-SR could represent a fundamental shift in the treatment of people with MS because it may improve neurological function rather than treating the symptoms or slowing the progression of disease, as current treatments do. We have obtained Orphan Drug designations from the FDA for fampridine in both MS and incomplete SCI.

In MS, the myelin sheath is damaged by the body's own immune system, causing areas of myelin sheath loss, also known as demyelination. When a nerve fiber is demyelinated after injury, large numbers of the specialized potassium channels on the surface of the axon that are normally hidden or covered by the myelin sheath are exposed and leak potassium ions, causing the nerve fiber to short circuit its electrical impulses. Fampridine blocks these exposed channels, thereby simulating the insulation normally provided by the myelin sheath permitting the nerve fiber to transmit impulses again, even in a demyelinated state. Fampridine may also serve to amplify electrical signals at sites of contact or synapses between nerve cells by blocking the same channels in the tips of the nerve fiber, thereby improving the function of surviving tissue in the injured nervous system.

We have a worldwide, exclusive license from Elan for all of its rights to, among other things, develop, promote, distribute, use and sell Fampridine-SR in all human clinical indications, and to develop, promote, distribute, use and sell other patented sustained-release formulations of the drug. Elan also manufactures Fampridine-SR for us.

We believe there are compelling reasons to develop Fampridine-SR as a new therapy for improving walking ability in people with MS:

According to a patient registry maintained by the North American Research Committee on Multiple Sclerosis (NARCOMS), approximately 85% of people with MS experience some degree of walking impairment. This is considered one of the most limiting aspects of the disease. This figure includes individuals who report minimal walking disabilities through those who are no longer able to walk at all. Further research will be needed to identify the population of people with MS who are still able to walk but report regular problems with walking.

Market research has shown that, while there is a high incidence of walking impairment in MS patients, the diagnosis and treatment of such a condition by healthcare professionals goes largely unaddressed. This is due in part to the lack of drugs to improve walking ability in

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people with MS and the limited or short-lived effectiveness of other interventions, such as physical therapy.

Our Phase 3 clinical trials of Fampridine-SR in MS patients have consistently shown improvement in walking ability.

Based on its novel mechanism of action, Fampridine SR, if approved, could enhance the current standard of MS drug therapy that includes only disease modifying agents and therapies for symptom management.

Clinical Trials of Fampridine-SR

We have conducted a series of clinical trials to establish the safety, pharmacokinetics and optimal dosing of Fampridine-SR in MS and SCI, as well as to assess its efficacy. More than 1,600 people have been treated with Fampridine-SR in over 32 clinical trials.

In September 2006, we announced positive results from MS-F203, our first Phase 3 clinical trial of Fampridine-SR for the improvement of walking in patients with MS, which was performed under an SPA from the FDA. Statistical significance was achieved on all three efficacy criteria defined in the SPA. The FDA agreed in the SPA that this trial, if successful, could qualify as one of the pivotal efficacy studies required for drug approval. In May 2007, we reached agreement with the FDA in a second SPA for the second Phase 3 trial of Fampridine-SR in MS, MS-F204. Under the SPA, a single efficacy criterion was required, to replicate the primary result of the first Phase 3 study in showing a significantly greater proportion of patients treated with Fampridine-SR experienced a consistent improvement in walking speed, compared to those treated with placebo. We completed this trial in June 2008 and the data met the primary endpoint, as defined in the SPA. Although the FDA did not require that this trial also demonstrate maintenance of effect over the treatment period, nor that there be a statistically significant improvement in the MSWS-12 for Timed Walk responders versus Timed Walk non-responders, subsequent analysis of the MS-F204 data showed that both these additional outcomes were consistent with the earlier study. Pending review of the clinical results, the FDA has agreed that this trial, together with our first Phase 3 trial, MS-F203, would be adequate to provide evidence of efficacy for an NDA for Fampridine-SR.

Consistent with the FDA's recently established standard requirements for all new compounds, a Thorough QT cardiac study was initiated in September 2007 and favorable results from that study were released in January 2008. This study evaluated the potential to cause an increase in the electrocardiographic QT interval. Fampridine-SR, at both therapeutic and supratherapeutic doses, was found to be no different than placebo.

Completed Phase 3 Trials in Multiple Sclerosis

MS-F203. Results of our first MS Phase 3 clinical trial, MS-F203, were published in the February 28-March 6, 2009 edition of the medical journal, *The Lancet*. The trial was initiated in June 2005, pursuant to our SPA from the FDA. MS-F203 was a double-blind trial for which we enrolled a total of 304 patients at 33 MS clinical centers in the United States and Canada. Subjects completed a Timed 25-Foot Walking Test at each visit during the clinical trial, which included a 14-week treatment period. This test involves timing the subject's completion of a 25-foot walk as fast as he or she can do so safely. This test is widely used to measure walking function in patients with a range of diseases and conditions that affect mobility, and has been shown to relate closely to an individual's ability to walk longer distances. Neurologists employ this test as an indicator of the overall progression of MS, since many different pathways in the brain and spinal cord influence walking, including motor, sensory, position sense, balance and visual system pathways, as well as intrinsic locomotor pathways in the spinal cord.

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In addition, subjects were asked to fill out a 12 Item MS Walking Scale (MSWS-12) questionnaire. The MSWS-12 is a subjective measure of the degree to which walking disability impacts walking-related activities of daily life.

Statistical significance was achieved on all three efficacy criteria defined in the SPA. A significantly greater proportion of people taking Fampridine-SR had a consistent improvement in walking speed, the study's primary outcome, compared to people taking a placebo. In addition, the effect was maintained throughout the 14-week treatment period, and there was a statistically significant improvement among Timed Walk responders compared to Timed Walk non-responders in the MSWS-12.

Trial results were analyzed using our proprietary responder analysis that was accepted by the FDA in our SPA and for which we have applied for a patent. A subject was deemed to be a Timed Walk responder if his or her score on the 25-foot walk was better during the majority of his or her visits in the treatment phase of the trial, than the best visit during the non-treatment phase. The primary endpoint of the trial was the comparison of the percentage of Timed Walk responders in the Fampridine-SR group to the percentage of Timed Walk responders in the placebo group. To validate the clinical importance of improvements in the timed walk measurements, the MSWS-12 scores of the Timed Walk responders were compared against those of Timed Walk non-responders. This analysis was designed to ensure that being deemed a Timed Walk responder was clinically meaningful to the subject. In addition, the trial tested for significant improvement in walking ability in the Fampridine-SR-treated responder group at the last treatment visit versus the placebo group. This analysis was designed to ensure that the improvements seen by Timed Walk responders were maintained over the entire 14-week duration of the time on treatment. As a secondary outcome, the trial also measured lower extremity muscle strength, as assessed by the modified British Medical Research Council manual muscle testing procedures, referred to as the Lower Extremity Manual Muscle Test or LEMMT. Other secondary outcomes included a subject global and clinician global impression, each rated on a seven-point scale, and the Ashworth score, a measure of spasticity.

The design of the MS-F203 trial was closely modeled on the design of the preceding Phase 2 clinical trial, MS-F202, and built on our clinical trial experience in measuring improvements in neurological function against the variability in function that is inherent in people with MS. Individuals who suffer from MS vary in the severity of the impairments they experience on a day-to-day basis, depending on the activity of the disease on a given day. As a result, from one clinical trial visit to the next, a subject's walking ability can vary significantly. This variability makes it difficult to distinguish treatment-related changes in walking ability from disease-related changes in walking ability. Our review of data from our MS-F202 trial demonstrated that a responder form of analysis helps overcome the effect of the inherent variability of disease activity that people with MS experience.

MS-F204. Our second Phase 3 clinical trial (MS-F204) was initiated in March 2007, pursuant to a separate SPA from the FDA. This was a double-blind trial for which we enrolled a total of 240 patients at 39 MS clinical centers in the United States and Canada. As in the MS-F203 study, subjects completed a Timed 25-Foot Walking Test at each visit during the clinical trial, which included a 9-week treatment period. Subjects were also asked to fill out the MSWS-12 questionnaire.

Statistical significance was achieved on the single primary efficacy criteria defined in the SPA. A significantly greater proportion of people taking Fampridine-SR had a consistent improvement in walking speed, compared to people taking a placebo, using the same Timed Walk responder criterion employed in study MS-F203.

The only pre-defined secondary outcome in this trial was lower extremity muscle strength, as assessed by the LEMMT. Other measures were included in the study, without planned analysis, to

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allow comparison of overall trends between the two Phase 3 studies. These included the MSWS-12, the subject global impression and clinician global impression, and the Ashworth score for spasticity.

Figure 2, below, summarizes the results of the MS-F203 and MS-F204 studies for the three criteria defined in the SPA for MS-F203. For both studies, statistical significance was achieved on all three efficacy criteria defined in the SPA for MS-F203, although only the increased response rate was required in the SPA for MS-F204. A significantly greater proportion of people taking Fampridine-SR had a consistent improvement in walking speed measured by the Timed 25-Foot Walk compared to people taking placebo (*MS-F203*: 34.8% vs. 8.3%; *MS-F204*: 42.9% vs. 9.3%) ($p < 0.001$ for each study. A p-value is a statistical term that indicates the probability that the observed difference between the treatment groups could have been by chance alone. The smaller the p-value, the lower the likelihood that the observed difference was random. Generally a p-value of less than 0.05 is considered to represent a statistically significant difference.). In addition, the effect was maintained in these studies throughout the efficacy treatment period (14 weeks for MS-F203 and eight weeks for MS-F204; $p < 0.001$ for each study) and there was a statistically significant reduction in patient self-assessed walking disability as shown in the average change in the MSWS-12 for Timed Walk responders vs. Timed Walk non-responders (*MS-F203*: $p < 0.001$; *MS-F204*: $p < 0.001$).

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Figure 2. Summary Study Results for the SPA Criteria (Intent to Treat Population)

MS-F203 **MS-F204**

ABBREVIATIONS: FNR=Fampridine-SR Timed Walk Non-responders; FR=Fampridine-SR Timed Walk Responders

*
p-value versus Fampridine-SR Timed Walk Responder group.

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Change in Walking Speed over Time

Figure 3, below, summarizes the changes from baseline in walking speed over time for the two studies.

Figure 3. Percent Changes from Baseline in Walking Speed at each Double-Blind On-Treatment Efficacy Visit (Intent to Treat Population)

MS-F203

MS-F204

ABBREVIATIONS: FNR=Fampridine-SR Timed Walk Non-responders; FR=Fampridine-SR Timed Walk Responders

Note (Imputed): If a double-blind walking assessment was missing at scheduled time point, then the double-blind average was imputed for the missing value.

**:

Significantly better than placebo and Fampridine-SR Timed Walk Non-responders.

*:

Significantly better than placebo only.

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Results for the Fampridine-SR Timed Walk non-responders are also illustrated in this figure and show that there was a relatively small, transient improvement in average walking speed at the earliest visit in MS-F203, two weeks after initiation of treatment, though this was not seen in the MS-F 204 study. At later visits, there was no consistent difference between the non-responders and the placebo-treated groups.

Leg strength. A statistically significant improvement in leg strength, as measured by the average change from baseline in the LEMMT, was seen in Fampridine-SR Timed Walk responders compared to the placebo treated patients in both studies ($p<0.001$ in MS-F203 and $p=0.028$ in MS-F204). The Fampridine-SR Timed Walk non-responders were also statistically superior to the placebo group with respect to the average change from baseline in LEMMT during the double-blind period in the MS-F203 study ($p<0.046$) but not the MS-F204 study. This suggests that improved leg strength may contribute to walking speed improvement in some patients, but, based on data from individual patients, does not account for the improvement in walking ability among Timed Walk responders as compared to Timed Walk non-responders. The data also suggest that patients treated with Fampridine-SR may achieve functional benefits, such as improved leg strength, even if they do not have consistent improvement in walking speed.

Measurement of Walking Disability in MS. Our clinical trials have concentrated on walking because gradual loss of walking ability is a key physical problem for patients, a clear indicator of progression of MS, and widely used by neurologists to measure the neurological status of their patients. We have used the Timed 25 Foot Walk because it is the most standardized, objective measure that can be readily implemented in large, multi-center studies. A number of published studies have shown that walking ability measured with this test correlates well with other measures, such as the Six Minute Walk, that involve more extensive walking efforts. Changes in the Timed Walk, that are usually measured in seconds, are therefore representative of more substantial changes in the patient's walking-related daily activities, A number of studies have shown that changes of 20% in the Timed Walk correlate significantly with changes in broader measures of neurological status and disability.

Our two most recent trials have shown that approximately 35-43% of people with MS treated with Fampridine-SR have a consistent improvement in walking speed, measured with the Timed 25 Foot Walk. The average improvement in walking speed among Fampridine-SR Timed Walk responders was approximately 25%. Consistent with previous data on the clinical impact of changes in the Timed Walk, our trials showed that Timed Walk responders as a group reported significantly greater improvement in their self-assessed walking disability, as measured by the MSWS-12. The MSWS-12 is a questionnaire that was developed specifically to provide a reliable and valid patient-based measure of the impact of MS on daily activities that depend on walking.

Fampridine-SR Timed Walk responders were distributed across the full range of baseline disability, defined by our inclusion criterion of average walking times for the 25 Foot Timed Walk from eight to 45 seconds. Response to Fampridine-SR also appears to be independent of the type or duration of MS, as well as of concomitant treatment with other drugs or physical therapy. Response to Fampridine-SR also appears to be independent of gender, age, type or duration of MS, or concomitant treatment with other drugs.

Clinical Trials in Spinal Cord Injury

Recent clinical research using imaging and post-mortem studies has shown that the majority of people with SCI do not have severed spinal cords and maintain some nerve fibers that cross the site of injury. However, these surviving nerve fibers are often damaged and lose their myelin sheath. A series of preclinical studies and clinical trials have indicated that fampridine can potentially

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improve conduction in nerve fibers injured by spinal cord injury and improve function in people with spinal cord injury.

Phase 3 Clinical Trials. In March 2004, we released results from two Phase 3 double-blind clinical trials of Fampridine-SR in people with SCI. The trials did not reach statistical significance in their primary endpoints, which were reduction of spasticity, as measured by the Ashworth scale, and improvement of patients' Subject Global Impression, or SGI. The Ashworth scale is a validated, 5-point clinician assessment of an individual's spasticity. The SGI is a seven-point scale in which trial participants rate how they feel about the overall effect of the trial drug. In one of the SCI trials, the data showed a positive trend (p=0.069) toward improvement on the Ashworth scale when analyzed across all observations during the double-blind trial treatment period, which was the trial's pre-specified plan of analysis. When analyzed based on the subjects' last observation carried forward, a commonly used method of analysis, the improvement in, or reduction of, Ashworth score in that trial was statistically significant (p=0.006). The drug groups in both trials showed a progressive mean improvement on the Ashworth score during the double-blind treatment period. However, the placebo group in one of the trials showed a more pronounced reduction in Ashworth score than expected.

The design of these Phase 3 clinical trials was based on a series of earlier Phase 2 clinical trials in which the most consistent finding was a greater reduction in spasticity in Fampridine-SR-treated subjects relative to placebo-treated subjects, as measured by the Ashworth score. Other benefits observed in the Phase 2 trials were improved motor, bowel, bladder and sexual function. Unlike the design of our Phase 3 clinical trials, our Phase 2 clinical trials did not require a minimum spasticity level for enrollment and the treatment period was from one to four weeks rather than 14 weeks. These changes were made in the Phase 3 trials because the FDA required minimum twelve week duration of treatment for approval of a long-term therapy of this kind and because adequate measurement of benefit required a certain degree of spasticity at baseline.

Based on the entire body of data in clinical trials of fampridine in people with SCI and the new approaches to evaluating response to the drug that we have learned in MS trials, we may resume development of Fampridine-SR for SCI after we have completed further development of the drug for MS.

Safety Profile of Fampridine-SR

In addition to our placebo-controlled clinical studies, as part of our continuing evaluation of safety, we have established extension studies that allow subjects in completed clinic trials to receive Fampridine-SR on an unblinded, or open-label basis, with their progress followed at regular clinical visits. These studies are intended primarily to gain sufficient subject experience to satisfy the regulatory guidelines for long-term and overall safety assessments, though some additional uncontrolled efficacy data is also assessed. Under guidelines produced by the International Conference on Harmonization of Technical Requirements for Registration of Pharmaceuticals for Human Use, also known as the ICH Guidelines, it is usually expected that for consideration for drug approval at least 1,500 people will have been exposed to the drug, at least 300 for six months and at least 100 people for one year. Overall, Fampridine-SR has been taken by more than 1,600 subjects in our clinical studies. As of December 31, 2008, our extension studies in people with MS have included treatment of approximately 601 people for more than six months and 482 people treated for more than one year. Therefore we believe we have sufficient drug exposure to exceed the ICH Guidelines.

As of December 31, 2008, 177 subjects from MS-F202 had been enrolled in an extension trial and 92, or approximately 52%, remained active in the trial, with duration of treatment ranging over four years. As of the same date, 269 patients from MS-F203 had been enrolled in an extension

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study and 186 of these, or approximately 69%, remained active, with duration of treatment ranging over three years. Also, as of this same date, 214 patients from MS-F204 had been enrolled in a third extension study and 185, or approximately 86%, remained active, with a duration of treatment ranging up to 17 months. The total exposure to Fampridine-SR in our MS studies to that date, including both double-blind and open label studies, is over 1400 patient-years. By contrast, in our controlled trials we have collected safety data on placebo treatment that totals to approximately 54 patient-years.

The adverse events most commonly experienced in the MS-F203 and MS-F 204 studies were urinary tract infection, falls, insomnia, dizziness, headache, nausea, asthenia (weakness), upper respiratory track infection, back pain, balance disorder and fatigue. The majority of these events were mild to moderate in intensity. Among these types of event, only insomnia, dizziness, headache, nausea, back pain and balance disorder were seen at more than double the frequency in the Fampridine-SR-treated than the placebo-treated patients in our controlled trials.

Seizures have been reported in a small number of subjects over the course of the development program and have also been reported in cases of overdose with fampridine outside the program. We are carefully monitoring the potential for seizure as a side effect, including the possibility of interaction with other drugs that are known to lower the threshold for seizure in susceptible subjects. The incidence of seizures appears to be dose-related. Overall, the incidence of seizures at the current dose of 10 mg twice a day has been within the rates reported for placebo-treated groups in long-term, controlled studies of interferon drugs in MS patients. These rates have ranged up to 2% of patients in a two year study, or 1 seizure per 100 patient years. Incidence of seizures has been reported to be higher in actively treated groups in studies of interferon drugs, ranging up to 5% over a two year study or 2.5 seizures per 100 patient years. The proportion of patients treated with beta interferons in our studies of Fampridine-SR has been in the range of 40-45%.

We have excluded from our studies subjects known to be at risk for seizures because they have had seizures previously or because they have an abnormal electroencephalogram indicative of such risk, which was not the case for studies of interferon drugs. We have therefore also compared the incidence of seizure events in the open label extension studies with the expected incidence rates for a first seizure in the MS population, based on the epidemiological literature, and find these rates to be comparable for the 10 mg twice a day dose of Fampridine-SR. With our current knowledge, there are no means of determining in advance which individual patient may face an increased seizure risk when taking Fampridine-SR

Depending on dose, fampridine is known to block a wide range of potassium ion channels in cell membranes, which are potentially important not only in the nervous system but also in the heart. A Thorough QT cardiac study was initiated in September 2007 and favorable results from that study were released in January 2008. This study evaluated the potential to cause an increase in the electrocardiographic QT interval. Fampridine-SR, at both therapeutic and supratherapeutic doses, was found to be no different than placebo. In addition we have completed studies to examine the specific effects of the drug on the cardiac potassium channels of principal interest from the point of view of cardiac safety, the human ether-a-go-go related gene or hERG channel. These are standardized tests of the potential for a drug to affect the QT interval, a measure of heart function. Prolongation of the QT interval is believed to be a risk factor for triggering potentially fatal cardiac arrhythmias. These laboratory studies showed that fampridine blocks the hERG channel by 50% at a concentration which is approximately ten thousand times the average peak concentration expected in the blood of patients taking 10 mg doses of Fampridine-SR. Based on these observations, fampridine would not be expected to affect the hERG channel at clinically relevant concentrations. In another standard test, we have also performed studies on isolated dog cardiac Purkinje fibers. These showed no effect on the electrical behavior of these heart cells in the range of concentrations relevant to clinical experience, including concentrations 100 times higher than the

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expected average peak levels in the blood of patients. Additional studies of cardiac safety in dogs showed no notable changes in cardiac electrical behavior or function, up to maximum tolerated doses.

Other Research and Development Programs

Remyelination Programs

Our remyelination programs include two distinct therapeutic approaches to stimulate repair of the damaged myelin sheath in MS, neuregulins/GGF2 and remyelinating antibodies. These two approaches address remyelination by different and potentially complementary routes. Both programs require finalizing production of clinical-grade material and completion of preclinical toxicology tests before moving into clinical development. We believe a therapy that could permanently repair myelin sheaths has the potential to restore substantial neurological function to those affected by demyelinating conditions.

Neuregulins/GGF2

The neuregulins form a family of growth factors related to epidermal growth factor. These molecules bind to erbB receptors, which translate the growth factor signal to the cell and cause changes in cell growth, protein production and gene expression. Neuregulins have been shown in published studies to have a range of effects in protection and repair of cells both in the nervous system and in the heart. In 2002, we obtained from CeNeS Pharmaceuticals plc., or CeNeS, an exclusive worldwide license to its neuregulin patents and related technology, including GGF2, our lead molecule for the neuregulin family.

Neuregulins covered in the portfolio from CeNeS have a number of potential applications. Neuregulins and their erbB receptors are essential for cardiac development and have been shown to protect cardiac muscle cells from stressors that can lead to congestive heart failure, including myocardial infarction. Additionally, neuregulins have been shown to protect the heart and brain from the toxicity of commonly used chemotherapeutic agents, such as anthracyclines. Studies in mouse, rat and dog models of congestive heart failure have shown that neuregulins significantly improve cardiac function and survival. Neuregulins have been shown to stimulate remyelination in animal models of MS and to protect the brain in animal models of stroke. Therefore, the neuregulins offer us the potential for multiple CNS and cardiac indications, including MS, stroke and congestive heart failure as well as protection from chemotherapy-induced damage. In 2008, we began to work with a contract manufacturer to develop production and purification methods for manufacturing GGF2 under cGMP in preparation for a potential future IND application to support human clinical trials. We anticipate filing an IND in late 2009, subject to the results of ongoing toxicological studies and other preclinical activities.

Remyelinating Antibodies Program

Our remyelinating antibodies program is based on research performed at Mayo Foundation for Medical Education and Research, or Mayo Clinic. Under a license agreement entered into with Mayo Clinic in September 2000, we have exclusive worldwide rights to patents and other intellectual property for these antibodies related to use and treatment of CNS disorders. Studies have demonstrated the ability of this family of antibodies to stimulate repair of the myelin sheath in three different animal models of MS. In particular, these antibodies were found to react with molecules on the surface of the cells that make the myelin sheath and stimulate them in a number of ways, leading to increased remyelination activity. First identified in mice, similar antibodies were subsequently identified in human blood samples by the Mayo Clinic and we have been able to produce a recombinant human antibody that may be suitable for clinical development.

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We have also supported preclinical studies at Mayo Clinic to learn more about the ways the antibodies act to stimulate the myelin sheath-forming cells. In 2004, Mayo Clinic received a $2 million grant to develop and manufacture clinical-grade material and progress the program towards clinical development. In May 2006, Mayo Clinic and the FDA had a pre-IND meeting to discuss the details of a preclinical development program. We have been working with a contract manufacturer to produce rHIgM22, one of these antibodies, under cGMP, and had previously anticipated filing an IND in late 2009, but this activity has been delayed due to the manufacturer's filing for bankruptcy in 2008.

Chondroitinase Program

We have developed a program based on the concept of breaking down the matrix of scar tissue that develops as a result of an injury to the CNS. Published research has demonstrated that this scar matrix is partly responsible for limiting the regeneration of nerve fibers in the CNS. A similar matrix exists even in uninjured parts of the CNS tissue and restricts plasticity, the ability to modify or re-establish nerve connections. One or both forms of matrix may also inhibit repair of the myelin sheath by restricting the movements of the myelinating cells into the area of damage.

A major component of these two forms of matrix are chondroitin sulfate proteoglycans, or CSPGs. Cell culture studies and a number of animal studies have shown that these CSPGs inhibit the growth of nerve fibers and are likely to be key factors in the failure of the spinal cord or brain to regenerate and repair. Studies also have shown that bacterial enzymes called chondroitinases break down the CSPG molecules, thereby reducing their inhibitory activity.

Six independent laboratories have published animal studies showing that application of chondroitinase results in recovery of function following injuries to various areas of the brain or spinal cord. These functions have included walking, forelimb grasping, sensation, and visual and bladder function. We have successfully tested the ability of one of these molecules, Chondroitinase ABC-I, to improve function in an animal model of spinal cord injury. These studies were published in the Journal of Neurotrauma in February 2005. In these studies, rats that sustained a spinal cord injury were treated with either chondroitinase or an ineffective enzyme control and evaluated over 10 weeks of recovery. Animals treated with chondroitinase showed significant improvements both in motor function of the limbs and in bladder function, compared to those treated with the control enzyme. We have also produced and successfully tested in animal models a recombinant version of naturally-occurring Chondroitinase ABC-I.

We are conducting a research program, which has been funded in part by federal and state grants, to develop second generation approaches to overcoming the proteoglycan matrix. These include novel enzyme molecules and alternative approaches to blocking matrix formation. We are exploring the possibility of obtaining additional research grants from the NIH as well as potential partnerships with other companies to support completion of our preclinical program in chondroitinase. In 2003, we obtained an exclusive worldwide license to certain patents and technology from Cambridge University Technical Services Limited and King's College London related to our chondroitinase program. We are also building our intellectual property position with respect to this technology with patent applications around uses of the known compound and new chemical structures.

Sales and Marketing

We have established two sales channels for marketing Zanaflex Capsules: an internal, field-based specialty sales force and an external telesales group.

Internal, Field-Based Specialty Sales Force. We employ a team of highly experienced sales professionals to call on neurologists and other prescribers who specialize in treating people

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with conditions that involve spasticity. Our sales professionals have had an average of approximately 14 years of sales experience prior to joining us. Our specialty sales force of 61 sales professionals calls on neurologists, other specialists, and primary care prescribers treating patients with conditions that involve spasticity, and who are high volume prescribers of tizanidine. We expect to approximately double the size of our sales force in anticipation of the potential launch of Fampridine-SR, if approved.

Internal Managed Care Team. We employ an internal and field-based team responsible for payer strategy, as well as contracting and account management of managed care organizations, pharmacy benefit managers, specialty pharmacies, wholesale drug distribution customers, the Veterans Affairs institutions and the Department of Defense.

Contract Pharmaceutical Telesales Organization. We have retained TMS Professional Markets Group, LLC (which purchased various telesales assets from Access Worldwide Communications, Inc., with whom we had previously contracted) to provide a small, dedicated sales force of pharmaceutical telesales professionals to contact primary care physicians and specialty physicians to determine their interest in receiving samples of Zanaflex Capsules or a visit from one of our sales representatives. TMS Professional Markets Group also contacts pharmacies to assist us in educating pharmacists that Zanaflex Capsules are not interchangeable with Zanaflex or tizanidine tablets.

We believe that, in general, people with MS and SCI are knowledgeable about their conditions, actively seek new treatments, and directly influence their prescriber's evaluation of treatment options. We have existing relationships with the major advocacy groups that focus on MS and SCI. We provide regular scientific updates regarding our development programs and we sponsor or support several educational initiatives. We have implemented a comprehensive series of educational and promotional programs to support Zanaflex Capsules. These include educational materials, a peer-to-peer speakers' program, samples, medical information and drug safety monitoring services, as well as a patient assistance program. At the request of the FDA, we have also implemented an educational program to inform pharmacists, prescribers and patients that Zanaflex tablets or generic tizanidine tablets are not therapeutically equivalent to Zanaflex Capsules and that, as a result, a prescription for Zanaflex Capsules should not be substituted with any tablet formulations at the pharmacy.

Similar to other pharmaceutical companies, our principal customers are wholesale pharmaceutical distributors. We currently depend on three key customers. For the year ended December 31, 2008, Cardinal Health, McKesson Corporation and AmerisourceBergen Corporation accounted for approximately 46.4%, 33.7% and 14.7% of our shipments, respectively.

We believe that the expertise we are developing through commercializing Zanaflex Capsules will provide a strong foundation for our marketing of Fampridine-SR, if approved, as well as for additional potential treatments in CNS conditions. As a result, we plan to promptly launch and market Fampridine-SR ourselves in the United States and possibly in Canada, if it is approved in both countries. We are currently developing and implementing programs to educate consumers, physicians, and other healthcare professionals about the challenges of walking disability in the MS population. For example, in 2008, Acorda was a national sponsor of the National Multiple Sclerosis Society's Walk MS program. This sponsorship allowed us to engage thousands of people with MS, as well as their families, physicians and caregivers, in a discussion about the impact of walking disability on their lives. We will be a national sponsor once again in 2009 and in 2010.

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Scientific and Medical Network

We have an established advisory team and network of well-recognized scientists, clinicians and opinion leaders in the fields of MS and SCI. Depending on their expertise, these advisors provide assistance in trial design, conduct clinical trials, keep us apprised of the latest scientific advances and help us identify and evaluate business development opportunities.

Collaborations, Alliances and License Agreements

Elan Corporation plc

Fampridine-SR

In January 1997, we licensed from Elan exclusive worldwide rights to Elan's sustained release formulation of fampridine, Fampridine-SR, for the treatment of SCI. In April 1998, we formed MS Research & Development Corporation, or MSRD, with Elan's subsidiary, Elan International Services, Ltd., or EIS, to develop Fampridine-SR for treatment of MS. At that time, MSRD licensed from Elan exclusive worldwide rights to Fampridine-SR for the treatment of MS.

In September 2003, we entered into a termination and assignment agreement with Elan, EIS and MSRD pursuant to which MSRD assigned to us its assets, including the license from Elan for Fampridine-SR for MS. We paid MSRD approximately $11.5 million for all the assets and assumed liabilities of MSRD. MSRD distributed the purchase price to its shareholders according to their equity ownership interest. We received a distribution of approximately $9.5 million. We also purchased EIS's shares at par value, and own approximately 88% of MSRD, which now has no assets or liabilities and is inactive.

In September 2003, we entered into an amended and restated license with Elan, which replaced the two prior licenses for Fampridine-SR in oral sustained release dosage form. Under this agreement, Elan granted us exclusive worldwide rights to Fampridine-SR for all indications, including SCI, MS and all other indications. We agreed to pay Elan milestone payments of up to $15.0 million and royalties based on net sales of the product, if approved. We have not made any payments under this agreement through December 31, 2008.

Elan is also supplying us with product for our clinical trials under this agreement.

Elan may terminate our license in countries in which we have a license, including the United States, if we fail to file regulatory approvals within a commercially reasonable time after completion and receipt of positive data from all preclinical and clinical studies required for the related NDA or any NDA equivalent. We could also lose our rights under the license agreement if we fail to launch a product in such countries within 180 days of NDA or equivalent approval or if we fail to fulfill our payment obligations under the license agreement. If Elan terminates our license in any applicable country, Elan is entitled to license from us our patent rights and know-how relating to the product and to market the product in the applicable country, subject to royalty payments to us.

We have the right to terminate the Elan license at any time by written notice. In addition, the Elan license may be immediately terminated by either party following an incurable breach of any term or provision by the other party. The Elan license may also be terminated by either party following notice and a cure period with respect to an uncured breach by either party.

Subject to the early termination provisions, the Elan license terminates on a country by country basis on the last to occur of fifteen years from the date of the agreement, the expiration of the last to expire Elan patent or the existence of competition in that country.

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Zanaflex

In July 2004, we entered into an Asset Purchase Agreement with Elan pursuant to which we acquired all of Elan's research, development, distribution, sales and marketing rights to Zanaflex Capsules and Zanaflex tablets in the United States. The assets acquired include the products' FDA registrations and FDA dossiers, proprietary product know-how, a patent and two related patent applications, certain inventory of Zanaflex tablets and certain product books and records. Elan also granted us a license allowing us to use the Zanaflex trademarks in the United States, with the right to buy the Zanaflex trademark for a nominal sum once specified milestone and royalty payments were made. Those payments have been made, and we purchased and now own the trademarks. Elan also granted us an exclusive, perpetual and royalty-free license to certain intellectual property relating to technology contained in Zanaflex Capsules and Zanaflex tablets or used in the manufacture of Zanaflex Capsules, for use in connection with the sale and marketing of Zanaflex Capsules and Zanaflex tablets in the United States. We also acquired the right to develop new indications, formulations, dosage forms, delivery systems and process improvements of Zanaflex. Under the agreement, Elan agreed not to directly or indirectly market, distribute or sell any products containing tizanidine as an active pharmaceutical ingredient in the United States until the later of the end of our obligation to pay royalties to Elan or valid termination of our supply agreement with Elan. In addition, we agreed not to directly or indirectly market, distribute or sell any products containing tizanidine as its active pharmaceutical ingredient in the United Kingdom or Ireland until July 2007.

Our agreement with Elan obligated us to pay a combination of sales-based milestone payments of up to $19.5 million, all of which have been achieved and paid, and royalties on sales of Zanaflex Capsules and Zanaflex tablets. We have no further Zanaflex milestone payment obligations with Elan. We also agreed to use commercially reasonable efforts to commercialize Zanaflex Capsules.

As part of the acquisition, we assumed certain of Elan's rights and obligations relating to Zanaflex under a license agreement with Novartis, to the extent that these rights and obligations arise subsequent to our acquisition of Zanaflex. Under this agreement we obtained certain rights to market and sell tizanidine products and rights to product improvements developed by Novartis.

Elan manufactures Zanaflex Capsules for us and we are in contract negotiations with Patheon Inc. for the manufacture of Zanaflex tablets. See " Manufacturing."

In December 2005, we entered into a financing arrangement with Paul Royalty Fund, or PRF, pursuant to which we assigned PRF the right to receive a portion of our net revenues from Zanaflex Capsules, Zanaflex tablets and any future Zanaflex products. This agreement was amended in November 2006 potentially to increase the total amount of royalty payments to which PRF is entitled and to provide for additional lump-sum payments both from us to PRF and from PRF to us. The arrangement covers all Zanaflex net revenues generated from October 1, 2005 through and including December 31, 2015, unless the arrangement is terminated earlier. See "Management's Discussion and Analysis of Financial Condition and Results of Operations Liquidity and Capital Resources Financing Arrangements."

Rush-Presbyterian St. Luke's Medical Center

In 1990, Elan licensed from Rush-Presbyterian St. Luke's Medical Center, or Rush, know-how relating to fampridine for the treatment of MS. We subsequently licensed this know-how from Elan. In September 2003, we entered into an agreement with Rush and Elan terminating the Rush license to Elan and providing for mutual releases. We also entered into a license agreement with Rush in 2003 in which Rush granted us an exclusive worldwide license to its know-how relating to

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fampridine for the treatment of MS. Rush has also assigned to us its Orphan Drug Designation for fampridine for the relief of symptoms of MS.

We agreed to pay Rush a license fee, milestone payments of up to $850,000 and royalties based on net sales of the product for neurological indications. We have made an aggregate of $100,000 in payments under this agreement through December 31, 2008. The Rush license may be terminated by either party following an uncured material breach by the other party and notice. The Rush license may also be terminated upon the filing or institution of bankruptcy, reorganization, liquidation or receivership proceedings, or upon an assignment of a substantial portion of the assets for the benefit of creditors by the other party. We also entered into an agreement with Elan relating to the allocation of payments between us and Elan of certain payments to Rush under the Rush license. Subject to the early termination provisions, the Rush license terminates upon expiration of the royalty obligations, which expire fifteen years from the date of the agreement.

Canadian Spinal Research Organization

In August 2003, we entered into an Amended and Restated License Agreement with the Canadian Spinal Research Organization (CSRO). Under this agreement we were granted an exclusive and worldwide license under certain patent assets and know-how of CSRO relating to the use of fampridine in the reduction of chronic pain and spasticity in a spinal cord injured subject.

We are required to pay to CSRO royalties based on a percentage of net sales of any product incorporating the licensed rights, including royalties on the sale of Fampridine-SR for any indication. No royalty payments have been made to date.

We have the right to terminate the CSRO agreement at any time by written notice. In addition, the CSRO agreement may be terminated by either party following an uncured material breach by the other party. The CSRO agreement may also be terminated by either party upon the filing or institution of bankruptcy, reorganization, liquidation or receivership proceedings, or upon an assignment of assets, by the other party. Subject to the early termination provisions, the CSRO agreement will expire upon the termination of all royalty or other payment obligations on a country-by-country basis, which will be no longer than the earlier of the expiration of the last to expire licensed patent in such country or ten years from the date of the first commercial sale of the product in such country.

Cornell Research Foundation, Inc.

In February 2003, we entered into a license agreement with Cornell Research Foundation, Inc., or Cornell, pursuant to which we were granted an exclusive license under a patent for the use of fampridine in the treatment of anterior horn cell diseases. In consideration for the license, we paid Cornell an upfront license fee and are required to make payments of up to $150,000 to Cornell upon the achievement of certain milestones relating to the successful reissuance or reexamination of the patents licensed to us and, the completion of a clinical trial testing the use of Fampridine-SR in amyotrophic lateral sclerosis. We have made an aggregate of $50,000 in payments under this agreement through December 31, 2008. We are also obligated to pay Cornell an annual royalty on net sales of Fampridine-SR in any and all indications, subject to a minimum annual royalty requirement of $25,000.

Under the Cornell agreement, Cornell is responsible for all patent prosecution and maintenance activities relating to the licensed patent, and we are responsible for paying all fees incurred by Cornell in connection therewith. We have the right under this agreement to enforce any patent rights within the licensed patents against infringement by third parties at our own expense.

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We have the right to terminate the Cornell agreement at any time by written notice. In addition, the Cornell agreement may be terminated by either party following an uncured material breach by the other party. Subject to the early termination provisions, the term of the Cornell agreement will continue until the expiration of the last to expire valid claim under the licensed patent.

Cambridge University Technical Services Limited and King's College London

In December 2003, we entered into a license agreement with Cambridge University Technical Services Limited and King's College London, pursuant to which we were granted an exclusive worldwide license, including the right to sublicense, under a U.S. patent application and its foreign counterpart to develop and commercialize products related to enzymatic methods, including chondroitinase, of treating CNS disorders. We were also granted a non-exclusive worldwide license, including the right to sublicense, under the same U.S. and foreign patent applications to develop and commercialize products related to small molecule inhibitors for use in treating CNS disorders.

In consideration for these licenses, we paid an upfront license fee and are required to make payments of up to $2.15 million upon the achievement of certain milestones. We have made an aggregate of $45,000 in payments under this agreement through December 31, 2008. We are also obligated to pay royalties on net sales and on any sublicense royalties that we receive.

The King's College license may be terminated by any party following an uncured material breach by any other party. The King's College license may also be terminated by any party if any other party ceases to carry on business, is declared by a court of competent jurisdiction to be bankrupt or upon the appointment of a liquidator of that party. Subject to the early termination provisions, the King's College license agreement will continue until the expiration of the last to expire valid claim under the licensed patent applications, at which time the licenses granted under the license agreement will automatically become non-exclusive, worldwide, fully paid-up and irrevocable.

Mayo Foundation for Medical Education and Research

In September 2000, we entered into a license agreement with Mayo Foundation for Education and Research, or Mayo Clinic, pursuant to which we were granted an exclusive worldwide license to its patents and other intellectual property on remyelinating antibodies. Under this agreement, we have the right to develop, make, use and sell the remyelinating antibody products for the prevention, mitigation and treatment of CNS disorders. We have worked closely with one of Mayo Clinic's research groups on developing and patenting this emerging technology in connection with the therapeutic use of these antibodies, specifically myelination and remyelination in MS and SCI. Mayo Clinic has the right to continue researching the antibodies and, in the event it develops other applications related to the licensed patent, which are outside of the scope of our current license, but are for the treatment of CNS disorders. Mayo Clinic is required to offer rights in these new applications to us before it offers such rights to a third party.

Under the Mayo Clinic agreement, we are obligated to make milestone payments of up to $1.875 million. We also pay royalties based on net sales. We have not made any milestone or royalty payments under this agreement through December 31, 2008. The Mayo Clinic agreement may be terminated by either party following an uncured material breach by the other party. We may terminate the Mayo Clinic agreement at will upon prior written notice to Mayo Clinic. In addition, either party also has the right to terminate upon the insolvency of the other party, the filing of bankruptcy by or against the other party, or the assignment of assets to the benefit of creditors by the other party. Unless otherwise terminated, this license agreement will terminate upon the expiration of the last licensed patent in any such licensed product.

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We have also supported preclinical studies at Mayo Clinic to learn more about the ways the antibodies act to stimulate the myelin sheath-forming cells. In 2004, Mayo Clinic received a $2 million grant to develop and manufacture clinical-grade material and progress the program towards clinical development. A subsequent letter agreement between Mayo Clinic and us acknowledges that the work under this grant is being performed subject to and pursuant to the Mayo Clinic agreement.

CeNeS Pharmaceuticals plc

In November 2002, we entered into two license agreements with CeNeS Pharmaceuticals plc, or CeNeS. The first agreement relates to an exclusive worldwide sublicense under certain patents, patent applications and know-how to make, have made, use, import, offer for sale and sell protein products composed of GGF2 and non-protein products developed through the use of material covered by a valid claim in the patents. The license to these patents and the right to sub-license these patents were granted to CeNeS by the Ludwig Institute for Cancer Research.

Our payment obligations to CeNeS include payment of an upfront license fee, royalties based on annual net sales of the product, if any, as well as payments of up to $8.5 million upon achieving certain milestones in connection with the development, testing and regulatory approval of any protein products. We have not made any payments under this agreement through December 31, 2008. We are obligated to make minimum royalty payments commencing on the third calendar year following the first commercial sale of any licensed product. If we fail to pay any minimum royalty, CeNeS will have the option to convert our license or any sublicense to a non-exclusive license. This agreement with CeNeS is effective until the later of November 12, 2017 or the expiration of the last-to-expire valid claim in the licensed patents. We may terminate this agreement at will upon prior written notice to CeNeS. In addition, this first agreement may be terminated by either party following an uncured material breach by the other party and if this agreement is terminated under that provision, we may retain the exclusive worldwide sublicense granted to us under this agreement, provided that we continue to pay royalties.

The second agreement relates to an exclusive worldwide sublicense to us under certain patents, patent applications and know-how to make and have made, use and have used, sell, offer for sale, have sold and import protein products composed of one or more proteins encoded by the growth factor gene nrg-2 and non-protein products developed through the use of material covered by a valid claim of the patents. The license to this patent and the right to sub-license this patent was granted to CeNeS by the President and Fellows of Harvard College.

We have agreed to a timeline to achieve certain milestones relating to the research and development and the clinical testing and filing of regulatory approvals for the products. We are also required to make milestone payments of up to $5.93 million. If we are unable to meet a milestone, CeNeS has agreed to negotiate in good faith with us to agree for a reasonable extension of the time to achieve the milestone up to one year. We are obligated to pay CeNeS a license fee and royalties based on a percentage of net sales of protein products and non-protein products covered under the agreement. We have made payments of $25,000 in connection with this agreement through December 31, 2008.

This second agreement may be terminated by either party following an unremedied default of a material obligation by the other party. CeNeS may terminate this agreement upon our failure to cure a default in our obligations relating to maintenance of insurance liability or our failure to meet certain milestones. Harvard may terminate the underlying Harvard license if CeNeS becomes insolvent, makes an assignment of assets for the benefit of creditors, or has a petition bankruptcy filed for or against it. In that case, Harvard is required, upon our written request, to enter into a direct license with us under the same terms as those set forth in the agreement. We have the right

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to terminate this agreement upon written notice to CeNeS. The license granted to us pursuant to this agreement continues after the expiration of this agreement and may continue after the termination of this agreement, depending upon the circumstances under which this agreement is terminated.

Subject to early termination provisions, this agreement remains effective until the last patent, patent application or claim included in the licensed patents has expired, been abandoned or been held finally rejected or invalid.

Manufacturing

Fampridine-SR

In September 2003, we entered into an agreement with Elan for the supply of Fampridine-SR. Under that agreement, we are required to purchase at least 75% of our annual requirements of Fampridine-SR from Elan unless Elan is unable or unwilling to meet our requirements. In addition, the agreement also obligates us to make compensatory payments if we do not purchase 100% of our requirements from Elan.

As permitted by our agreement with Elan, we have designated Patheon, Inc. (Patheon) as a qualified second manufacturing source of Fampridine-SR. In connection with that designation, Elan assisted us in transferring manufacturing technology to Patheon. We and Elan have agreed that we may purchase up to 25% of our annual requirements from Patheon if we make compensatory payments to Elan. In addition, Patheon may supply us with Fampridine-SR if Elan is unable or unwilling to meet our requirements.

Zanaflex

We currently rely on Elan to supply us with Zanaflex Capsules. Zanaflex Capsules are manufactured using Elan's proprietary SODAS (spheroidal oral drug absorption system) multiparticulate drug delivery technology. We provide Elan with monthly written 18-month forecasts, and with annual written two-year forecasts, of our supply requirements for Zanaflex Capsules. In each of the five months following the submission of our 18-month forecast, we are obligated to purchase the quantity specified in the forecast, even if our actual requirements are greater or less. Elan is not obligated to supply products in excess of our forecast requirements, but will use commercially reasonable efforts to fulfill any such orders. The initial term of the agreement expires in 2009, with two automatic two-year renewal terms. Either party may terminate the agreement by notifying the other party at least 12 months prior to the expiration of the initial term or any renewal term. In addition, either party may terminate the agreement if the other party commits a material breach that remains uncured. If a failure to supply occurs under the agreement, other than a force majeure event, or if we terminate the supply agreement for cause, Elan must use commercially reasonable efforts to assist us in transferring production of Zanaflex Capsules to us or a third-party manufacturer, provided that such third party is not a technological competitor of Elan. If we need to transfer production, Elan has agreed to grant us a royalty-free, fully paid-up license of its manufacturing know-how and other information and rights related to the production of Zanaflex Capsules, including a license to use its SODAS technology for specified purposes. We have the right to sublicense this know-how to a third party manufacturer, provided that this third party is not a technological competitor of Elan. In the event of termination of the supply agreement due to a force majeure event that continues for more than three months, Elan has agreed to enter into negotiations with us to preserve the continuity of supply of products, including the possibility of transferring manufacturing of Zanaflex Capsules to us or a third party manufacturer.

Prior to March 2007, we relied on a single manufacturer, Novartis, for the manufacture of Zanaflex tablets and for the supply of tizanidine, the active pharmaceutical ingredient (API) in

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Zanaflex Capsules and Zanaflex tablets. Novartis has discontinued production of tizanidine and will no longer supply tizanidine to Elan for the production of Zanaflex Capsules or to us for the production of Zanaflex tablets. We have recently received FDA approval to use the tizanidine manufactured by a new supplier, Farmak, as the active ingredient in the production of Zanaflex Capsules. However, we are still required to obtain FDA approval for a new supplier of the tizanidine needed for the production of Zanaflex tablets. Elan has agreed to supply us with Novartis-manufactured tizanidine for the manufacture of Zanaflex tablets to satisfy supply requirements through the first quarter of 2010. If we fail to gain FDA approval of a new tizanidine supplier for Zanaflex tablets prior to February 2010, we may experience an interruption in our supply.

We are currently in contract negotiations with Patheon regarding the manufacture of Zanaflex tablets, and Patheon has agreed to manufacture Zanaflex tablets prior to the contract being executed. If either Elan or Patheon experiences any disruption in their operations, a delay or interruption in the supply of our Zanaflex products could result until the affected supplier cures the problem or we locate an alternate source of supply. We may not be able to enter into alternative supply arrangements on terms that are commercially favorable, if at all. Any new supplier would also be required to qualify under applicable regulatory requirements. We could experience substantial delays before we are able to qualify any new supplier and transfer the required manufacturing technology to that supplier.

We do not anticipate an interruption in Zanaflex Capsule or Zanaflex tablet API supply given the current Zanaflex sales forecast, the quantity of Elan tizanidine inventory and tizanidine's long-term stability profile.

Preclinical Products

We have established the internal capability to manufacture research quantities of antibody and protein product candidates and have contracted for testing and manufacturing development activities for GGF2 to be performed by an outside contractor. In March 2008, we signed a Master Services Agreement with CMC ICOS Biologics to develop methods to produce GGF2 under cGMPs.

Intellectual Property

As of February 13, 2009, our intellectual property portfolio included intellectual property rights to over 35 U.S. patents, over 110 foreign patents and over 100 pending patent applications world-wide. There are five major families of subject matter in our patent portfolio: Fampridine-SR, Zanaflex, neuregulins, remyelinating antibodies, and chondroitinase. Our intellectual property also includes confidential and trade secret information as well as a portfolio of trademarks.

Fampridine-SR

We have a patent portfolio with multifaceted coverage on fampridine-related subject matter. Overall, our fampridine intellectual property estate includes approximately 18 fampridine and responder analysis-related patent applications. A total of 54 fampridine-related patents are issued and are being maintained world-wide (six in the U.S., 38 in the European Union, and ten in other jurisdictions).

We hold an exclusive, worldwide license from Elan to three U.S. patents, with over 20 corresponding foreign patents and pending applications in a number of foreign countries. These patents and applications relate to timed delivery formulations of a family of aminopyridine compounds, including fampridine, and methods of treatment directed to classes of relevant neurological conditions. One of the three U.S. patents expires in 2011 and the other two U.S. patents expire in 2013.

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In addition to those Elan patents, we have two pending U.S. patent applications and corresponding applications in a number of foreign countries covering methods of using aminopyridines, such as Fampridine-SR. If granted, a patent resulting from any of these applications would be expected to expire in 2025.

We hold an exclusive license from Cornell University for an issued U.S. patent that relates to the use of aminopyridine compositions, including fampridine, for the treatment of diseases of anterior horn cells, including amyotrophic lateral sclerosis, which is also known as Lou Gehrig's disease. This patent expires in 2016.

We hold an exclusive, worldwide license from the Canadian Spinal Research Association (CSRO) for one U.S. patent and over 20 foreign counterpart patents covering the use of fampridine in the treatment of spasticity and chronic pain in patients with SCI. The U.S. patent expires in 2013.

In February 2008, we acquired certain assets of Neurorecovery, Inc. (NRI). This acquisition enabled us to broaden our intellectual property portfolio on fampridine, and explore additional therapeutic indications for Fampridine-SR, as well as provide access to pre-clinical compounds that may have utility in nervous system disorders. Under the terms of the purchase agreement, we were assigned two key licensing and research agreements relating to the use of aminopyridines in peripheral neuropathies and to two early stage development candidates. We also acquired NRI's pre-clinical and clinical data, regulatory filings (including Orphan Drug designations), copyrights, trademarks and domain names relating to the three products. Two Phase 2 studies of the aminopyridine compound Ampydin® (IR) for the treatment of chronic functional motor and sensory deficits resulting from Guillain-Barre Syndrome (GBS) have been completed. During the past year, we evaluated the technologies acquired from NRI. Based on this evaluation, we identified certain non-aminopyridine technologies and devices that were not sufficiently relevant to our goals or business interests, and have begun the requisite process for returning the intellectual property relating to those technologies to their original licensor, the University of Alabama. We will continue to retain intellectual property that includes an issued U.S. patent and corresponding foreign patents covering the use of mono-aminopyridines, such as fampridine, to treat GBS.

Zanaflex

As part of our purchase from Elan of the Zanaflex assets, we acquired one issued U.S. patent and two pending U.S. patent applications. Our issued patent is generally directed to certain methods of reducing somnolence and reducing peak plasma concentrations in patients receiving tizanidine therapy. This issued patent expires in 2021. Our two pending U.S. patent applications are directed to multiparticulate formulations of tizanidine and certain other methods of using tizanidine.

In addition, we entered into a Supply Agreement with Elan as part of the acquisition, whereby Zanaflex Capsules are manufactured for us by Elan using Elan's proprietary SODAS technology and proprietary information. This proprietary technology is owned by Elan and, in the event Elan ceases to manufacture Zanaflex Capsules, Elan has agreed to grant us a royalty-free, fully paid-up license of its manufacturing know-how and other information and rights related to the production of Zanaflex Capsules, including a license to use its SODAS technology for specified purposes. We have the right to sublicense this know-how to a third-party manufacturer, so long as this third party is not a technological competitor of Elan.

We have purchased the Zanaflex trademarks in the United States from Elan.

In August 2007, we received a Paragraph IV Certification Notice from Apotex Inc. advising that it had submitted an Abbreviated New Drug Application (ANDA) to the FDA seeking marketing approval for generic versions of Zanaflex Capsules. In October 2007, we filed a lawsuit against Apotex Corp. and Apotex Inc. (collectively, Apotex) for patent infringement in relation to the filing of

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the ANDA by Apotex. The defendants have answered our complaint, asserting patent invalidity and non-infringement and counterclaiming, seeking a declaratory judgment of patent invalidity and non-infringement. We have denied those counterclaims. In March 2008, Apotex filed a motion, which we opposed, for partial judgment on the pleadings dismissing the Company's request for relief on the ground that the case is "exceptional" under U.S.C. §§ 271(e)(4) or 285. The court ruled in our favor and denied Apotex' motion in December 2008. We and Apotex are currently proceeding with discovery in the case. If the FDA approves the ANDA and Apotex is successful in challenging the validity of the patent, Apotex could be permitted to sell a generic tizanidine hydrochloride capsule in competition with Zanaflex Capsules and Zanaflex tablets.

Neuregulins

Our neuregulin patent portfolio contains over 20 pending applications, and over 80 Neuregulin-related issued patents.

We are the exclusive licensee under a license agreement with CeNeS Pharmaceuticals, plc, of a worldwide portfolio of patents, patent applications and IP rights related to products of neuregulin genes, including GGF2. Collectively, these patents claim the use of particular neuregulins to treat various pathophysiological conditions, particularly uses to stimulate myelinating cells in order to treat conditions of the central and peripheral nervous system that involve demyelination. These patents also claim a number of additional potential uses of neuregulins, including stimulation of growth in cardiac and mammalian muscle cells, as well as treating cardiac failure, ischemic brain events, peripheral neuropathy and nerve injury.

In June 2008, we obtained an issued patent which covers methods for eliciting muscle cell survival, cell division or development by use of neuregulins. In January 2009, we received a Notice of Allowance on a U.S. application directed to neuregulin subject matter, which we expect will issue in 2009. This allowed case covers using specified neuregulin sequences to treat a central or peripheral nervous system injury associated with demyelination.

Remyelinating Antibodies

We have approximately ten remyelinating antibody-related patent applications, along with 13 corresponding issued patents (two in the U.S. and 11 foreign). We are the exclusive licensee of a portfolio of patents and patent applications related to a series of remyelinating antibodies discovered in the laboratory of Dr. Moses Rodriguez at the Mayo Clinic for the treatment of CNS disorders. We have two U.S. patents, one of which was issued in January 2009 and is directed to antibody compositions than can induce remyelination, as well as several issued related foreign counterparts.

There are numerous pending U.S. and foreign pending applications in our portfolio. Work actively continues on this subject matter and the remyelinating antibodies patent portfolio is expected to expand with relevant new patent filings expected throughout 2009.

Chondroitinase

We have two chondroitinase-related U.S. patents, obtained in 2008, an issued Australian patent, and over 50 pending chondroitinase patent applications.

We have a license to a U.S. application and its foreign counterpart from King's College and University of Cambridge directed to treatment of CNS damage. We have filed a number of U.S. patent applications and their foreign counterparts directed to chondroitinase enzymes and methods of use and preparation. In particular, we have filed U.S. applications and foreign equivalents relating to chondroitinase enzymes, including fusion proteins of chondroitinase, chimeric proteins including

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chondroitinase, deletion mutants and certain methods relating to chondroitinase. One of the issued U.S. patents covers chondroitinase ABCI mutant enzymes and related methods of use, while the other covers novel chondroitinase compositions.

Trademarks

In addition to patents, our intellectual property portfolio includes trademarks. The marks "Acorda Therapeutics," and our stylized Acorda Therapeutics logo are registered trademarks that we own. We also own the rights to the marks "Zanaflex" and "Zanaflex Capsules" in the U.S. Our trademark portfolio also includes several pending trademark applications for potential product names and for disease awareness activities.

Competition

The market for developing and marketing pharmaceutical products is highly competitive. We are aware of many biotechnology and pharmaceutical companies that are engaged in development and/or marketing of therapeutics for a broad range of CNS conditions. Many of our competitors have substantially greater financial, research and development, human and other resources than we do. Furthermore, many of these companies have significantly more experience than we do in preclinical testing, human clinical trials, regulatory approval procedures and sales and marketing.

MS and SCI

Current disease management approaches to MS are classified either as relapse management or disease course management approaches. For relapse management, the majority of neurologists treat sudden and severe relapses with a four-day course of intravenous high-dose corticosteroids. Many of these corticosteroids are available generically. For disease course management, there are a number of FDA-approved MS therapies that seek to modify the immune system. These treatments attempt to reduce the frequency and severity of exacerbations or slow the accumulation of physical disability for people with certain types of MS, though their precise mechanisms of action are not known. These products include Avonex from Biogen-IDEC, Betaseron from Schering AG, Copaxone from Teva Pharmaceutical Industries, Ltd., Rebif from Merck Serono, and Tysabri from Biogen-IDEC and Elan.

Several biotechnology and pharmaceutical companies, as well as academic laboratories, are involved in research and/or product development for various neurological diseases, including MS and SCI. We are aware that Sanofi-Aventis is developing a sodium/potassium channel blocker, HP 184, with a potential indication in SCI, MS and other conditions. We believe that HP 184 is in clinical trials for MS and, depending on the results of those trials, any resulting product might compete with Fampridine-SR. We also believe that EUSA Pharma is developing a 3,4-diaminopyridine compound that is in clinical development for use in Lambert Eaton Syndrome. If this product were sucessfully developed and approved, physicians might prescribe it instead of Fampridine-SR even if it were not approved for MS. In certain circumstances, pharmacists are not prohibited from formulating certain drug compounds to fill prescriptions on an individual patient basis. We are aware that at present compounded fampridine is used by some people with MS or SCI. Although we expect this use to decrease substantially if Fampridine-SR is approved, it is possible that some people will continue to use compounded fampridine. Several companies are engaged in developing products that include novel immune system approaches and cell transplant approaches to remyelination for the treatment of people with MS. These programs are in early stages of development and may compete with Fampridine-SR or our preclinical candidates in the future.

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Our lead product candidate, Fampridine-SR, is the first product to our knowledge that acts to improve the function of nerve fibers in subjects with MS. We are not aware of other companies in clinical development with products that specifically address improvement of walking ability in subjects with MS. As a result of its focus on improving function, we believe that Fampridine-SR may be complementary to both the relapse management and disease course management therapies that are commercially available. Nonetheless, Fampridine-SR may compete for market acceptance with these current treatments because they have been accepted and regularly prescribed to people with MS by physicians.

Spasticity

Tizanidine, the active pharmaceutical ingredient in Zanaflex Capsules, Zanaflex tablets and generic tizanidine tablets, is one of the two leading FDA-approved treatments for spasticity, a symptom suffered by both MS and SCI patients. Zanaflex tablets were approved by the FDA in 1996 and lost compound patent protection in 2002. Twelve generic manufacturers of tizanidine are distributing their own tablet formulations. As noted earlier, the Company is in litigation with Apotex with regard to its filing of an ANDA for the approval of a purported generic version of Zanaflex Capsules and certification against the Company's patent. In addition, several companies have reported that they are working on potential new delivery formulations of tizanidine. Baclofen, which is also available generically, is the other leading drug for the treatment of spasticity. The mechanism of action and associated effects of baclofen are different from those of tizanidine. Due to the different pharmacokinetic profile of Zanaflex Capsules, Zanaflex tablets and generic tizanidine tablets are not AB-rated with Zanaflex Capsules.

Government Regulation

FDA Regulation and Product Approval

The FDA and comparable regulatory agencies in state and local jurisdictions and in foreign countries impose substantial requirements upon the preclinical testing, clinical development, manufacture, distribution and marketing of pharmaceutical products. These agencies and other federal, state and local entities regulate research and development activities and the testing, manufacture, quality control, safety, effectiveness, labeling, storage, distribution, record keeping, approval, advertising, sale, promotion, import and export of our products and product candidates.

In the United States, Zanaflex tablets, Zanaflex Capsules, and some of our product candidates, including Fampridine-SR, are regulated by the FDA as drugs. Other of our product candidates are potentially regulated both as drugs and as biological products. Drugs are subject to the Federal Food, Drug, and Cosmetic Act, as amended, and the regulations of the FDA, as well as to other federal, state, and local statutes and regulations. Biologics are regulated under both the Federal Food, Drug, and Cosmetic Act, as amended, and the Public Health Service Act, as amended. Violations of regulatory requirements at any stage may result in various adverse consequences, including the FDA's and other health authorities' delay in approving or refusal to approve a product. Violations of regulatory requirements also may result in enforcement actions, including withdrawal of approval, labeling restrictions, seizure of products, fines, injunctions and/or civil or criminal penalties. Similar civil or criminal penalties could be imposed by other government agencies or agencies of the states and localities in which our products are manufactured, sold or distributed.

The process required by the FDA under these laws before our product candidates may be marketed in the United States generally involves the following:

- preclinical laboratory and animal tests;

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submission to the FDA of an IND, an application which must become effective before clinical trials may begin;

completion of two adequate and well-controlled human clinical trials to establish the safety and efficacy of the proposed pharmaceutical in our intended use(s);

FDA review of whether the facility in which the product is manufactured, processed, packed or held meets standards designed to assure the product's continued quality; and

submission to the FDA of an NDA in the case of a drug, or a Biologics License Application, or BLA, in the case of a biologic, that must be approved containing preclinical and clinical data, proposed labeling and information to demonstrate that the product will be manufactured to appropriate standards.

The research, development and approval process requires substantial time, effort, and financial resources and we cannot be certain that any approval will be granted on a timely or commercially viable basis, if at all.

Preclinical studies include laboratory evaluation of the product candidate, its chemistry, formulation and stability, as well as animal studies to assess its safety and potential efficacy. We then submit the results of the preclinical studies, together with manufacturing information, analytical data and any available clinical data or literature to the FDA as part of an IND application, which must become effective before we may begin human clinical trials. The IND becomes effective 30 days after the FDA filing, unless the FDA, within the 30-day time period, raises concerns or questions about the conduct of the clinical trial. In such a case, the IND sponsor and the FDA must resolve any outstanding concerns before the clinical trial can begin. Further, an independent Institutional Review Board charged with protecting the welfare of human subjects involved in research at each medical center proposing to conduct the clinical trials must review and approve any clinical trial before it commences at that center. Study subjects must provide informed consent before their participation in the research study.

Human clinical trials are typically conducted in three sequential phases, which may overlap:

Phase 1. The drug is initially administered into healthy human subjects or subjects with the target condition and tested for safety, dosage tolerance, absorption, metabolism, distribution and excretion.

Phase 2. The drug is administered to a limited patient population to identify possible adverse effects and safety risks, to determine the efficacy of the product for specific targeted diseases and to determine dosage tolerance and optimal dosage.

Phase 3. When Phase 2 evaluations demonstrate that a dosage range of the drug is effective and has an acceptable safety profile, Phase 3 clinical trials are undertaken to confirm the clinical efficacy from Phase 2 and to further test for safety in an expanded population at geographically dispersed clinical trial sites.

In the case of product candidates for severe or life-threatening diseases such as MS, the initial human testing is often conducted in affected patients rather than in healthy volunteers. Since these patients already have the target condition, these clinical trials may provide initial evidence of efficacy traditionally obtained in Phase 2 clinical trials and thus these clinical trials are frequently referred to as Phase 1b clinical trials.

Before proceeding with a study, sponsors may seek a written agreement from the FDA regarding the design, size, and conduct of a clinical trial. This is known as an SPA. Three types of studies are eligible for SPAs: (1) carcinogenicity studies, (2) final product stability studies and (3) clinical studies for pivotal Phase 3 studies whose data will form the primary basis to establish a

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product's efficacy. Where the FDA agrees to an SPA, the agreement may not be changed by either the sponsor or the FDA except if the sponsor and the FDA agree to a change, or an appropriately senior FDA official determines that a substantial scientific issue essential to determining the safety or effectiveness of the product was identified after the testing began. SPAs thus help establish up front agreement with the FDA about the adequacy of the design of a clinical trial to support a regulatory approval, but the agreement is not binding if new circumstances arise. In addition, even if an SPA remains in place and the trial meets its endpoints with statistical significance, the FDA could determine that the overall balance of risks and benefits for the product candidate is not adequate to support approval, or only justifies approval for a narrow set of clinical uses or approval with restricted distribution or other burdensome post-approval requirements or limitations. There is thus no guarantee that a study will ultimately be adequate to support an approval even if the study is subject to an SPA.

Federal and state law requires the submission of registry and results information for most clinical trials. These requirements do not apply to Phase 1 clinical trials. Many of the federal law requirements for submission of results information have not yet been implemented and will be phased-in over time. Once fully implemented, the federal requirements will preempt similar requirements at the state and local level.

U.S. law requires that studies conducted to support approval for product marketing be "adequate and well controlled." In general, this means that either a placebo or a product already approved for the treatment of the disease or condition under study must be used as a reference control. Studies must also be conducted in compliance with good clinical practice, or GCP, requirements.

We cannot be certain that we will successfully complete Phase 1, Phase 2 or Phase 3 testing of our product candidates within any specific time period, if at all. Furthermore, the FDA or the Institutional Review Boards or the sponsor may prevent clinical trials from beginning or may place clinical trials on hold or terminate them at any point in this process if, among other reasons, they conclude that study subjects or patients are being exposed to an unacceptable health risk.

In the U.S., the results of product development, preclinical studies and clinical trials must be submitted to the FDA for review and approval prior to marketing and commercial shipment of the product candidate. If the product candidate is regulated as a drug, an NDA must be submitted and approved before commercial marketing may begin. If the product candidate, such as an antibody, is regulated as a biologic, a BLA must be submitted and approved before commercial marketing may begin. The NDA or BLA must include a substantial amount of data and other information concerning the safety and effectiveness (and, in the case of a biologic, purity and potency) of the compound from laboratory, animal and clinical testing, as well as data and information on manufacturing, product stability, and proposed product labeling.

Each domestic and foreign manufacturing establishment, including any contract manufacturers we may decide to use, must be listed in the NDA or BLA and must be registered with the FDA. The application will generally not be approved until the FDA conducts a manufacturing inspection, approves the applicable manufacturing process for the drug or biological product, and determines that the facility is in compliance with cGMP requirements. If the manufacturing facilities and processes fail to pass the FDA inspection, we will not receive approval to market these products. The FDA may also inspect clinical trial sites and will not approve the product unless the clinical studies have been conducted in compliance with GCP.

Under the Prescription Drug User Fee Act, as amended, the FDA receives fees for reviewing a BLA or NDA and supplements thereto, as well as annual fees for commercial manufacturing establishments and for approved products. These fees can be significant. For the FDA's fiscal year

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2009, the NDA or BLA review fee alone is $1,247,200, although certain limited deferrals, waivers and reductions may be available.

Under applicable laws and FDA regulations, each NDA or BLA submitted for FDA approval is usually reviewed for administrative completeness and reviewability within 45 to 60 days following submission of the application. If deemed complete, the FDA will "file" the NDA or BLA, thereby triggering substantive review of the application. The FDA can refuse to file any NDA or BLA that it deems incomplete or not properly reviewable. If the FDA refuses to file an application, the FDA will retain 25% of the user fee as a penalty. The FDA has established performance goals for the review of NDAs and BLAs six months for priority applications and 10 months for regular applications. However, the FDA is not legally required to complete its review within these periods and these performance goals may change over time. Moreover, the outcome of the review, even if generally favorable, typically is not an actual approval but an "action letter" that describes additional work that must be done before the application can be approved. The FDA's review of an application may involve review and recommendations by an independent FDA advisory committee.

The FDA may deny an NDA or BLA if the applicable regulatory criteria are not satisfied or may require additional clinical data. Even if such data is submitted, the FDA may ultimately decide that the NDA or BLA does not satisfy the criteria for approval. If the FDA approves a product, it may limit the approved therapeutic uses for the product as described in the product labeling, require that contraindications, warning statements or precautions be included in the product labeling, require that additional studies or clinical trials be conducted following approval as a condition of the approval, impose restrictions and conditions on product distribution, prescribing or dispensing in the form of a risk management plan or risk evaluation and mitigation strategy (REMS), or otherwise limit the scope of any approval or post-approval, or limit labeling. Under a REMS, the FDA may impose significant restrictions on distribution and use of a marketed product, may require the distribution of medication guides, patient labeling and/or communication plans, and may impose a timetable for submission of assessments of the effectiveness of a REMS. Once issued, the FDA may withdraw product approval if compliance with regulatory standards is not maintained or if problems occur after the product reaches the market. The FDA may also impose a REMS post-approval if it becomes aware of new safety information that it believes necessitates a REMS. In addition, the FDA may require testing and surveillance programs to monitor the effect of approved products which have been commercialized, and the agency has the power to prevent or limit further marketing of a product based on the results of these post-marketing programs.

Satisfaction of the above FDA requirements or similar requirements of state, local and foreign regulatory agencies typically takes several years or more and the actual time required may vary substantially, based upon the type, complexity and novelty of the pharmaceutical product candidate. Government regulation may delay or prevent marketing of potential products for a considerable period of time or permanently and impose costly procedures upon our activities. We cannot be certain that the FDA or any other regulatory agency will grant approval for any of our product candidates on a timely basis, or on a commercially viable basis, if at all. Success in preclinical or early stage clinical trials does not assure success in later stage clinical trials. Data obtained from preclinical and clinical activities is not always conclusive or applicable to humans and may be susceptible to varying interpretations which could delay, limit or prevent regulatory approval. Even if a product candidate receives regulatory approval, the approval may be significantly limited to specific indications. Further, even after regulatory approval is obtained, later discovery of previously unknown problems with a product may result in restrictions on the product or even complete withdrawal of the product from the market. Delays in obtaining, or failures to obtain regulatory approvals would have a material adverse effect on our business. Marketing our product candidates abroad will require similar regulatory approvals and is subject to similar risks. In

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addition, we cannot predict what adverse governmental regulations may arise from future U.S. or foreign governmental action.

Any products manufactured or distributed by us pursuant to FDA clearances or approvals are subject to pervasive and continuing regulation by the FDA, including record-keeping requirements, reporting of adverse experiences with the drug, other reporting, advertising and promotion restrictions. The FDA's rules for advertising and promotion require, among other things, that we not promote our products for unapproved uses and that our promotion be fairly balanced and adequately substantiated by clinical studies. We must also submit appropriate new and supplemental applications and obtain FDA approval for certain changes to the approved product, product labeling or manufacturing process. On its own initiative, FDA may require safety labeling changes to the labeling of an approved drug if it becomes aware of new safety information that the agency believes should be included in the approved drug's labeling. Drug manufacturers and their subcontractors are required to register their establishments with the FDA and certain state agencies, and are subject to periodic unannounced inspections by the FDA and certain state agencies for compliance with cGMP, which impose certain procedural and documentation requirements upon us and our third-party manufacturers. We cannot be certain that we or our present or future suppliers will be able to comply with the cGMP and other FDA regulatory requirements. The FDA also enforces the requirements of the Prescription Drug Marketing Act, or PDMA, which, among other things, imposes various requirements in connection with the distribution of product samples to physicians. Similar provisions exist at the state level.

In addition to inspections related to manufacturing, we are subject to periodic unannounced inspections by the FDA and other regulatory bodies related to the other regulatory requirements that apply to marketed drugs manufactured or distributed by us. The FDA also may conduct periodic inspections regarding our review and reporting of adverse events, or related to compliance with the requirements of the PDMA concerning the handling of drug samples. When the FDA conducts an inspection, the inspectors will identify any deficiencies they believe exist in the form of a notice of inspectional observations, or Form FDA 483. The observations may be more or less significant. If we receive a notice of inspectional observations, we likely will be required to respond in writing, and may be required to undertake corrective and preventive actions in order to address the FDA's concerns. For example, the FDA conducted an inspection in February 2009. This was the first FDA inspection we have had since a previous inspection the FDA conducted in 2006, which had resulted in a Form FDA 483 regarding our reporting of adverse events for marketed products. Although the 2009 inspection showed that we had resolved issues from the previous inspection, it also resulted in a Form FDA 483 with five inspectional observations. The observations cited the failure to submit NDA field alert reports for Zanaflex Capsules in a timely manner, the failure to review adequately complaints concerning distributed product, the late submission of NDA annual reports, and inadequate written procedures for our quality control unit, NDA field alert reporting, and the training of our personnel. We intend to undertake corrective and preventive actions in order to address the FDA's concerns cited in the Form FDA 483. However, we cannot assure you that the FDA will find our remedial actions to be adequate or that the FDA will not identify different or additional deficiencies in subsequent inspections.

We and our product candidates are also subject to a variety of state laws and regulations in those states or localities where they are or will be marketed. Any applicable state or local regulations may hinder our ability to market our product candidates in those states or localities.

The FDA's policies may change and additional government regulations may be enacted which could prevent or delay regulatory approval of our product candidates. Moreover, increased attention to the containment of health care costs in the United States and in foreign markets could result in new government regulations which could have a material adverse effect on our business. We

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cannot predict the likelihood, nature or extent of adverse governmental regulation which might arise from future legislative or administrative action, either in the United States or abroad.

Orphan Drugs

Under the Orphan Drug Act, special incentives exist for sponsors to develop products for rare diseases or conditions, which are defined to include those diseases or conditions that affect fewer than 200,000 people in the U.S. Requests for orphan drug designation must be submitted before the submission of an NDA or BLA. We have received orphan drug designation for Fampridine-SR for the treatment of both MS and incomplete SCI.

Products designated as orphan drugs are eligible for special grant funding for research and development, FDA assistance with the review of clinical trial protocols, potential tax credits for research, reduced filing fees for marketing applications. If a product that has an orphan drug designation is the first such product to receive FDA approval for the disease for which it has such designation, the product is entitled to orphan product exclusivity. This means that, subsequent to approval, the FDA may not approve any other applications to market the same drug for the same disease, except in limited circumstances, for seven years. FDA may approve a subsequent application from another person if FDA determines that the application is for a different drug or different use, or if FDA determines that the subsequent product is clinically superior, or that the holder of the initial orphan drug approval cannot assure the availability of sufficient quantities of the drug to meet the public's need. If the FDA approves someone else's application for the same drug that has orphan exclusivity, but for a different use, the competing drug could be prescribed by physicians outside its FDA approval for the orphan use, notwithstanding the existence of orphan exclusivity. A grant of an orphan designation is not a guarantee that a product will be approved. If a sponsor receives orphan drug exclusivity upon approval, there can be no assurance that the exclusivity will prevent another person from receiving approval for the same or a similar drug for the same or other uses.

Generic Drugs, AB Ratings and Pharmacy Substitution

Generic drugs are approved through an abbreviated process, which differs in important ways from the process followed for innovative products. Generally an abbreviated new drug application, or ANDA, is filed with the FDA. The ANDA must seek approval of a product candidate that has the same active ingredient(s), dosage form, strength, route of administration, and conditions of use (labeling) as a so-called "reference listed drug" approved under an NDA with full supporting data to establish safety and effectiveness. Only limited exceptions exist to this ANDA sameness requirement, including certain limited variations approved by the FDA through a special suitability petition process. The ANDA also generally contains clinical data to demonstrate that the product covered by the ANDA is absorbed in the body at the same rate and to the same extent as the reference listed drug. This is known as bioequivalence. In addition, the ANDA must contain information regarding the manufacturing processes and facilities that will be used to ensure product quality, and must contain certifications to patents listed with the FDA for the reference listed drug.

Many states require or permit pharmacists to substitute generic equivalents for brand-name prescriptions unless the physician has prohibited substitution. Managed care organizations often urge physicians to prescribe drugs with generic equivalents, and to authorize substitution, as a means of controlling costs of prescriptions. They also may require lower copayments as an incentive to patients to ask for and accept generics.

While the question of substitutability is one of state law, most states look to the FDA to determine whether a generic is substitutable. FDA lists therapeutic equivalence ratings in a publication often referred to as the Orange Book. In general, a generic drug that is listed in the

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Orange Book as therapeutically equivalent to the branded product will be substitutable under state law and, conversely, a generic drug that is not so listed will not be substitutable. To be considered therapeutically equivalent, a generic drug must first be a pharmaceutical equivalent of the branded drug. This means that the generic has the same active ingredient, dosage form, strength or concentration and route of administration as the brand-name drug. Tablets and capsules are presently considered different dosage forms that are pharmaceutical alternatives and not substitutable pharmaceutical equivalents.

In addition to being pharmaceutical equivalents, therapeutic equivalents must be bioequivalent to their branded counterparts. Bioequivalence for this purpose is defined in the same manner as for ANDA approvals, and usually requires a showing of comparable rate and extent of absorption in a small human study.

Solid oral dosage form drug products generally are rated "AB" in the Orange Book if they are considered therapeutic equivalents. If bioequivalence has been adequately demonstrated, the products will be rated "AB."

Foreign Regulation and Product Approval

Outside the United States, our ability to market a product candidate is contingent upon receiving a marketing authorization from the appropriate regulatory authorities. The requirements governing the conduct of clinical trials, marketing authorization, pricing and reimbursement vary widely from country to country. At present, foreign marketing authorizations are applied for at a national level, although within the European Community, or EC, registration procedures are available to companies wishing to market a product in the entire European Economic Area (EEA) or in more than one individual EC member state. This foreign regulatory approval process involves all of the risks associated with FDA approval discussed above.

Other Regulations

In the U.S., the research, manufacturing, distribution, sale, and promotion of drug and biological products are potentially subject to regulation by various federal, state and local authorities in addition to the FDA, including the Centers for Medicare and Medicaid Services (formerly the Health Care Financing Administration), other divisions of the U.S. Department of Health and Human Services (e.g., the Office of Inspector General), the U.S. Department of Justice and individual U.S. Attorney offices within the Department of Justice, and state and local governments. For example, sales, marketing and scientific/educational grant programs must comply with the anti-kickback and fraud and abuse provisions of the Social Security Act, as amended, the False Claims Act, also as amended, the privacy provisions of the Health Insurance Portability and Accountability Act, or HIPAA, and similar state laws. Pricing and rebate programs must comply with the Medicaid rebate requirements of the Omnibus Budget Reconciliation Act of 1990, as amended, and the Veterans Health Care Act of 1992, as amended. If products are made available to authorized users of the Federal Supply Schedule of the General Services Administration, additional laws and requirements apply. All of these activities are also potentially subject to federal and state consumer protection and unfair competition laws.

We are also subject to numerous federal, state and local laws relating to such matters as safe working conditions, manufacturing practices, environmental protection, fire hazard control, and disposal of hazardous or potentially hazardous substances. We may incur significant costs to comply with such laws and regulations now or in the future.

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Reimbursement and Pricing Controls

In many of the markets where we or our collaborative partners would commercialize a product following regulatory approval, the prices of pharmaceutical products are subject to direct price controls (by law) and to drug reimbursement programs with varying price control mechanisms.

In the United States, there has been an increased focus on drug pricing in recent years. Although there are currently no direct government price controls over private sector purchases in the United States, federal legislation requires pharmaceutical manufacturers to pay prescribed rebates on certain drugs to enable them to be eligible for reimbursement under certain public health care programs such as Medicaid. Various states have adopted further mechanisms under Medicaid and otherwise that seek to control drug prices, including by disfavoring certain higher priced drugs and by seeking supplemental rebates from manufacturers. Managed care has also become a potent force in the market place that increases downward pressure on the prices of pharmaceutical products. Federal legislation, enacted in December 2003, has altered federal reimbursement for physician-administered drugs covered by Medicare. Under the new reimbursement methodology, physicians are reimbursed based on a product's "average sales price," or ASP. This new reimbursement methodology has generally led to lower reimbursement levels. The new federal legislation also added an outpatient prescription drug benefit to Medicare, effective January 2006. This benefit is provided primarily through private entities, which attempt to negotiate price concessions from pharmaceutical manufacturers.

Public and private health care payers control costs and influence drug pricing through a variety of mechanisms, including through negotiating discounts with the manufacturers and through the use of tiered formularies and other mechanisms that provide preferential access to certain drugs over others within a therapeutic class. Payors also set other criteria to govern the uses of a drug that will be deemed medically appropriate and therefore reimbursed or otherwise covered. In particular, many public and private health care payers limit reimbursement and coverage to the uses of a drug that are either approved by the FDA and/or appear in a recognized drug compendium. Drug compendia are publications that summarize the available medical evidence for particular drug products and identify which uses of a drug are supported or not supported by the available evidence, whether or not such uses have been approved by the FDA. For example, in the case of Medicare coverage for physician-administered oncology drugs, the Omnibus Budget Reconciliation Act of 1993, or OBRA '93, with certain exceptions, provides for Medicare coverage of unapproved uses of an FDA-approved drug if the unapproved use is reasonable and necessary and is supported by one or more citations in the American Hospital Formulary Service Drug Information, the national Comprehensive Cancer Network Drugs and Biologics Compendium, Thompson Micromedix, DrugDex, or Clinical Pharmacology. Another commonly cited compendium, for example under Medicaid, is the DrugDex Information System.

Different pricing and reimbursement schemes exist in other countries. For example, in the European Union, governments influence the price of pharmaceutical products through their pricing and reimbursement rules and control of national health care systems that fund a large part of the cost of such products to consumers. The approach taken varies from member state to member state. Some jurisdictions operate positive and/or negative list systems under which products may only be marketed once a reimbursement price has been agreed. Other member states allow companies to fix their own prices for medicines, but monitor and control company profits and may limit or restrict reimbursement. The downward pressure on health care costs in general, particularly prescription drugs, has become very intense. As a result, increasingly high barriers are being erected to the entry of new products, as exemplified by the National Institute for Clinical Excellence in the UK which evaluates the data supporting new medicines and passes reimbursement recommendations to the government. In addition, in some countries cross-border imports from low-priced markets (parallel imports) exert a commercial pressure on pricing within a country.

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EMPLOYEES

As of February 13, 2009, we had 174 employees. Of the 174 employees, 42 perform research and development activities, including preclinical programs, clinical trials, regulatory affairs and biostatistics, 98 work in sales, marketing, medical affairs, business development, manufacturing and communications, and 34 perform general and administrative tasks.

CORPORATE INFORMATION

We were incorporated in 1995 as a Delaware corporation. Our principal executive offices are located at 15 Skyline Drive, Hawthorne, New York 10532. Our telephone number is (914) 347-4300. Our website is *www.acorda.com*.

ADDITIONAL INFORMATION AND WHERE TO FIND IT

Our Annual Report on Form 10-K, Quarterly Reports on Form 10-Q, Current Reports on Form 8-K and amendments to those reports filed or furnished pursuant to Section 13(a) or 15(d) of the Securities Exchange Act of 1934 are available on our website (*http://www.acorda.com* under the "SEC Filings" caption) as soon as reasonably practicable after we electronically file such material with, or furnish them to, the Securities and Exchange Commission (SEC).

Item 1A. Risk Factors.

An investment in our common stock involves a high degree of risk. You should consider carefully the following risk factors and the other information contained in this prospectus and the documents incorporated by reference herein before you decide to purchase our common stock. Additional risks that are not currently known or foreseeable to us may materialize at a future date. Our business and financial results could be adversely affected in a material way if any of these risks or uncertainties occur and you might lose all or part of your investment.

Risks related to our business

We have a history of operating losses and we expect to continue to incur losses and may never be profitable.

As of December 31, 2008, we had an accumulated deficit of approximately $344.4 million. We had net losses of $74.3 million, $38.0 million, and $60.0 million for the years ended December 31, 2008, 2007 and 2006, respectively. We have had operating losses since inception as a result of our significant clinical development, research and development, general and administrative, sales and marketing and business development expenses. We expect to incur losses for at least the next several years as we expand our sales and marketing capabilities, prepare for the potential launch of Fampridine-SR, and continue product development and research and development activities.

Our prospects for achieving profitability will depend primarily on how successful we are in executing our business plan to:

- obtain FDA approval for and commercialize Fampridine-SR;
- increase sales of Zanaflex Capsules;
- continue to develop our preclinical product candidates and advance them into clinical trials; and
- evaluate and act on appropriate opportunities for increasing shareholder value.

If we are not successful in executing our business plan, we may never achieve or may not sustain profitability.

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If we are unable to obtain regulatory approval for Fampridine-SR in the U.S., the European Union, or other markets, or any approval is unduly limited in scope or delayed, our business prospects will be materially adversely affected.

We have filed an NDA for approval of Fampridine-SR for the improvement of walking in patients with MS, based on positive results from two Phase 3 clinical trials conducted pursuant to SPAs from the FDA. If the FDA determines that our NDA is incomplete or otherwise not adequate to support review, it will refuse to accept the NDA for filing and will not proceed to further review. If the FDA accepts the NDA for filing but determines that there is a new substantial scientific issue regarding walking in the MS population or Fampridine-SR, the FDA may alter its opinion expressed in the prior SPAs regarding the adequacy of the design of the Phase 3 studies. The FDA or the regulatory authorities in the European Union or other markets where we may apply for approval may also determine that the risks and benefits shown by the data that we have submitted do not support approval, or support only a limited approval, or an approval conditioned on burdensome post-approval commitments.

The FDA may identify a need for further studies in order to confirm efficacy or to examine safety or other properties or characteristics of Fampridine-SR. For example, in October 2007, we met with the FDA to discuss the completed preclinical studies proposed for the NDA for Fampridine-SR and the FDA asked us to complete a series of bridging studies to bring our older preclinical toxicology studies to current scientific standards. This included a requirement to complete new studies to fully characterize the toxicokinetics of fampridine in the blood of experimental animals given doses that were used in the full range of our previously performed preclinical toxicology studies, so the FDA can evaluate the suitability of those doses and routes of administration of drug in its evaluation of safety. These additional studies were completed before the submission of our NDA in January 2009. The FDA also required us to execute a Thorough QT study of cardiac safety which was completed in January 2008. Although our QT consultants concluded that this study showed no safety signal for a risk of cardiac QT prolongation with Fampridine-SR at a therapeutic or supra-therapeutic dose, the FDA will make its own evaluation of the data as part of the NDA review and its interpretation of the results may differ.

We may also determine, on our own, to conduct additional studies from time to time to support our filing of an NDA or regulatory filings in other markets or to otherwise provide additional data regarding the safety or efficacy of Fampridine-SR. If the studies that we are required to conduct, or any studies that we determine, on our own, to conduct, cause us to incur unanticipated expenses or delays, or yield unfavorable results, our ability to obtain regulatory approval of Fampridine-SR could be seriously delayed or impaired, in which case our business prospects will be materially adversely affected.

In June 2008, we submitted a request to the FDA for Fast Track designation for Fampridine-SR. The FDA did not grant our request, stating that we had not at that time demonstrated that Fampridine-SR addresses an unmet medical need under the criteria for Fast Track designation. We presented additional information on the ways in which Fampridine-SR improves walking ability in patients with MS and differs in its effects from existing MS therapies as part of our request for reconsideration of that decision, but the FDA did not change its decision. We did not apply for Priority Review, and our NDA is therefore subject to FDA's normal 10 month review time under the Prescription Drug User Fee Act, rather than an expedited review time of six months.

Notwithstanding the results of our clinical trials and pre-clinical studies, the FDA could determine that the overall balance of risks and benefits for Fampridine-SR is not adequate to support approval, or only justifies approval for a narrow set of uses or approval with restricted distribution or other burdensome post-approval requirements and limitations. Subjects taking Fampridine-SR have experienced adverse events, including falls, urinary tract infection, insomnia,

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dizziness, asthenia, headache, fatigue, nausea and balance disorder. A small number of subjects have also experienced seizures while taking Fampridine-SR, and there is a possibility that additional seizures will occur even at low doses of the drug. If the FDA denies approval of Fampridine-SR in MS, if FDA approval is substantially delayed, if approval is granted on a narrow basis or with restricted distribution or other burdensome post-approval requirements, or if the Fampridine-SR program is terminated, our business prospects will be materially adversely affected.

In March 2004, we completed two Phase 3 clinical trials of Fampridine-SR in SCI in which our results failed to reach their primary endpoints. We may resume development of Fampridine-SR for SCI after we have completed further development of the drug for MS. However, we cannot predict whether future clinical trials of Fampridine-SR in SCI will achieve their primary endpoints, how long these clinical trials will take or how much they will cost.

We will be substantially dependent on sales of one product, Zanaflex Capsules, to generate revenue for the foreseeable future.

We currently derive substantially all of our revenue from the sale of Zanaflex Capsules and Zanaflex tablets, which are our only FDA-approved products. Although we currently distribute Zanaflex tablets, our marketing efforts are focused on Zanaflex Capsules and we do not, and do not intend to, actively promote Zanaflex tablets. As a result, prescriptions for Zanaflex tablets have declined and we expect that they will continue to decline. Our goal is to convert sales of Zanaflex tablets and generic tizanidine tablets to sales of Zanaflex Capsules. We believe that sales of Zanaflex Capsules will constitute a significant portion of our total revenue until we begin to generate sales of Fampridine-SR if approved. If we are unable to convert tablet sales to capsule sales or are otherwise unable to increase our revenue from the sale of this product, our business, financial condition and results of operations could be materially adversely affected.

We expect to further increase our sales force in anticipation of the possible launch of Fampridine-SR and sales of Zanaflex Capsules may not grow sufficiently, or Fampridine-SR may not get approved, to offset the increased costs associated with this expansion.

Our internal sales force has increased to 61 people as part of our strategy to increase sales of Zanaflex Capsules, which increased our fixed expenses significantly. We expect to further increase our sales force, approximately doubling it, in anticipation of the possible launch of Fampridine-SR, if approved. If we expand our sales force and an NDA is not approved by the FDA, or, if approved, we are not able to achieve our expected level of sales of Zanaflex Capsules and Fampridine-SR, our cash flow and our prospects for achieving profitability will be adversely affected. In addition, we may not be able to hire, train and retain skilled sales and marketing personnel, in a timely manner or at all, or integrate and manage our larger sales and marketing organization.

There are currently 12 companies with generic versions of tizanidine tablets on the market and they are significantly cheaper than either Zanaflex Capsules or Zanaflex tablets. As of December 31, 2008, these generic versions of tizanidine tablets constituted approximately 93% of tizanidine sales in the United States. Although Zanaflex Capsules have a different pharmacokinetic profile when taken with food and are available in a higher dose than Zanaflex tablets and their generic equivalents, we may be unsuccessful in convincing prescribers, patients and third-party payers, such as government health administrative authorities, including Medicaid and Medicare, private health insurers and other such organizations that these differences justify the higher price of Zanaflex Capsules. Despite our increased investment in sales personnel, we may be unable to convert a significant additional number of current users of Zanaflex tablets or generic tizanidine tablets to Zanaflex Capsules. If that is the case, our ability to continue to generate meaningful revenue from this product will be adversely affected.

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We will need to obtain regulatory approval in foreign jurisdictions where we seek to market our products.

In order to market our products in the European Union and many other foreign jurisdictions, we must obtain separate regulatory approvals and comply with numerous and varying regulatory requirements. Approval procedures vary among countries and can involve additional clinical testing. The time required to obtain approval may differ from that required to obtain FDA approval. We may fail to obtain foreign regulatory approvals on a timely basis, if at all. In addition, individual countries, within the European Union or elsewhere, may require additional steps after regulatory approval to gain access to national markets, such as agreements with pricing authorities and other agencies, that may affect our ability to market and sell our products outside the United States. Approval by the FDA does not ensure approval by regulatory authorities in other countries, and approval by one foreign regulatory authority does not ensure approval by regulatory authorities in other foreign countries or by the FDA. We may not be able to file for, and may not receive, necessary regulatory approvals to commercialize our products in any foreign market, which could adversely affect our business prospects.

During 2008, we met with national regulatory authorities in four European Union member states regarding Fampridine-SR and we believe, based on these discussions, that our current data are sufficient to allow us to file a centralized MAA with the EMEA. The EMEA may determine that such an MAA cannot be filed centrally, in which case we would be required to seek approval separately from each member state in which we wish to market Fampridine-SR through the European Union's decentralized or mutual recognition procedures. The EMEA may also determine that the data we submit are not sufficient to support an application for marketing approval of Fampridine-SR, which could lead to additional information requirements, including the submission of data from supplemental clinical trials other than those that support our U.S. filings with the FDA. Any requirements to conduct supplemental trials would add to the cost of developing our product candidates. Additional or supplemental trials could also produce findings that are inconsistent with the trial results we have previously submitted to the FDA, in which case we would be obligated to report those findings to the FDA.

Our product candidates must undergo rigorous clinical testing, the results of which are uncertain and could substantially delay or prevent us from bringing them to market.

Before we can obtain regulatory approval for a product candidate, we must undertake extensive clinical testing in humans to demonstrate safety and efficacy to the satisfaction of the FDA and other regulatory agencies. Clinical trials of new product candidates sufficient to obtain regulatory marketing approval are expensive and take years to complete, and the outcome of such trials is uncertain.

Clinical development of any product candidate that we determine to take into clinical trials may be curtailed, redirected, delayed or eliminated at any time for some or all of the following reasons:

- negative or ambiguous results regarding the efficacy of the product candidate;
- undesirable side effects that delay or extend the trials, or other unforeseen or undesirable safety issues that make the product candidate not medically or commercially viable;
- inability to locate, recruit and qualify a sufficient number of patients for our trials;